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CYCLOPÆDIA

OF THE

DISEASES OF CHILDREN

MEDICAL AND SURGICAL.

THE ARTICLES WRITTEN ESPECIALLY FOR THE WORK BY
AMERICAN, BRITISH, AND CANADIAN AUTHORS.

EDITED BY
JOHN M. KEATING, M.D.

VOL. IV.

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CYCLOPÆDIA
OF THE
DISEASES OF CHILDREN.

PART I.
THE EAR.

DISEASES OF THE EAR IN CHILDREN.

By CHARLES H. BURNETT, M.D.

DISEASES OF THE EXTERNAL EAR.

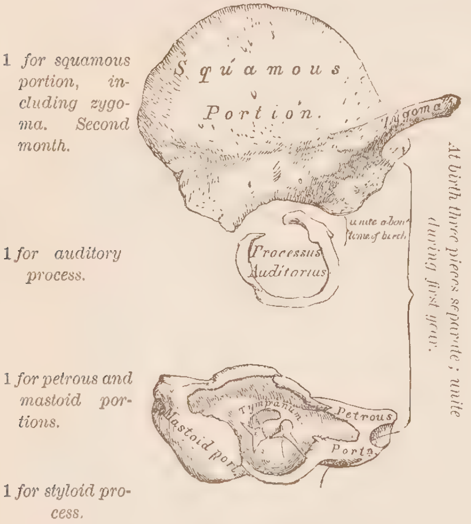
In the following pages those under fifteen years of age are considered children. The percentage of children in ear-cases varies in different countries, or in different parts of the same country. Marian, of Bohemia, states that twenty-five per cent. of his cases of ear-disease are children, about equally divided between the sexes. Bürkner, of Göttingen, places the percentage at forty-seven and three-tenths, and Bezold, of Munich, places it at twenty-one and two-tenths, respectively, in their experience.

In this country, Blake, of Boston, finds that twelve per cent. of his cases are children. Spencer, of St. Louis, states that in private practice seventeen per cent. of his cases are children, while in the public clinic children constitute twenty-seven per cent. of the cases. In New York A. H. Buck finds twenty per cent. of his cases in private practice are children, and Sexton gives fifteen per cent. My own experience shows that in Philadelphia twenty-two and a half per cent. of the cases in the Polyclinic are children, while in private practice the percentage is eight and a half in my last two hundred consecutive cases.

The auditory apparatus of a child differs in some respects from that of an adult. In the child the auricle and the auditory canal are much smaller than in the adult, while the membrana tympani and the ossicles in the middle ear are of full size at birth. The drum-cavity is of the same size in the new-born child as in the adult, but there is no mastoid process in connection with it, as the mastoid cells are developed at a later period. The internal ear is the same in the child as in the adult, though the semicircular canals stand out in the cranial cavity, free from the dense osseous tissue which finally encases them in the older bone. These differences, especially those in the external ear, should receive a little closer regard. Therefore the reader's attention is called to the following anatomical description.

The external ear of the child consists of the auricle and a fibro-cartilaginous auditory canal, the latter being terminated at its fundus by the membrana tympani. In the adult we find between the auricle and the membrana tympani an osseous canal, an inch or more in length. In the child, however, there is no osseous auditory canal in its complete state. Between infancy and maturity the annulus tympanicus may be said to be gradually transformed into an osseous tube. Natural dehiscences persist in the anterior wall of the auditory canal, sometimes until the fifth year. They are analogues of the incisuræ Santorini.

Fig. 1.

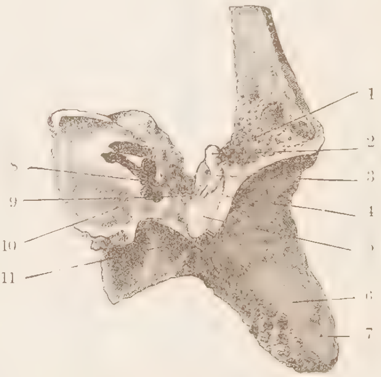

1 for squamous portion, including zygoma. Second month.
1 for auditory process.
1 for petrous and mastoid portions.
1 for styloid process.
Squamous Portion.
Zygoma
unite about time of birth
Processus Auditorius
At birth three pieces separate; unite during first year.
Tympanum.
Petrous
Mastoid Port.
Porta

Infant's temporal bone. (Gray.)

At birth the temporal bone consists of three separate parts,—the pyramid, the squama, and the annulus tympanicus. (Fig. 1.) By the end of the first year of life osseous union has taken place between these parts. The child's temporal bone, however, is very different from that of the adult. Development continues until the age of puberty, when the bone is complete. In the pyramid of the young child, in that part containing the internal ear, the semicircular canals are not covered in by the cancellated tissue which is found later in life. In the squama we find no developed mastoid cells,—merely the trace of an antrum. The external osseous auditory canal does not exist, excepting in the annulus tympanicus, which finally develops outwardly into the bony canal. The auricle is joined to the annulus tympanicus by fibrous tissue, is very pliable, but quite short, which brings the concha of the auricle very much nearer the drum than in older children or in adults. Hence the membrana tympani in the infant may be said to be superficially placed, and easily reached—sometimes very unfortunately so—by any one manipulating in the external meatus.

The middle ear, or tympanic cavity, is the space lying between the outer surface of the pyramid and the inner surface of the annulus tympanicus, and the membrana tympani, and bounded behind by the union of the squama and the pyramid in the region of the mastoid antrum. Anteriorly the tympanic cavity is open to the Eustachian tube.

FIG. 2.



LEFT TYMPANIC CAVITY LAID OPEN. (Burnett.)—1, mastoid cells laid open; 2, head of malleus; 3, malleus; 4, annulus tympanicus; 5, membrana tympani; 6, lower mastoid cells; 7, mastoid process; 8, the vestibule; 9, stapes in the oval window; 10, position of cochlea, removed; 11, jugular fossa.

In the tympanic cavity are swung the three auditory ossicles,—the malleus, the incus, and the stapes. The first is inserted by its so-called handle into the membrana tympani, and by its head articulates with the body of the incus. The incus by its long process is attached to the stapes. The stapes by its foot-plate is inserted into the oval window, thus completing the junction between the membrana tympani and the internal ear.

Causes of Ear-Disease in Children.—The commonest causes of aural disease in childhood are the acute exanthemata, acute and chronic catarrh of the nares and naso-pharynx, diphtheria, diseases of the heart, and hereditary syphilis; while in older children typhoid fever has much to answer for. Measles and diphtheria together, however, do not affect one-quarter as many ears as scarlet fever.

Malformations.—Shallowness of the niches of the round and oval windows, on the inner wall of the drum-cavity, favors a retrogression of swelling in the mucous membrane of these parts. A greater depth of these niches, especially in that of the oval window, where the stapes sits, is unfavorable for healing and favors chronicity of disease. Thus may be explained many cases of profound deafness, even deaf-dumbness, in children, without resorting to an hypothesis of disease in the internal ear, or labyrinth.

In hydrocephalus the temporal bone is pushed downward by the superincumbent water, the external auditory canal being forced in the same direction.

An arrest of development may occur in the middle ear, the external ear being normal; and the reverse may also be found.

In infancy and up to the fourth year (Huschke) a deficiency or a gap in the bone of the external auditory canal at the anterior lower wall may be found, closed, however, by connective tissue. Through this opening inflammation may extend from the external ear, by ulceration of the skin, to the parotid gland and the lower jaw. Defects in ossification are frequent in the tegmen tympani, with normal dura mater, in the carotid canal, near the front part of the tympanic cavity, and in the facial canal. These explain the ready occurrence of facial palsy in otitis media. Defects in the bone are found also in the floor of the tympanum over the jugular fossa, in the rudimentary cortex of the mastoid, and in the squama. Inflammation may, through these deficiencies in the bone about the ear, be transmitted to the brain, inducing a fatal termination of an aural disease in a child.

Hyperæmia of the petrous bone occurs markedly in variola and in typhoid fever. The ear-disease so often following these maladies may be thus accounted for.

Hyperostosis of the petrous bone may be the result of ossifying periostitis in fœtal life or in early childhood. It always leads to great deafness and deaf-dumbness.

Caries, or ulcerative ostitis of Schwartze, attacks the petrous bone most frequently of all the cranial bones. It is usually the result of an acute or chronic suppuration of the soft tissues of the ear, which has extended to the adjacent bone. It is rarely the result of primary periostitis. Caries of the temporal bone often heals without much loss of hearing if the labyrinth has escaped the attack. Necrosis is much less common than caries. However, nearly the whole temporal bone may be thrown off, and yet the patient survive. The fatal results of caries and necrosis usually are due to purulent meningitis, abscess of the brain, phlebitis of the sinuses, with pyæmia, or to a combination of them all. Death sometimes results simply from œdema of the brain, caused probably by interference in the circulation by a remote phlebitis and embolism. It is now admitted that brain-abscess is the result and not the cause of aural necrosis.[1]

True *tubercles* are rarely found in the petrous bone, but they do exist, as shown by Zaufal.

Malformations of the Auricle.—Complete absence of one or both auricles may be the result of arrested development. There may also be absence of parts of the auricle, or there may exist simply a microtia, the parts being perfect in form. Usually with great deformity of the auricle, malformations in the deeper parts of the ear exist,—*e.g.,* atresia, stenosis, or absence of the meatus or the labyrinth. Such anomalies are referred by Virchow to disturbances in the closure of the first branchial cleft, and are often associated with cleft palate and unilateral atrophy of the face (Schwartze). Sometimes excessive development of the auricle is observed.

Fistula auris congenita of Heusinger is not uncommon, and may be regarded as a remnant of the first branchial cleft. It is characterized by a small opening one centimetre above and in front of the tragus. Sometimes the opening is found in the lobule (Betz). The cicatricial depressions in the skin in the same region are also due to imperfect closure of the branchial fissures. These defects are sometimes associated with other auricular malformations and with fistulæ in the neck. The fistula auris congenita sometimes discharges a creamy matter containing pus.

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[1] Schwartze, Pathological Anatomy of the Ear, p. 17.

Inflammation and its Results.—Erythema, eczema, and intertrigo of the auricle are common in early childhood. The latter is usually seen behind the auricle. Gangrene of a spontaneous nature may be found in nurslings, but is not common (Schwartze). Syphilitic lupus, pemphigus, and congenital ichthyosis are often seen in the auricle. In fact, all skin-diseases which affect the integument near the auricle may also affect the latter. Eczema is the most common affection of the skin attacking the auricle, both in the acute and the chronic form. If allowed to become very chronic, it may permanently thicken and discolor the auricle. One of the most annoying results of eczema of the auricle is the matting of the hair brought about by the discharges from the broken skin. This skin-disease is often due to disorders in the child's digestion, but in most cases the disease is greatly aggravated by the local irritation and interference from the patient's fingers, and the improper management of others.

Very often wearing a cap leads to maceration of the baby's auricle and the side of the head behind it. Intertrigo is the first step, and then eczema. Even in this first stage, the parts should not be washed with soap and water, nor even with water alone. The parts affected may be smeared with bland sassafras or quince-seed mucilage, or, still better, sprinkled with a powder composed of equal parts of Hubbuc's oxide of zinc and starch. The pellicle, or crust, which this forms with the secretions from the eczematous skin, should be allowed to remain, as it protects the inflamed skin and favors healing. If the yellowish crusts of hardened serum get very thick, and must be removed, in the more chronic form, then soften with sweet oil, and gently remove them; but avoid this in the acute stages. In acute eczema the skin must be protected as in burns. Adhesions between the auricle and the side of the head may be prevented by anointing the parts with sweet oil, cosmoline, vaseline, or lanoline. In children who are eating solid foods—in those from four to fifteen years of age—all highly nitrogenized foods must be avoided. In children of the age of ten years I have found chronic eczema of the auricle kept up by eating pies, cheese, sweets, and pickles.

For the treatment of all other skin-diseases of the child's auricle the reader is referred to works on cutaneous diseases,—with this caution, that the various applications to the diseased skin of the external ear must not be allowed to clog the external auditory canal nor to run down upon the drum-membrane.

Boxing the ears, pulling the ears, and swabbing the canal for imaginary wax and dirt must be most carefully avoided. Boxing the ears is apt to produce rupture of the drum-membrane, by the force of the column of air driven suddenly against it. The mere rupture of the normal drum is not as serious an injury as many suppose, as merely the protector of the mucous lining of the drum-cavity has been ruptured, and not an important factor in hearing. Therefore, if this should occur, healing by first intention will generally ensue if nothing is dropped into the ear, and if the meatus is filled with cotton to prevent the ingress of air. The injury to hearing which may attend the rupture of the drum by a blow on the ear is not attributable to the rent in this membrane, but to a concomitant concussion of the auditory nerve. These important differential facts should be borne in mind in the consideration of the medico-legal aspects of rupture of the drum from a "box on the ear." It must also be shown in any asserted case of injury of this kind that the membrana tympani and the rest of the auditory apparatus were in good order before the blow, and also whether immediately after the injury any form of remedy was put into the ear by the patient, his friends, or his physician, as such treatment, by entering the drum-cavity through the hole, would certainly injure the middle ear, and cause the real malady in this important organ.

Pulling the ears is open to nearly as much objection as "boxing" them, since the attachments of the auricle to the auditory canal are of such a nature[1] that traction upon them is communicated to the sensitive fundus of the canal, and even to the membrana tympani. Hence pain and injury are often the result of this rude and culpable manipulation of the ear.

On the other hand, an excessive care of the ear, by washing away the wax from the meatus, often abrades and inflames the concha and the meatus. This should be avoided, as wax is not dirt, and should remain where nature puts it. The superfluous wax will roll out in little crumbs, every day or two, and removes itself by rolling into the concha. But more on this topic will be given when considering the affections of the external auditory canal.

Wounding the cartilage in piercing the lobule for ear-rings sometimes occurs. This is due to the fact that this remnant of barbarous adornment is relegated to jewellers for preparation, and, as they are ignorant of anatomy, they are not aware that sometimes the cartilage of the auricle extends far into the lobule. This is wounded in the piercing and usually causes severe inflammation. The ears had better not be pierced at all.

With the exception of the auricle, the various parts of the auditory apparatus cannot be seen without special manipulation and the use of instruments. Hence a short consideration of the means of examining the auditory canal, the membrana tympani, and in some instances the middle ear, is now in order.

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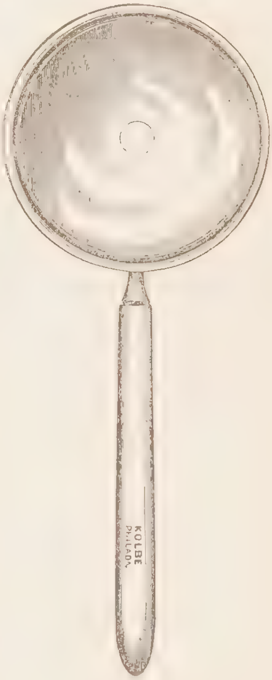
[1] Sexton and Pinkerton.

EXAMINATION OF THE EAR.

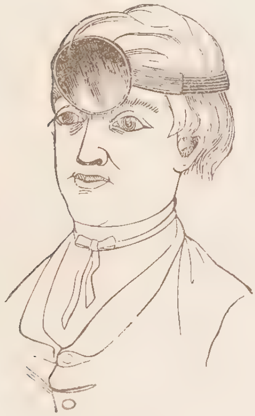
This can be done properly only by means of good reflected light, as it is neither easy nor satisfactory to examine the ear by direct rays of any kind of light, excepting those from an electric light held on the head by means of a forehead-band. Usually the reflected light of day or of a candle or lamp will be found amply sufficient. A speculum or ear-funnel is always necessary, even in infants. Down this speculum the light may be thrown or reflected from either a hand-mirror or a forehead-mirror (see Figs. 3 and 4).

FIG. 3.



Hand-mirror.

FIG. 4.



Forehead-mirror.

The auricle must be drawn slightly upward and backward, while the speculum is directed downward and forward. Care must be taken not to push the speculum too far into the child's meatus, for reasons already given,—viz., the shortness of its auditory canal and the proximity of the membrana tympani to the external meatus. Hence it is easy with a slender speculum to touch and wound the drum-membrane. The speculum must have a diameter of from two to three millimetres for infants, and, as a rule, no child's ear will take a speculum with a diameter of more than four or five millimetres.

If the meatus and canal are filled with secretion, syringe these parts gently with warm water, or mop them out gently with absorbent cotton on a cotton-holder. After syringing the external canal, it should be mopped dry by means of absorbent cotton.

Cerumen collects sometimes, but not often, in the meatus of children. Masses of epidermis and wax also sometimes collect here and form the so-called *keratosis obturans.* It is said by Von Troeltsch that after scarlet fever the external auditory canal may become filled with desquamated epithelium. If so, syringing with warm water will remove the obstruction.

At birth the so-called *vernix caseosa* fills the external auditory canal. The membrana tympani is at this time covered with a thick layer of epithelium or caul, and it seems to lie nearly horizontal, as the auricle and its fibrous canal are pushed upward on the infant's head, which brings the lower wall of the auditory canal nearly, if not quite, in contact with the membrana tympani. Hence to get a view of the membrana the eye must be directed upward, or the auricle must be pulled somewhat downward in order to straighten the canal.

From this peculiar condition of the external ear in the new-born, it is probable that at birth all children hear poorly. This, however, is soon overcome by the lowering of the auricle and the external canal, and by the drying of the aforesaid matters in the canal and their exfoliation and dropping from the ear. They could be syringed out if necessary, which, however, is rarely the case.

Syringing the Ear.—In syringing the ear of a child or an adult, but especially in syringing an infant's ear, the nozzle of the syringe must be larger than the meatus of the ear, in order to prevent the entrance of the instrument into the canal. Hence the so-called ear-syringes which are made with a kind of nipple-like prolongation of the nozzle are especially reprehensible, as they can enter the meatus, and, from their length of half an inch or more, can reach and wound the membrana tympani. That this, and bruising the meatus by this means, are done, the author knows well. Hence the ordinary hard-rubber enema-syringe, No. 2, the so-called male syringe, is not only amply sufficient, but the only safe syringe within the reach of all. With this an ounce or two of warm water may be thrown into an infant's or a young child's ear, and the return current caught on a towel held closely under the ear. To hold a cup of any kind under the ear of a young patient will be found very inconvenient.

THE AUDITORY CANAL.

Following the anatomical order, the next part of the ear after the auricle is the external auditory canal, or meatus. Its peculiar fibrous nature and freedom from bone in the infant have been described. Gradually, as the child grows, the outer surface of the annulus tympanicus, in which the membrana tympani is inserted, grows outward, and we find the indications of the formation of an osseous canal between the auricle and the membrana. This is not complete in development before adult life, though practically, and hence clinically, we must regard the child of five years as having already an osseous external auditory canal. The skin which is reflected from the auricle and lines this canal is exquisitely thin and sensitive, and is held closely to the bony canal by dense, tight fibrous tissue. In fact, the skin lining the external auditory canal is very much like a periosteum.

Malformations may occur in the auditory canal, with malformations elsewhere in the head. The canal may be entirely absent, a congenital condition, with entire absence of auricle and membrana tympani. Also a congenital atresia caused by membranous or osseous tissue may be detected in some instances. Of course such cases are hopelessly deaf-mutes.

Foreign Bodies in the Ear.—This is a subject which is of great importance to the general practitioner, because he is generally the first to see a child who has got something in its ear. Afterwards, the specialist's aid is invoked if, as is generally the case, the manipulation of the ear has been either useless or harmful. Let it be written at the outset in the most emphatic manner that the mere entrance of a foreign substance into the ear is, in itself, of very little importance. In no case has injury to the child ever arisen from the mere presence of a foreign substance, like a bead, a seed, or a button, in its ear. It is the unskilful, rough, and lacerating efforts at its removal which have invariably produced the real injury.

In addition to these statements, let it be remembered that whatever the child has put into its own ear, or had put there by other children, is certainly small enough to come out, and can be removed easily if the methods are understood and there is no swelling of the meatus or canal brought about by previous rough efforts at removal.

No child ever complains of pain from the entrance into the ear of the substances named, or of those similar in form and surface. A rough, jagged substance can never get into the ear, because the endeavor to put it there hurts and causes the child to resist, or desist. I have known beads and small seeds to lie for years in children's ears, without any discomfort.

The first advice, therefore, to the patient—and the family physician too, unless he can make a diagnosis in the case—is to let the ear alone, when a child says there is something in it. Usually at this point the parents get alarmed, run for the first doctor, and frighten him into the belief that something is in the ear. Instead of calming their fears by stating how harmless is the mere presence of such a thing in the ear, and assuring himself by careful examination with mirror and speculum whether there is any foreign body in the canal, he too often proceeds directly to probe the ear or to look in by direct light, and concludes by thinking that he sees something, and increases the alarm of parents and child by dwelling on the serious consequence of allowing anything to remain in the ear. He proceeds now from bad to worse by using improper instruments to remove sometimes an object which is not in the ear at all. In fact, no one but an aurist of experience should ever touch an ear with any kind of metallic instrument, even of the most delicate and special form. At this stage of the case we have sketched, if there is a small bead or seed in the ear, a few syringefuls of warm water will bring out the foreign substance. This I have done, even when the auditory meatus and canal were swollen and tender from antecedent rough handling on the part of others.

First, then, do not be alarmed yourself, and you will allay the fears of the patients' parents.

Second, do not do anything but ascertain whether there is really, as asserted, anything in the ear.

Third, do nothing but syringe the ear for the removal of the object if really there.

Should you not be able to diagnose its presence,—and I trust you will know enough to diagnose between a pearl button and the membrana tympani,—invoke the aid of some one who you think can. Duty to yourself and to your patient commands you to be quick to see your own ignorance in many cases, and you do the best service to your young patient by acknowledging, at least to yourself, your inability to make a diagnosis in a case of foreign body in the ear. You can never injure the patient by gently syringing the ear with warm water. But you dare not do more with your limited knowledge, without incurring the risk of doing the child irreparable injury—perhaps of destroying its life—by your mismanagement. I know many instances where the hearing has been entirely destroyed and the child tortured with pain, not by the presence of the foreign body in its ear, but by utterly unjustifiable efforts at its removal by probes, forceps, etc., in the hands of those whose common sense should have taught them better. It is impossible for any one not a specialist to diagnose positively the presence of a foreign body in the ear, if it has gone beyond the meatus, and of course it follows that he is also unable to remove it, excepting by *warm*-water syringing. Therefore, I have given all the treatment necessary for the successful management of such cases by the general practitioner. If you cannot remove the foreign body by syringing, let it remain in the ear, until the patient can consult an aurist. No foreign body can ever reach the brain, unless pushed there by the hand of the surgeon.

When roaches, fleas, or insects of any kind get into the ear, a few drops of sweet oil, or any fixed oil, will smother them, and relieve the suffering caused by their movements. The removal of the dead insect falls under the same form of proceeding as already given for beads, etc. Use no instruments; for nothing worse than temporary deafness will result from its remaining in the ear, but death may result if the physician should forget his unfitness for such work and attempt its removal by picks, pincers, etc. The sooner this conservative proceeding is followed by the general practitioner in cases of foreign body in the ear of children, the sooner we shall cease to read of the disaster attending the entrance of a foreign substance into the ear, which in reality is a very trivial matter if not improperly treated at the outset.

In some very rare instances maggots have been known to get into the ears of children affected with otorrhœa. If such an accident occur, a drop or two of chloroform or ether will destroy a maggot's life instantly, whereas syringing the ear with warm water only makes the maggot more lively and the pain in the ear more intense.

Reflex Phenomena.—In rare instances epileptiform phenomena have arisen from the presence of a foreign body in the ear. Sometimes paretic symptoms have also shown themselves on the side of the body corresponding to the ear in which the foreign substance lies. Whenever such phenomena not otherwise easily explicable arise in children, the ears should be examined as the possible seat of the cause of irritation. Of course the line of action is plain if a foreign body is found in the ear: it must be taken out. But all the precautions already given as to this procedure must be carefully observed.

Wax in the Ear.—Wax, or cerumen, rarely accumulates in plugs in a child's ear to such an extent as to interfere with hearing. If, however, such masses form, syringing with warm water is all that is required for their removal. All forms of spoons, picks, etc., will hurt the ear, and act just as unfortunately as in attempts to remove any other foreign substance from the ear.

Accumulations of wax in the ear may be softened by instilling into the ear, a few times before syringing, five or ten drops of the following:

℞ Sodii bicarb., gr. xx;
Glycerini, f℥i;
Aquæ, f℥vii.
Misce.
S.—Apply warm to the ear.

Now and then there are found in the ears of children from five to ten years old hard, leathery, or even horny plugs composed of laminæ of epithelium with a little cerumen in the outer end, near the meatus. These plugs quite fill the canal and render the ear totally deaf. Their removal is tedious, and can be accomplished only after continued use of the above-named solvent drops and patient syringing. If this does not accomplish their removal, it must be done by means of special instruments under illumination from the forehead-mirror. It is needless to say that by this time the case should pass into the hands of one specially qualified to treat it. I have known the deafness and pain arising from the pressure of these hard plugs in the auditory canal to be treated for years as due to other causes,—of course, without benefit. Finally, when the true cause was found out by one able to make an examination of the ear and a diagnosis, restoration to hearing and health soon followed.

Both these accumulations—of wax and of laminæ of epithelium—often owe their beginning to the efforts of parents and nurses at cleaning the ears of children. It is a mistake to regard wax as dirt, and a greater error to make efforts at its removal from the auditory canal. Some wax is needed for the protection and comfort of the ear, and the superfluous wax will roll out into the concha every day or two, and can be easily removed from that part of the ear. If, however, a swab or any form of spoon is used for removing wax from the canal, as much as, or perhaps more than, is removed by such implements will be pushed into the canal and gradually packed down upon the drum-membrane. At the same time more or less abrasion of the delicate skin of the canal occurs. When wax and pieces of epithelium are pushed down upon the drum, deafness is soon the result, of which the very young child is unconscious, the natural escape of flakes of dead skin and pieces of wax from the ear is interrupted, and as more epithelium is thrown off behind the mass the latter gradually grows to one of the aforesaid keratosis plugs. Beneath these plugs sooner or later maceration and ulceration take place, polypi spring up, and the membrana tympani may be eroded and tympanic inflammation finally result from improper efforts at a toilet of the ear. Furthermore, the presence of these plugs of wax and skin, when they have begun to press upon the skin of the canal and irritate the external ear and the membrana tympani, may induce epileptiform phenomena in any one, but especially in children.

It is certainly wise, therefore, to refrain from any toilet of the ear, which is not only not required but may even lead to a direful train of phenomena.

Aspergillus.—If the wax is removed from the external auditory canal, the fungus called the aspergillus, a variety of mould, may grow in the fundus of the ear upon the membrana tympani. The spores of the plant do not flourish in the presence of ear-wax, as I have tested by experiment, but readily enter the ear and grow upon any morbid secretion found there if the wax has been wiped away and if in so doing the patient or the attendants have abraded the skin and permitted the escape of a little blood or serum into the canal. This furnishes the fitting soil, and soon the spores of the aspergillus sprout from it, form a mycelial web upon the drum-membrane, and bring about a dermatitis of the fundus of the canal and the membrana tympani.

The ear now becomes painful, deaf, and from it a watery discharge soon ensues. Syringing with warm water will generally remove the false membrane and the spores, after detachment sets in, which usually takes place soon after the discharge appears. In these cases, again, successful treatment depends much upon the correct diagnosis at the outset; otherwise no treatment is of value. Any oleaginous or greasy application is injurious, because it favors the growth of the fungus, and the ear goes on to a condition of painful eczema in little children, with many disagreeable symptoms from the nervous irritation so easily set up in the ear, and felt in the general nervous system.

There are many drugs which have been cited as destroyers of the aspergillus, but I have found only one that is prompt and painless as well as efficient,—viz., salicylate of chinoline, one part to sixteen parts of boric acid. This powder should be insufflated into the ear after all the easily-detachable pieces of the false membrane have been removed by warm-water syringing. Usually one application of this powder to the affected membrana tympani destroys the aspergillus and cures the disease.

Otitis Externa Diffusa.—This name is applied to the diffuse inflammation attacking the skin of the auditory canal as a result of the irritation arising from the ingress of improper medicaments, cold air or cold water, from picking and swabbing the ear, and also from the continued presence of the fungus aspergillus. Direct violence, from putting snow in the ear in rude play, blowing into the ear, and subjecting the child to sudden changes of temperature, will also have to be held accountable for this disease, in many cases. It is a very painful affection, and, by its tendency to involve the subcutaneous tissues and even the periosteal lining of the osseous part of the auditory canal, it assumes very often all the features of a periostitis. The skin rapidly becomes red and swollen, and, from its confined position in a cartilaginous and osseous canal, is thrown into several thick folds or ridges, which uniting in the centre of the canal soon obstruct all view of the drumhead and render the patient hard of hearing. Movement of the auricle by the hands of another becomes very painful at this point of the disease. Tinnitus is also complained of, as well as intense pain. Several days usually elapse, with all these painful annoyances to the patient, before secretion sets in. Then the skin often exudes, at first from several points, a bloody serum, followed in a day or two by a purulent discharge. The quantity of serum discharged in such cases is often very copious, wetting a number of towels or cloths in the course of twenty-four hours. Sometimes the inflammation may extend to the membrana tympani, and involve it, so that perforation ensues and mucus is found in the discharges of the ear.

Treatment.—When seen in the first stages, while the skin of the canal is swollen and tender, the best treatment is to make one or two deep incisions, down to the bone if necessary, into the congested skin. This will often cut short the disease; but the method is painful. The next best means of relief will be to apply a dossil of cotton moistened with the following mixture:

℞ Black wash, f℥i;
Glycerin, fʒi;

or with a fifteen-per-cent. solution of ichthyol in water. I have known each of these to abort both the circumscribed (furuncles) and the diffuse form of otitis externa. These applications usually control pain much better than morphine drops, cocaine, or atropine, which are in my experience nearly impotent to allay pain in such cases of earache. If, however, suppuration is fully established, the ear must be gently syringed with weak salt-and-water, warmed, or with boric-acid solution, or with a two-per-cent. solution of carbolic acid, or with plain warm water, and then gently mopped with absorbent cotton; and, if the acute stage has fully passed and the ear is no longer sensitive to touch, boric acid in fine powder, or boric acid seven parts and iodoform one part, may be insufflated. If the ear is thus cleansed once or twice daily while the discharge is copious, and then once a day or every second day as the discharge diminishes, the organ will soon heal. But all fats, oils, vegetable matters, and poultices must be kept away from the ear, at this time and at all others. They only macerate the tissues of the ear, promote granulations, and cause breaking down and sloughing of the fundus of the canal and the membrana tympani.

As the discharge ceases, granulations or small polypi may be seen on the walls of the canal and on the membrana tympani, while the latter may be found perforated. The granulations, if not yet pedicellated, will generally disappear under the use of the powders named above,—under the so-called dry treatment. If they do not, they may be removed, under proper illumination of the canal, with forceps or snare. The perforation also generally closes under proper treatment as just marked out, and the hearing becomes normal. Unfortunately, as soon as the earache stops the ear is often neglected and allowed to remain filled with decomposing and irritating matter, until a chronic otorrhœa is established.

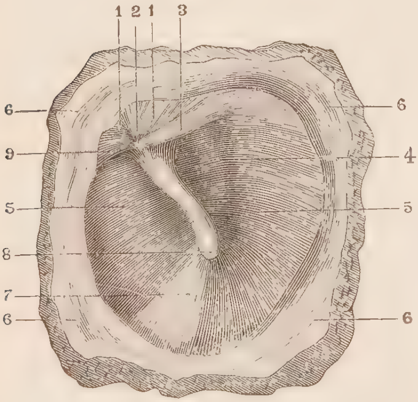
Effect of Teething on the External Auditory Canal.—During teething the external auditory canal may become congested and painful. Not uncommonly the engorged vessels are relieved by an escape of pinkish serum, or suppuration may ensue, after considerable fretting or great crying on the part of the child. This crying is too frequently referred to the gums,—which, in fact, are rarely the seat of pain. If the ear is found congested or inflamed by this cause,—the irruption of a tooth,—dry heat applied to the external ear of the little patient gives great relief and favors resolution of the congestion. The simplest, and often most efficient, means is a bottle of hot water held against or in front of the auricle.

The effects of diseased teeth are often reflected upon the skin of the auditory canal and tympanic cavity of large children and adults. The reflex effect of dental irritation upon the drum-cavity and the membrana tympani of infants will be considered when discussing diseases of the drum-cavity in children.

MEMBRANA TYMPANI.

This part of the conducting auditory apparatus is of the same size in a child as in the adult. Its general appearance is represented in the accompanying wood-cut (Fig. 5). This is the air-tight boundary between the auditory canal and the middle ear. It serves the double purpose of protecting the latter from the external air, and of broadening the surface of the handle of the malleus, the great factor in the leverage of sound-waves upon the other ossicles and the fluid of the labyrinth.

Fig. 5



Outer Surface of the Drum-Membrane. Magnified 3½ times. (Politzer.)—1, 1, the flaccid part of the drum-membrane; 2, the short process of the hammer-bone; 3, back fold of the drum-membrane; 4, the long limb of the anvil-bone, shining through the membrane; 5, 5, the true membrana tympani, or membrana vibrans; 6, 6, 6, 6, inner end of bony canal, forming frame for drum-membrane; 7, the pyramid of light; 8, lower part of the hammer; 9, front fold of the drum-membrane.

Congenital malformations of the membrana tympani are sometimes seen in early life, as a small opening in the upper part of the drum-membrane over the short process of the malleus, the so-called foramen Rivini. This is an arrest of development or a failure to close, like hare-lip, coloboma, etc. If such an opening in the membrana of a child is observed, care should be taken to avoid the entrance of water into the ear, as it might inflame the drum-cavity, should it find its way into this space.

Appearances.—Owing to the thick dermis of the membrane in children, their membranæ rarely possess the lustre of the adult's drum-membrane. In general terms, it may be said that inspection of the membrana tympani of an infant or very young child, by means of the so-called speculum or ear-funnel and a hand- or forehead-mirror, reveals at the bottom of the fundus a grayish or pearl-colored, circular, membranous diaphragm, with a lighter-colored ridge running in one of the radii from in front and above backward and downward. The latter is the handle of the hammer-bone. It terminates above at the so-called short process, a prominent knob, and below it ends at the umbo, or central depression of the membrana tympani. Backward and forward from the short process of the hammer, or malleus, to the periphery of the membrane run the so-called folds of the membrana tympani, and above these folds lies the membrana flaccida, or Shrapnell's membrane. This is free from fibrous tissue, and is composed of the skin layer of the drum-membrane and the mucous membrane of the attic of the drum-cavity, which here come together and form a loose membrane in the so-called segment of Rivinus.

The pyramid of light, a reflection found in the lower anterior quadrant of the drum-membrane of larger children and adults, is not found always in infants. It is plainly visible, however, at two or three years of age.

The membrana tympani forms an hermetical diaphragm between the external and the middle ear. It acts as a protector to the mucous membrane of the middle ear, and undoubtedly augments the leverage of the malleus, in the chain of ossicles, by its expansion about the handle of the hammer-bone, which is thus enabled to catch sound-waves which fall upon its comparatively broad surface. But, as perforation or even large destruction in the membrane does not appear to affect the hearing in numerous cases, it cannot be considered a very important factor in the chain of sound-conductors. It probably acts as a supporter to the malleus and enables it to maintain itself from locking too firmly with the incus.

Not only the cutaneous surface but also the mucous layer of the membrana tympani of a young child is thick and highly developed. Hence its vascularity is readily augmented by irritants from without and within, and a myringitis, or inflammation of the drum-membrane, is excited at this age more easily than in later life.

Diseases.—The diseases of the membrana tympani may be named as follows: traumatic perforation, acute inflammation, chronic inflammation, with or without perforation, and myringitis from the growth of aspergillus in the fundus of the canal. Being composed of skin from the external canal and mucous membrane of the middle ear, it readily partakes of the affections of these parts, but rarely is the seat of a purely idiopathic disorder. A strict myringitis is, in fact, very difficult to define, yet clinically it has practical existence.

Traumatic perforation of the drum-membrane from "boxing" the ear has been considered under Injuries of the Auricle, to which the reader is referred (p. 5). The other forms of traumatic perforation occurring in children are due to the accidental thrusting of slender objects, like pencils, knitting-needles, pens, etc., into the canal and through the membrana. In addition to the wound of the membrana, injury may be done to the ossicula or even to the internal ear through the oval window, by the penetrating instrument.

If only the membrana tympani is perforated, the same cautious proceeding must be observed as to avoidance of putting anything into the canal, as was recommended when speaking of boxing the ear. At the same time the meatus of the canal must be protected by cotton, and the air thus kept from falling directly upon the exposed mucous membrane of the drum-cavity. If no traumatic inflammation ensue, the wound in the drum-membrane will heal, often in a few hours. If the penetrating wound has dislodged or injured any of the ossicula, a serious result may be looked for. And if it has extended to the internal ear, cerebral symptoms soon show themselves, and are followed sooner or later by profound deafness. As rarely anything more than a perforation of the membrana occurs from the violence alluded to, it will not be necessary to refer to the graver complication of injury to the ossicula or the internal ear, except to say that, if it occur, only a skilled aurist can manage it.

Sometimes the membrana tympani is ruptured by diving and by loud and sudden noises, and sometimes, though rarely, by coughing.

The perforation looks like a red line, or a gaping slit, either before or behind the malleus. The latter is, as a rule, larger and more plainly visible than the former. The perforation whistle can always be heard if the Eustachian tube is pervious. There is generally a little pain at first.

Perforation of the Flaccid Membrane.—Sometimes a perforation is detected in the flaccid membrane above the short process of the malleus, accompanied by little or no discharge. This may be due to erosion, from a foreign substance in the external auditory canal. More frequently, however, such a perforation gives exit to a discharge of offensive pus, and indicates grave, purulent disease in the upper part of the drum-cavity,—in the so-called attic of the drum. This form of tympanic disease will be considered when discussing affections of that part of the ear.

DISEASES OF THE MIDDLE EAR.

Diseases of the middle ear are the most numerous of all aural diseases, are especially common in childhood, furnish most of the cases of so-called "earache" among the young, and lie at the foundation of nearly all cases of permanent deafness.

Pathology of the Middle Ear.—We find here also congenital malformations, as, for example, an abnormally small tympanic cavity, absence of the fenestræ, one or both, or contraction of them by hyperostosis. The ossicles of hearing may be absent, or abnormal in size. The stapes is said to be most frequently at fault. (Von Troeltsch.) Malformations of the middle ear are usually associated with malformations in the external ear.

The Eustachian tube is rarely malformed. A mastoid cavity is found in children, but it is very small and its cortex is filled with natural dehiscences. This grows until puberty, when it is fully developed (p. 2).

The tympanic cavity of a new-born child does not contain air if the child has not breathed. It is not filled with mucus, but with a thick mucous membrane in a hyperplastic state. In the first years of life the middle ear is more disposed to disease than later. In early childhood the dura mater and the tympanic mucous membrane are more closely connected than in later life. This is effected by the direct tissue-circulation between these parts through the petro-squamous suture. Hence affections of one region influence the other.

The middle ear is further inclined to disease because of the connection between the Eustachian tube and the naso-pharynx. The latter region in children is very susceptible to "colds," and these effects are easily communicated thence to the middle ear.

The naso-pharynx is very rich in blood-vessels and glandular substance. Lying between the pharyngeal mouths of the two Eustachian tubes is the so-called *pharyngeal tonsil.* These vascular and glandular structures become congested and abnormally active in coryza and in the exanthemata. Then the Eustachian tubes become closed, aeration of the drum-cavity ceases or is greatly impeded, a vacuum is formed in the tympanum, and the membrana tympani is pushed inward by the external air. Swollen palatine tonsils and naso-pharynx act in the same way, by inducing stasis of circulation near the Eustachian tubes, and thus maintain an irritation in this delicate region.

Unimpeded nasal respiration is of the greatest importance, as thus aeration of the middle ear is maintained. The naris is often impeded for a long time before it is discovered, and the ear on that side becomes deaf before the cause is found out. The quality of the air respired is most important, as each respiration, swallowing, gaping, or sneezing forces air into the middle ear, if the Eustachian tubes are not tightly closed. If they remain closed long, extravasation occurs in the drum-cavity, which can, however, be removed by inflation or by paracentesis. When the nose is stopped, air is more readily forced from the naso-pharynx into the Eustachian tubes and middle ear than when the nares are free. The same condition prevails in expiration, coughing, or swallowing when the nose is stopped. Hence, when the child sucks the breast, swallowing with its nose stopped and its mouth closed by the teat induces in its middle ear what is known as the negative effect of aeration of the naso-pharynx,—viz., an exhaustion of air from the tympana; the membranæ are then drawn inward, causing subjective noises, discomfort in the ear, and even pain and deafness.

The same condition of the nares which thus favors the entrance of air from the fauces into the Eustachian tubes may also favor the entrance of water. Hence vomiting, as in whooping-cough, may force the contents of the stomach into the middle ear, and cause otitis media. Mucus also may be thus forced into the Eustachian tubes and middle ear.

Pus in the tympana of very young children (from two days to two weeks old) may be regarded as physiological and due to metamorphosis of the mucous cushion alluded to on p. 17. Dissections of the ears of children in their first year indicate that inflammation and exudation in the middle ear are very common in early life.

The most usual form is the acute catarrhal otitis media, which may develop into the acute purulent, or may assume the form of chronic catarrhal otitis media without purulency. But no sharp line can be drawn between acute otitis media catarrhalis, which leads to rupture of the membrana tympani, and otitis media purulenta acuta, since when perforation of the membrana ensues some pus will be found in the discharge from the drum-cavity, though the quantity may be so slight and so evanescent as to permit the disease to be named a catarrhal one, with hypersecretion of mucus as its chief result.

OTITIS MEDIA CATARRHALIS ACUTA.

A very young child with a severe coryza and stopped nares is practically in the first stage of otitis media catarrhalis. Very often infants in this condition cry greatly, but the true cause of their discomfort is not known. Usually only one ear is attacked at a time. If a discharge of mucus or pus occur in a few days after the great crying-spell, then it is recalled that the earache may have been the cause of the child's lamentation. Very often, however, the pain in the early stage of catarrhal otitis media is not great, the congestion in the tympanum soon undergoes resolution, no discharge comes from the ear, and it is never known that the ear has been the seat of disease, unless the hearing is dulled by the attack and the child is old enough to permit detection of this altered function. This failure in hearing soon passes off, and many cases never show any subsequent defects in hearing.

On the other hand, with a coryza the child may soon begin to cry bitterly, and if a year old will put its hand to its ear, indicating the seat of pain. When able to speak, it will state that its ear aches. If suppuration does not ensue, the pain is relieved either by proper treatment or by a rupture of the membrana tympani and an escape of sero-mucus.

Treatment.—When it is discovered that a child with coryza—for it is such who are usually attacked with this form of ear-disease—is suffering from earache, an endeavor should be made to free its nostrils and open the Eustachian tubes, for it is the swollen state of the latter and the vacuum formed thereby in the middle ear which cause the pain. If the tubes are not opened and air thus allowed to re-enter the tympanum, passive exudation ensues in the drum-cavity and soon leads to rupture of the drum or further complications in the middle ear.

A camel's-hair pencil anointed with sweet oil or cosmoline may be passed gently backward towards the fauces,—not upward towards the frontal sinus. This will promote sneezing or coughing and tend to open the Eustachian tubes. Still better, inflation of the tympana by Politzer's air-bag should be done. With infants and very young children this is a simple and efficient operation. It is required simply to place the nose-piece in the nares, and inflate. No swallowing of water nor any other effort on the patient's part is required, as it generally cries, and this lifts the velum and shuts off the nares from the pharynx. A gentle puff from the air-bag will now usually inflate the tympana, and often banish the earache. It must be done, however, very gently in the acute stage, or the ear is made more painful by the concussion it receives.

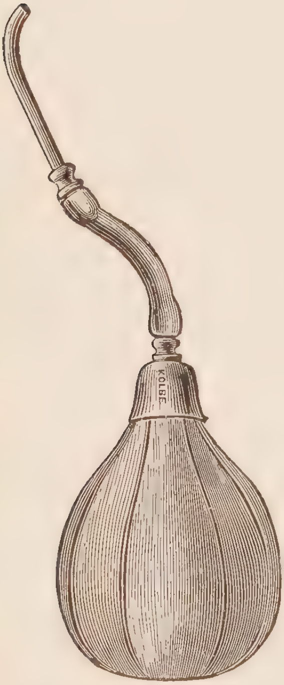
The pain may often be entirely relieved in a short time by the application of dry heat to the auricle or in front of the ear. A four-ounce bottle filled with hot water and held to the aching ear I have known to relieve soon, and I have seen the child fall asleep to wake with no further ear-disease. A hot stone, a hot salt-bag, a hop-pillow, or a hot-water bag will accomplish the same.

But beware of dropping anything into the ear in this stage of the disease, or of putting anything moist, resembling a poultice, in or about the ear. If you doubt this, just apply to your own well ear any of the great variety of domestic remedies so often ruthlessly applied to an inflamed ear, and you will perceive how quickly an acute otitis externa is excited, and how often this is added to the catarrhal inflammation in the middle ear. From my own experience I believe that very few catarrhal cases would ever pass into any very painful or serious stage if they were properly treated at the outset. It is certainly a fact that many are made worse by what is improperly and ignorantly put into the external ear. It does not reach the inflamed middle ear, but it inflames the external ear and increases the general congestion and pain in the organ. When I have seen a catarrhal otitis media at the outset and have had the control of all the treatment, I have never known the inflammation to fail to yield promptly to treatment. On the other hand, I have seen what was at the beginning a simple and manageable disease turned into a very painful and sometimes chronic one by improper treatment.

Politzer's Method of Inflation: the Air-Douche.—As this method of inflating the tympana is frequently mentioned in these pages, it should be said that it consists in blowing air through the nostrils into the naso-pharynx by means of a soft rubber bag made for the purpose, and held in the hand of the surgeon. (Figs. 6 and 7.)

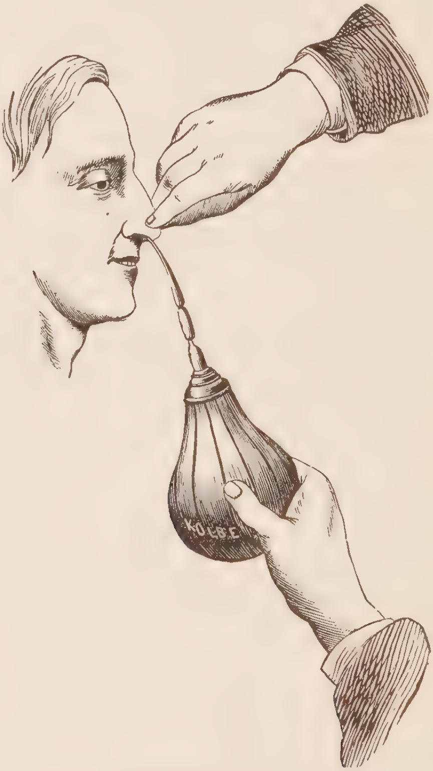
The nose-piece being inserted into one nostril, the other nostril is closed by pressure with the fingers of the surgeon. Then the ala of the nostril in which the nose-piece is, is gently pressed in front of the nose-piece,—not down upon it. Then the patient, having previously had some water given him, is told to swallow. This act lifts the velum, shuts off the naso-pharynx from the pharynx, and opens the Eustachian tubes. If at this moment air is thrown into the naso-pharynx by squeezing the air-bag, inflation of the tympana takes place. Instead of swallowing water, distention of the cheeks with air will accomplish the same object.

Fig. 6.



Politzer's inflation-bag.

Fig. 7.



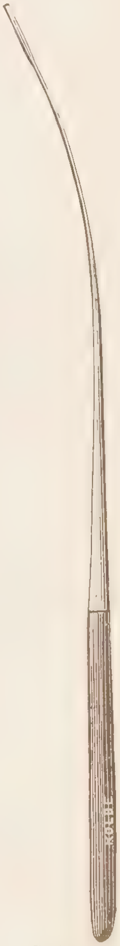
Application of the inflation-bag.

In very young children neither method can be used. Sometimes simple inflation by means of the air-bag, without any aid on the child's part, will force air into the tympana. Crying on the part of the child, by lifting the palate, will greatly aid inflation.

In addition to the measures for relief already named, the child must be kept warm, and, if possible, in the same room, while the ear is at all painful or sore. The temperature of the room must not be below 70° F. The patient must be kept warmly dressed, and, if able to walk, must be kept off the floor, if the weather is cold. A mild sudorific may be given, and the food must be light. Such management will usually bring about resolution of the inflammation in the naso-pharynx and middle ear in the course of a few hours or a day. The dulness of hearing may remain for several days, and, if so, Politzer's inflation should be performed once a day until the hearing improves, and less frequently as the hearing further improves.

Sometimes, however, though the pain may be made to abate, the exudation in the tympanum may be sufficient to cause rupture of the membrana tympani, and a yellowish mucus or muco-pus will be found escaping from the meatus into the concha of the auricle or even out upon the cheek. The disease has now become subacute, and may undergo a spontaneous cure in a few days, as evidenced by cessation of the discharge and improvement in hearing,—the latter being discernible only in children who can talk,—or it may pass into *chronic muco-purulent otitis media.*

Fig. 8.



Cotton-holder.

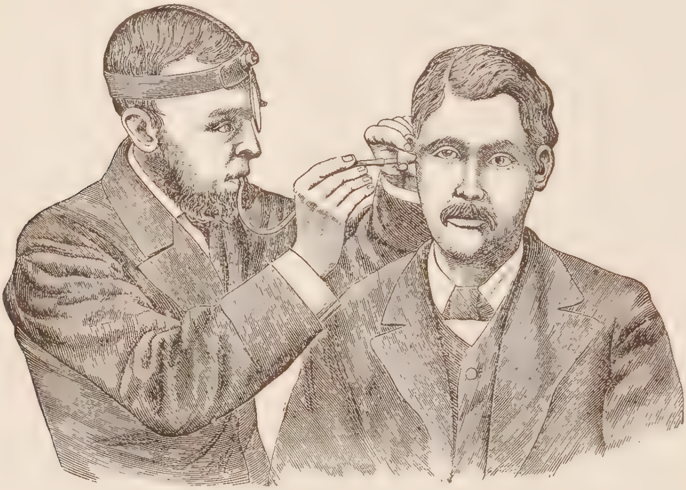
The ear should now be mopped with absorbent cotton on the cotton-holder, or syringed with warm water, in order to remove all septic matter. After such cleansing one of the following powders may be blown gently into the ear: finely-powdered boric acid; or iodoform, one part, and boric acid, seven parts; or boric acid treated with calendula or hydrastin. This treatment should be carried out every day if the discharge is copious, and less frequently as the discharge diminishes.

It is well to state here that the boric acid is combined with calendula or hydrastin in the following manner. Mix tincture of calendula and boric acid together, minim for grain, and dry over water-bath. Then repowder the dried mass and mix it with one or two parts of boric acid as desired. The hydrastin-boric-acid powder is prepared in the same way.

This treatment, if begun at once, will usually check the discharge from the ear in a week or two. If the discharge is neglected, a cure is effected much less rapidly. The cure is also hastened by the use of Politzer's inflation-bag. When the discharge has ceased, the membrana may still be found perforated or it may have healed. The perforation in the mucous form of otitis media is usually smaller than in the purulent forms.

The insufflation of boric acid in any of its combinations must not be done while the ear is tender or painful, as such treatment undoubtedly increases the pain. Its use is indicated, however, as soon as the pain and soreness leave the ear. In fact, during the painful stage of otitis media the less put into the ear the better it is for the patient. Before the membrana ruptures or is incised by the surgeon, the local application of dry heat and the systemic treatment are all that can be done. Some have obtained good results from the administration of small doses of tincture of aconite root. Locally cocaine has been vaunted by some, but I have gained no aid from it in acute otitis media. As I have already said, the catarrhal cases, if seen soon and before various improper home remedies have been applied, yield to the application of dry heat to the painful ear. The hot-water bag, the bottle filled with hot water, a hot stone in flannel, a hot sand-bag, a hot salt-bag, or a hot cushion of dry hops will give the best aid and certainly do no harm. Moist applications in the form of poultices macerate too much and tend to harm the ear, just as they do when applied to the eye. One or two Swedish leeches applied near the tragus or beneath the auricle will often cut short an acute catarrhal process and prevent the formation of muco-pus.

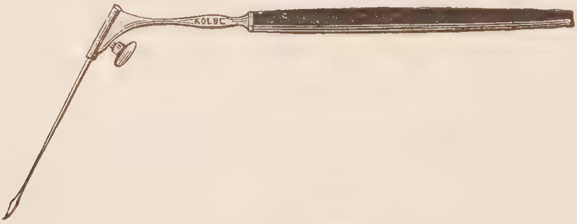
FIG. 9.



Author's method of insufflating powder into the ear.

Paracentesis of the Membrana.—Even in the catarrhal cases of otitis media, paracentesis of the membrana may be performed,—though this operation is more likely to be demanded in the purulent form of otitis media.

FIG. 10.



Paracentesis-knife.

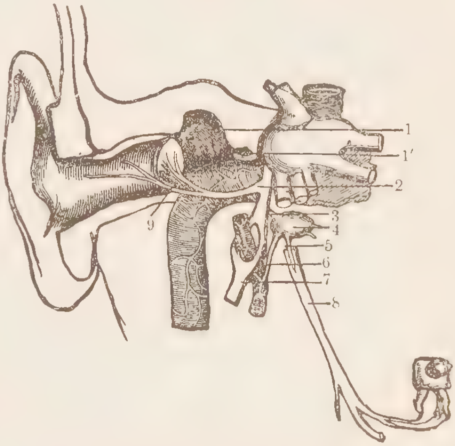
This must not be attempted, however, unless the surgeon can illuminate the ear with the forehead-mirror and conduct the delicate paracentesis-knife down to the bulging membrana. An incision may then be made in that part of the drum-membrane which is most distended by the products of inflammation in the drum-cavity. Such an operation usually gives speedy relief to the sufferings of the patient.

OTITIS MEDIA PURULENTA ACUTA.

In many instances the catarrhal form of otitis media cannot be relieved by any of the remedies I have indicated, but passes into *otitis media purulenta.* The pain in these cases will have defied all measures adopted for its relief, and terminates only when the membrana tympani ruptures spontaneously or is incised by the knife. If pus forms, it can be seen behind the membrana, and the latter will usually bulge outward. It is this form of ear-disease which usually causes the distressing earache of children. In very young children the pain is so intense as to cause convulsions. In infants a coryza or teething will bring on this disease in the ear. In larger children the exanthemata, exposure to inclement weather, or playing in the snow, or in summer excessive bathing in cold water or rapid cooling of the heated body by lying on the ground, will cause *otitis media.*

In scarlet fever or measles this disease may come on insidiously without much pain. In such cases it seems to be of a chronic nature from the outset. The perforation is usually large, as the membrana tympani sloughs easily in the exanthemata, especially in scarlatina.

FIG. 11.



NERVOUS CONNECTION BETWEEN THE TEETH AND THE EAR. (Woakes.)—1, tympanic cavity; 2, auricular branch of auriculo-temporal nerve; 3, branch from the ganglion furnishing vascular nerves to the internal carotid artery and its branch the tympanic artery; 4, otic ganglion; 5, branch from otic ganglion joining inferior dental nerve; 6, middle meningeal artery; 7, auriculo-temporal nerve; 8, inferior dental nerve to teeth and gums; 9, short tympanic branch of internal carotid artery.

The mode of reflex action concerned in the purulent otitis media of teething deserves our consideration. "A considerable portion of the blood-supply of the membrana tympani is derived from an artery that leaves the internal carotid in the carotid canal, and proceeds by a very short course directly to its destination. Being thus closely connected with a large arterial trunk, this small tympanal branch (Fig. 11 at 3) of the internal carotid artery possesses very favorable circumstances for a speedy augmentation of its blood-supply. Now, the nervi vasorum constituting the carotid plexus at this part of its course come largely from the otic ganglion (Fig. 11 at 4). The third branch of the fifth nerve is cut through in the diagram to show this ganglion. On the other hand, the inferior dental nerve (Fig. 11 at 8) supplying the decayed tooth or gums, as the case may be, also communicates with this ganglion (Fig. 11 at 4, 5). We thus arrive at a direct channel of nerve-communication, through the otic ganglion, between the source of irritation, the tooth, and the vascular supply of the drum-head. The effect then of the irritating impression proceeding from the decayed tooth or swollen gums will be to excite waves of vessel-dilatation in the correlated area, the drum-head. Its vessels now become largely distended, acute congestion is thus established, with its attendant stretching of the sensitive and tense tissue in which it occurs, and so occasions the pain experienced by the subject of these conditions. If the irritation be sufficiently prolonged, effusion into the tissues ensues, which under favorable circumstances will pass into suppuration and constitute a true otorrhœa. Owing to the free inosculation of the vessels of the drum-head with those supplying the tympanic cavity, it will not be long ere this region participates in the inflammatory process, so that this cavity may also become filled with pus or muco-purulent fluid." (Woakes.) Of course this accumulation must either escape by the Eustachian tube, as it can very easily in children, from the comparatively large size of this tube in them, or it ruptures the membrana and runs out at the external auditory meatus. Before discharge takes place from the drum-cavity, the pent-up matter may press upon the fenestræ and thence upon the contents of the inner ear, and may excite convulsions.

Before suppuration ensues in the drum-cavity, inflammation may extend from the drum to the meninges of the brain, by the way of the petro-squamosal suture, through which a fold of dura mater dips into the tympanic cavity and unites with the muco-periosteal lining of the latter. This fissure is wide and the portion of dura mater entering the tympanum through it is large in infancy. Towards adult life this fissure becomes narrowed or obliterated, but the vascular connection between the drum-cavity and the brain continues.

Treatment.—Since *otitis media purulenta* begins as a catarrhal inflammation, the remedies suggested for the relief of the latter malady (pp. 18–20) may be applied in this.

But I cannot too urgently object to the use of poultices or hot drops of tinctures and acids in this disease. By such procedure the ear is usually made worse, as an artificial otitis externa or a myringitis is brought on, and the original disease is masked. Very often this is the condition of the ear when the physician is called to the patient, because of the indiscriminate use of a host of senseless household remedies. Examination of the membrana tympani reveals a bulging either below or above the folds, in the membrana flaccida, rarely in both regions. In either condition it is best to perform paracentesis.

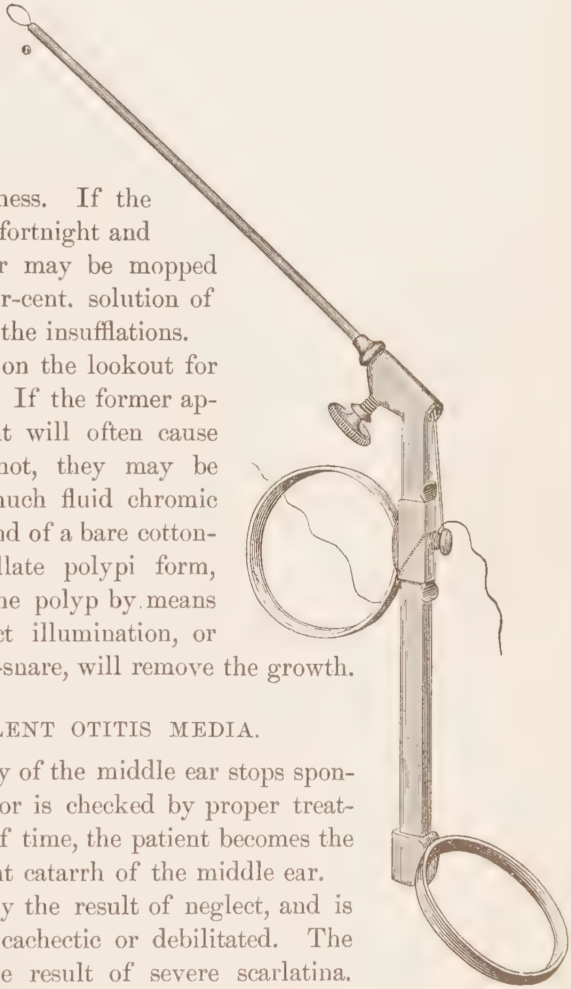
There are no "drops" which can relieve earache in children. If dry heat, inflation, or treatment of the inflamed nares will not do it, nor rest in bed in a warm room with antiphlogistic and sudorific treatment accomplish it, only the escape of pus, either spontaneously or by means of the paracentesis-knife, will give the desired relief.

After the discharge of pus has set in, the ear must be gently mopped with absorbent cotton, once or twice daily, or oftener, to keep it clean and dry.

This is preferable to the syringe. If, however, the discharge is copious and tenacious, the ear may be syringed at the beginning of the discharge, but not as the discharge diminishes. After the ear is cleansed, insufflations of boric acid alone or of boric acid and iodoform combined may be employed, if the ear has lost all pain and tenderness. If the discharge keeps up for a fortnight and is very purulent, the ear may be mopped with a two-and-a-half-per-cent. solution of carbolic acid, previous to the insufflations.

The surgeon must be on the lookout for granulations and polypi. If the former appear, the above treatment will often cause them to disappear; if not, they may be gently touched with as much fluid chromic acid as will cling to the end of a bare cotton-holder. If true pedicellate polypi form, either gentle torsion of the polyp by means of a probe, under perfect illumination, or snaring it with the polyp-snare, will remove the growth.

Fig. 12.



Author's polyp-snare.

CHRONIC PURULENT OTITIS MEDIA.

Unless acute purulency of the middle ear stops spontaneously in a few weeks or is checked by proper treatment in an equal period of time, the patient becomes the subject of chronic purulent catarrh of the middle ear.

This disease is usually the result of neglect, and is easily established in the cachectic or debilitated. The majority of cases are the result of severe scarlatina. Measles and intense coryzas at the time of teething furnish their share also.

If the canal is examined, it will be found either partly or entirely filled with pus and, in the earlier stages, strings of mucus from the Eustachian tube. I would say here that a discharge from the ear, especially if a copious one, is *prima facie* evidence of the existence of a perforation in the membrana tympani. After this discharge is removed from the canal, either by syringing with warm water or by mopping with absorbent cotton, the membrana tympani may be seen. Inspection will reveal that the membrane is perforated, usually at one point only, and that in the lower and hinder part. The perforation is sometimes large enough to permit a view of the red mucous membrane of the inner wall of the drum-cavity beyond.

Very often the entire membrana is gone, only the peripheral annulus tendinosus and the malleus being left. The latter is indrawn, and its lower end is seen lying against the promontory. If the perforation is smaller, the malleus will be seen to be entire and in normal position. The remnant of the membrana is then macerated, and in some cases denuded of its epithelium. In other cases the dermis of the membrane is intact, but thickened and white. If the perforation is large and extends far into the upper and posterior quadrant, the incus-stapes joint may be seen easily. Polypi are often found in this form of ear-disease, extending beyond the plane of the membrana tympani, outward into the external auditory canal. Their protrusion in this direction leads often to the idea that they are attached to the walls of the auditory canal; but, in reality, they originate from the mucous membrane of the drum-cavity, either from that on the inner wall or from that on the inner surface of the membrana tympani. The lining of the auditory canal is skin, and not mucous membrane. Hence polypi do not readily originate from it.

Treatment.—If in a case of chronic purulent otitis media a polypus is found, it should be extracted before any attempt is made at checking the discharge. In fact, a discharge cannot be checked while a polypus is in the ear. A polypus may be removed by delicate forceps, or it may be twisted off its stem by the use of forceps, but the surest way is to snare it off by means of the polypus-snare (p. 25). This instrument should be very slender, so that it can be passed down the canal to the polypus under the eye of the operator. This can be done if the instrument is slender enough to permit plenty of light from the illuminator to pass into the canal at the same time. A fine brass piano-wire, or the fine brass wire used by saddlers, is better than steel or silver wire, because it is more pliable and the bright color enables the operator to keep sight of the loop which he is endeavoring to place over the polyp. After the polyp is removed from its pedicle, the latter should be touched with a minute quantity of fluid (*i.e.*, deliquesced) chromic acid. Just as much as will cling to the end of a bare metal probe will suffice for the purpose of cauterizing the pedicle. The hemorrhage is inconsiderable in all cases. If neither the wall of the auditory canal nor the mucous membrane of the drum-cavity be touched, the patient will experience no pain. In fact, as the polyp has no sensibility, if the parts named be not touched, the patient will not know that anything is being done to him. After the removal of the polyp and the cauterization of the pedicle the ear may be insufflated with finely-powdered boric acid, alone or combined with iodoform as previously suggested (p. 21).

After the removal of a polypus from the ear the discharge usually stops, —sometimes even without further treatment. However, some form of treatment is usually demanded for a few weeks. If the discharge does not then cease, we may suspect that a concealed polypus exists in the upper part of the tympanic cavity,—the so-called attic.

The best treatment for checking the discharge after the polyp is removed is the dry form, by the powders named. Sometimes strong solutions of nitrate of silver may be required. Nothing weaker than fifty grains to the fluidounce of water will be of avail, and solutions of one hundred grains are often most efficient. These must be put into the ear by the surgeon,—*not* by the patient or an attendant. It is not necessary to neutralize them by salt water. Let the ear be gently and thoroughly syringed with plenty of tepid water a few moments after the application has been made. If this treatment has not a decidedly good effect after two or three applications have been made, it should be discontinued, or stronger solutions tried, even up to saturated solutions. But, of course, these must be arrived at with caution: the last-named strength will be needed rarely.

If fluid applications are demanded instead of powders, alcohol stands first in the list, in the treatment of chronic purulent otitis media. This may be used in the form of spirits of wine, or of absolute anhydrous alcohol. An application of ten drops may be made once or twice a day, after the ear is cleansed either by absorbent cotton or by syringing.

Solutions of sulphate of copper, one grain to the ounce of water, sulphate of zinc, from one to three grains to the fluidounce, and nitrate of lead, ten grains to the fluidounce, will be found of value in checking the discharge. As, however, the disease does best under the dry treatment, all fluid applications are but second choice. But cases may present themselves in which the dry treatment cannot be carried out, and in such the fluid applications I have named will be found of service.

Granulations, with broad base, need not be cut or scraped away. They will generally disappear under the antiseptic and dry treatment. But no fluid application excepting alcohol will be of use when granulations are present. In fact, the fluid treatment is contra-indicated when granulations are present, as moisture promotes their growth and development into polypi.

CHRONIC PURULENCY OF THE ATTIC OF THE TYMPANUM.—Chronic purulent disease in the attic appears under two forms: (1) in conjunction with chronic suppuration of the atrium, or lower and larger part of the tympanum, the latter being accompanied by perforation of the membrana tympani; and (2) as a chronic purulent disease limited to the attic, the membrana flaccida, the part above the short process of the hammer, alone being perforated, the membrana vibrans, the part below the line of the folds, being intact and often normal in appearance. In the first case the discharge is copious, while in the second form it is usually scanty.

The attic contains the head and neck of the malleus and the body of the incus; its upper wall is the tegmen tympani, the boundary between the tympanum and the cranial cavity. It is therefore manifest that disease in this space threatens the meninges by extension through the dehiscences which usually exist in the tegmen tympani. A combination of the two forms—*i.e.*, the coexistence of a perforation in the membrana flaccida with one in the membrana vibrans—is the rarest exception. The membrana flaccida is that part of the membrana tympani bounded above by the semicircular bony edge of the segment of Rivinus—*i.e.*, the inner edge of the upper wall of the auditory canal—and below by the folds of the membrana and the short process. As it is composed of two layers only,—skin and mucous membrane, being devoid of fibrous tissue,—it is loose, and has received the name of flaccid membrane. It is also called the membrane of Shrapnell, after him who first described it.

The second form exists rarely in children, but there is reason to believe that the first form, in which the atrium is also diseased, is often found in them. In such cases as the latter the incus is often partly or entirely destroyed by necrosis. The malleus also is partly destroyed in the handle, the upper parts alone remaining in union with the remnants of the membrana tympani. The stapes is usually intact, as it seems most resistant to purulent disease and necrosis.

Treatment.—The first form of attic disease demands, in addition to the treatment already laid down for chronic purulency of the drum, a thorough drainage of the attic. This is best accomplished by removal of the remnants of the membrana tympani, the malleus, and the incus. The stapes should be left undisturbed. This operation of excision of the remnants of the membrana and ossicula auditûs is performed with the patient under ether. The illumination of the ear is accomplished by means of an electric lamp arranged to be worn on the forehead. If the incus-stapes joint is visible, the latter should be separated from the former, by means of a knife specially devised for this purpose. If this joint is hidden by the still extant upper posterior quadrant of the membrana tympani, it should be exposed or sought for, by the excision of the obscuring part of the drum-membrane. If discovered by this act, the joint should be severed as stated above. The next step will be to sever the tendon of the tensor tympani muscle, by means of another form of knife, specially devised for this act. Then all remaining attachments of the membrana tympani to the annulus tympanicus should be severed by means of a blunt-pointed knife, when the malleus, being seized by delicate forceps or by a polyp-snare, can be lifted from the ear. The incus in these cases is generally destroyed by necrosis, but, if not, it can be removed by forceps, or by a hoe-like instrument if the ossicle has slipped high up in the attic.

If we are dealing with the second form,—viz., one in which the perforation is in the membrana flaccida only, through which the pus escapes, while the membrana vibrans is intact,—the procedure is a little different. In this form the incus-stapes joint is necessarily unexposed: at most, only the neck of the malleus can be seen. The first act, therefore, will be to excise the upper posterior quadrant of the membrana tympani and expose the incus-stapes joint. Then disarticulate these two bones, sever the tendon of the tensor tympani, and cut away all attachments of the intact membrana to the annulus tympanicus, by a circular cut embracing the entire periphery. The malleus, with the remnants of the membrana, can then be removed as stated above.

This operation may be the only means of obtaining a radical cure in some cases of chronic suppuration in the attic.[1] Much relief, however, and even long periods of immunity from the offensive purulent discharge, may be obtained by the use of various fluid remedies applied to the attic, through the perforation, by means of the tympanic syringe. The latter is a syringe holding about a fluidounce, to which is fitted a slender nozzle, which is introduced into the attic through the perforation. By this means the diseased cavity may be syringed with peroxide of hydrogen, and then by solutions of carbolic acid, sulphate of copper (gr. iii to f℥i), alcohol, etc., those named being the preferable ones.

Peroxide of hydrogen, having a peculiar affinity for albumen, breaks up pus as soon as it comes in contact with it, making by such action a copious foam. This not only is a great cleanser, but it reveals by this foam the presence of pus even in very small quantities. When the foam ceases to appear it may be concluded that all pus has been removed.

Mastoid Disease.—Affections of the mastoid, being usually the result of chronic purulent disease in the tympanic cavity, may be considered at this point.

For clinical convenience mastoid inflammation may be divided into:

1. Periostitis of its outer surface.

2. Congestion and inflammation of the mucous membrane lining the air-cells of the mastoid cavity.

3. Caries and necrosis; followed by thrombosis of the lateral and other sinuses of the brain, general embolism, pyæmia, or cerebral abscess.

The first form is not uncommonly observed as an attendant of acute inflammation of the middle ear, with consecutive inflammation in the external auditory canal. It may also appear during chronic suppuration in the tympanum. An abscess may form over the mastoid as a result of this periostitis, and in a strumous diathesis caries of the outer table of the mastoid may be induced. This latter, *asthenic* form is characterized by its painlessness; the former, or *sthenic* type, by great pain. The asthenic form may occur as a sequel of diphtheria in children.

When the sequestrum is found, it should be removed at once. Such a disease as this, occurring over the outer wall of the mastoid in a child, becomes of moment not only to the hearing but even to the life of the patient; because in children there is much greater probability of an extension inward of such a disease than there is of its passage outward, for the tissues over the mastoid in young children are much more resistant than the thin and somewhat cribriform outer table of their partly-developed mastoid. Sometimes that which appears to be an abscess pointing over the mastoid undergoes resolution by ordinary poultices.

The sthenic variety of mastoid periostitis is characterized by pain and tenderness in the mastoid portion, with some redness of the skin. It may mislead the observer into the idea that it is inflammation of the mastoid cells. But the less deep-seated pain in the ear and head and the readiness with which the periostitis yields to leeching or a deep incision (Wilde's incision) will serve as diagnostic points. It must be borne in mind, however, that inflammation of the external periosteum may be associated with deeper inflammation in the mastoid cavity. This, however, is hardly the case in young children, as they do not possess largely-developed mastoid cells. Hence the second and third forms need not be expected in them. Yet chronic purulent inflammation of the drum-cavity is often followed in children by thrombus in the cerebral sinuses, general pyæmia, and abscess in the brain.

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[1] See Schwartze, Chirurgischer Krankheiten des Ohres, and Samuel Sexton, M.D., Diseases of the Ear, William Wood & Co., 1888.

After a purulent inflammation has existed for some time in the middle ear, there may suddenly arise an acute and violent inflammation in the organ. This usually occurs after exposure to cold air or to a cold water bath, or after a blow on the ear. The first result of the engorgement of the vessels is a diminution or checking of the discharge. The pain is often severe, and of a throbbing or boring kind. Pain is often felt in the brow, in the parietal and in the mastoid region of the affected side. Facial paralysis is quite a common occurrence in these forms of inflammation in the middle ear. It is due to pressure of the greatly-swollen mucous membrane or the inspissated pus upon the facial nerve through a dehiscence in its canal. The discharge, which had at first ceased, may now be renewed, though altered in quality, being thinner and very offensive. The facial paralysis is, however, by no means the worst symptom in such cases, as it usually disappears if the patient survives. A more alarming symptom is swelling and pain in the jugular region on the side of the affected ear. This symptom indicates involvement of the lateral and other sinuses of the brain, and is apt to be followed by embolism in various parts of the body. If an abscess forms in the mastoid antrum of a child, it will much more readily discharge itself through the thin, cribriform outer plate of the mastoid process and point in the soft parts of this region.

Such a termination is a usual one in childhood. Cerebral abscess is a not uncommon result of chronic inflammation of the tympanic cavity with involvement of the mastoid. It is due to purulent absorption, as is shown by its occurrence sometimes on the side opposite the diseased ear.

Treatment of Mastoid Disease.—Inflammation of the mastoidal periosteum will usually yield to the local abstraction of blood by means of leeching or a deep incision—*down to the bone.* The incision is to be kept open for a day or two. An artificial perforation of the mastoid cortex will rarely, if ever, be demanded, in children. When the abscess makes its way to beneath the skin and points over the mastoid, the treatment is the same as for an abscess in any other part of the cutaneous surface. In fact, if the chronic purulent disease of the drum-cavity were more frequently heeded and promptly received proper treatment, there would be no mastoid involvement in children. Even when it does appear, ordinary poulticing will often give desired relief in children, on account of the soft and open mastoid cortex in them.

CHRONIC NON-PURULENT OTITIS MEDIA.

The onset of this disease in children is usually insidious. Infants may be attacked by it and their hearing greatly impaired before it is known that such a change has occurred. After a severe cold in the head and an acute catarrh of the Eustachian tube, the mucous membrane does not regain its normal state, but remains in a condition of chronic inflammation, which gradually assumes the form known as hypertrophic catarrh. This may set in without much or any pain. If the child is an infant, it may be remembered that it once had a bad attack of "snuffles or a bronchitis," but it was not noticed to be deaf. In an older child—one who has learned to talk—it will be observed that it does not hear well after a cold in the head. This may pass away, to return with another cold; and at last it is observed that the child is permanently dull of hearing, and perhaps growing worse. If at school, it falls behind in its studies, or it is supposed, often very unjustly, to be heedless, and gets blame which it does not deserve.

If in this early stage the ears, the nares, and the throat be examined, changes peculiar to the disease will be found to have taken place. The drum-heads, the membranæ, will be found more retracted than normal, perhaps less shining, and the color transmitted through them will be red or pink, from the congested mucous membrane over the inner wall of the drum-cavity.

The nares will present at this time a swollen and red mucous membrane over the turbinated bones, and the respiration through them will be impeded. A more or less sticky and copious discharge will come from the anterior nares and also flow backward into the fauces.

The mouths of the Eustachian tubes will be found swollen and closed and blocked by mucus. The fauces are not always affected, like the nares and the Eustachian tubes, but usually they present a follicular pharyngitis and a swollen velum palati.

If these conditions of the nares, Eustachian tubes, and middle ears have been present for several months, the little patient may have had attacks of *not severe* earache, or at times may have complained of sharp darts of pain. The child now breathes through its mouth and presents the very injurious condition known as "mouth-breathing," the nares having nearly ceased to do their usual work, and the alæ being collapsed, giving the nose a pinched look. The hearing may now have become very much reduced and lead the parents to seek medical aid. Children five years old or more will often complain of subjective noises in the ears. By the repetition of isolated words, like "man" or "pin," or "four" or "more," it will be found that the hearing is reduced to a few feet or even inches, in serious cases. The consonant sounds are lost first, the patient saying "tin" for "pin" when tested. A watch is a poor test, and I never employ it. The tuning-fork or a small music-box is much better as an aerial test. But the best test is the voice, in speaking words, as I have just indicated.

If the nares are badly impeded, the words with nasals in them will be mispronounced, as the nasal resonance is cut off: *e.g.*, the word "nose" will be pronounced "doze."

Many cases of *deaf-muteness* arise in this way. The infant has a chronic aural catarrh, the process being identical with that in an adult. In the infant the process is not recognized until a year or two after its inception, when the child should begin to talk. Inspection reveals all the symptoms of chronic aural catarrh, but too often when all attempts at restoration of hearing are in vain, because the thickening of the membrana tympani, and more especially the sclerosis in the drum-cavity and in the joints of the ossicles, have become indelibly fixed.

Many cases of deaf-dumbness are supposed to be congenital. More than half have really become such after birth. Perhaps not more than twenty-five per cent. can be justly called congenital. The majority certainly originate from diseases occurring after birth, in early childhood, and those occurring in early infancy are doubtless due in many cases to an insidious chronic aural catarrh, especially when no other distinct and probable cause can be assigned for the deafness.

There seems to be some tendency to transmission of chronic catarrh of the middle ear from parent to child. When a deaf parent seeks advice regarding a child with catarrhal deafness, the prognosis is always less encouraging. There is in such cases a transmission of the tendency for the nares and Eustachian tubes to assume a hypertrophic catarrhal condition, just as in some families there exists the tendency to catarrhal diseases of the bowels or of the lungs.

The cerumen of the auditory canal is diminished in quantity and sometimes brittle in quality in the early stages of chronic aural catarrh.

The membrana tympani, as stated, undergoes changes in color and tenuity. Instead of a thickening there may be a thinning of the membrane, especially if there is a tendency to ozæna in the case. The color of the membrane often appears red, from the transmitted tint of the congested mucous membrane beyond. Calcareous spots are rarely found in the membrana tympani of a child affected with catarrhal deafness.

The position of the membrana tympani changes, from the retraction it undergoes, after the catarrhal disease has affected the middle ear for some time. The malleus then appears foreshortened perspectively, and the short process seems unusually prominent. The manubrium of the malleus is not only drawn inward, but is pulled upward and backward, and, the curves of the membrana being thus altered, the pyramid of light, normally found in the antero-inferior quadrant, is either thrown upward in the anterior half of the membrane or disappears altogether. In fact, the normal pyramid of light is usually one of the first features of the membrana to change in chronic aural catarrh.

The manubrium of the malleus not only is indrawn in this disease, but it is rotated about its long axis, so as to pull the posterior half of the drumhead into greater prominence and to drag the anterior half into a greater depression. This is seen, however, only in older children and in adults.

The Pharynx and Naso-pharynx.—In children the moist form of hypertrophic catarrh is usually met, in which the nares, the pharynx, and the naso-pharynx are in a state of hypersecretion. The tonsils are usually enlarged, the nares impeded, and mouth-breathing is going on. If the case has assumed the atrophic state (ozæna), the secretion of all these parts will be found scanty, and the mucous membrane dry and covered with scales of inspissated mucus and pus. In these cases the mucous membrane of the posterior pharyngeal wall looks as if varnished, the turbinated bodies are atrophied, the space in the nares is thereby increased, and the nares, not being able to clear themselves by normal respiration, are filled with decomposing and highly malodorous scales of dried mucus and pus.

In some instances the velum palati shows a peculiar paresis if the case is at all chronic. The uvula then, instead of hanging in the median line, is drawn towards one side. This latter is found to be the side of the better ear, because the catarrh has not yet weakened the muscular structures of the velum and the Eustachian tube on this side. The loss of normal mobility in the velum is further seen when the patient is told to phonate the sound of broad *a*. Then the velum and uvula, instead of rising quickly to shut off the lower from the upper pharynx, will fail to perform this act as quickly or as well as the normal organ.

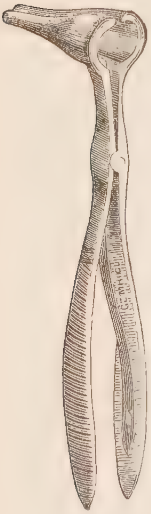
When the child blows its nose or when its Eustachian tube is artificially inflated, it often complains of cracking in the ear. This is caused by the air thus forced into the tube forming bubbles with the mucus. The mucous membrane of the nares and naso-pharynx may become very much congested and swollen, and so irritated thereby as to puff up and close the mouths of the Eustachian tubes, causing vertigo and faintness, if it is subjected to any irritant.

Adenoid growths often occur in the naso-pharynx in northern and stormy latitudes. These growths are benignant in nature, are more or less leaf-like and conical in form, and are usually placed high in the pharynx. They are friable, and hence bleed easily. Their height or length rarely exceeds three centimetres, and their thickness varies from a few lines to one or two centimetres. These growths interfere not only with normal respiration and enunciation, but also with the normal ventilation of the Eustachian tubes and the tympana. The pharyngeal tonsil, situate in the vault of the naso-pharynx, often becomes enlarged in children, and is productive of chronic catarrh of the naso-pharynx, Eustachian tubes, and middle ears, with resultant hardness of hearing.

Causes of Chronic Catarrh of the Middle Ear.—Very few parents can assign a satisfactory cause for chronic middle-ear catarrh in their children. Chronic cold in the head, or frequent colds in the head, are undoubtedly the fundamental cause of such chronic catarrhal deafness in children. Chronic aural catarrh is often found associated with and doubtless is aggravated by chronic catarrhal diseases in the mucous membrane elsewhere: by phthisis, by hereditary syphilis, by continued fever, by all eruptive fevers, and by eczematous conditions of the general integument. Whooping-cough and mumps are often followed by chronic deafness. After these two diseases, however, there is reason to believe that the auditory nerve is often primarily affected. It has also been observed that the children of Anglo-Saxons born in tropical countries seem specially liable to chronic aural catarrh.

Treatment of Chronic Catarrhal Otitis Media.—The treatment of this affection of the ear will depend upon the form of the disease in the case presenting itself. It must first be decided whether the disease partakes of the hypertrophic nature or of the atrophic. The treatment for the first is very different from that demanded by the latter. Let it be said at the outset that the *nasal douche* should *never* be used.

Fig. 13.



Bivalve speculum for examining the anterior nares.

If the patient is an infant, it will not be easy to determine the degree of deafness. If able to talk, the patient's hearing can easily be determined, as already shown on p. 31.

Let us suppose we are confronted by a case of the hypertrophic form of catarrh of the nares, naso-pharynx, and middle ear. The membrana tympani must be examined, after the hearing is tested; then the nares and naso-pharynx, as far as is possible in young patients. We must note whether the turbinated bodies are in the first and active stage of hypertrophic catarrh, or whether a sclerotic and contracted stage has been reached. The condition of the fauces must also be noted, as well as the state of the tonsils.

If a hypertrophic catarrh of the nares and naso-pharynx in the active, secretory stage is found, we may spray the nares and naso-pharynx with a solution of sulpho-carbolate of zinc, three grains to the fluidounce of water, with a fluidrachm of glycerin. Or we may employ a spray of a modified Dobell's solution, consisting of the following:

℞ Sodii bibor.,
Sodii bicarb., āā gr. ii;
Acid. carbol., gr. i;
Glycerini, f ʒ ss;
Aquæ, f ℥ i.

Another excellent spray is obtained by using a solution of iodide of zinc, two or three grains to the fluidounce of water.

These solutions are most easily and very efficiently atomized by using what is known as the Magic Hand-Atomizer No. 2, made by the Davol Manufacturing Company, of Providence, Rhode Island. There are, however, numerous forms of hand-atomizers which act perfectly well.

After the application of the watery spray it is advantageous to spray into the nares some liquid albolene. This is especially necessary in cold weather if the patient is likely to be exposed soon to the open air.

If a more advanced stage of catarrh of the naso-pharynx and middle ear has been reached, and there are evidences of sclerosis in the mucous tissues, a more stimulating spray may be employed. In my opinion, we are at this stage well served by a spray of the following:

℞ Listerine (Lambert's), f℥ss;
Aquæ, f℥iv.
Misce.

Or

℞ Acid. boric., gr. x;
Glycerini, fʒi;
Aquæ, f℥i.
Misce.

It may be said, however, that in children the active secretory stage, with puffed and red turbinated mucous tissues, is much commoner than the pale, contracted, sclerotic form.

The fauces will usually be benefited by the treatment applied to the nares, because some of the medication reaches these lower parts, and also because, the nasal respiration being improved by the nasal treatment, the child does not breathe through its mouth as much as previously, and the fauces thus escape the irritation of direct mouth-breathing.

After the application of spray to the nares, the Eustachian tubes and the tympana should be inflated by the air-douche, according to Politzer's method or its modifications. This is the only form of inflation of the middle ear practicable in children, and, fortunately, no other is needed.

If the patient be an infant, we have only to insert the nose-piece of the inflation-bag into one naris, and, with the two fingers of the left hand, gently compress the other naris and the ala of the one in which the nose-piece is (see p. 20). A moderate compression of the inflation-bag, in the right hand, will usually suffice to send air into the tympana. If the child cries, the velum is elevated and cuts off the naso-pharynx from the pharynx, thus facilitating the inflation of the tympana. In larger children this is readily brought about by asking them to swallow a little water, previously taken into their mouth, at the moment we desire to inflate, or by telling them simply to distend the cheeks,—according to the suggestion of Dr. Holt, of Portland, Maine. If this is done gently and deliberately, the child will not be hurt and will not resist the surgeon's subsequent efforts for its relief.

Here let it be said that no applications made to the external ear and the outer surface of the drum-membrane will do any good in chronic catarrh of the middle ear.

Excision of the tonsils is not demanded as an aid to the cure of chronic catarrhal deafness. Applications of iodine and glycerin, equal parts, may be of value as tending to diminish the size of the tonsils. If follicular pharyngitis is present, applications of tincture of chloride of iron and water, in equal parts, may aid.

Treatment of the Atrophic Form.—If we are called upon to treat a case of deafness dependent upon or associated with the atrophic form of naso-pharyngeal catarrh, the local treatment must be a stimulating one. This is accomplished by spraying the nares with a solution of iodine and carbolic acid, known as Boulton's solution, or with a solution of bicarbonate of sodium and biborate of sodium, of each half a drachm, to four fluidounces of listerine.

In larger children with well-marked ozæna, there may be employed a spray of thymol, from half a grain to a grain to the ounce of water. Some alcohol may be required to make a perfect solution of this last-named drug. Its application stings a little, but this is allayed by spraying with fluid albolene, which should always be done after each act of spraying the nares and naso-pharynx. After the nares have been treated as explained, the tympana should be inflated.

This treatment which I have sketched for the various forms of naso-aural catarrh, if applied properly and in time, will rescue an infant from the onset of deaf-dumbness, as set forth on p. 32.

Deaf-Muteness.—If the chronic aural catarrh productive of deaf-dumbness in an infant were discovered in time, it could, as I have observed, be cured, or sufficiently alleviated to prevent the child from being a mute. But if the case gets no treatment until the child is two years of age, its deaf-dumbness is incurable.

When chronic aural catarrh occurs in children who have already learned to talk, they should be encouraged to go on talking and not to make signs. Young children from three to five years of age will stop talking if they get deaf. They must now be forced to go on talking, while being treated for their chronic catarrhal deafness. If this is in part or entirely curable, they will retain their speech; but they must be carefully watched, to see that they do not begin to make signs or mispronounce words. If their deafness continues, they may nevertheless be rescued from being mutes by the precautions I have named as to their continued practice in the use of speech.

The same care in exercising their speech must be given to young children who become deaf from diseases of the internal ear, like that occurring in cerebro-spinal meningitis, in mumps, and from blows or falls on the head. These forms of aural disease will be considered farther on in this article.

Great assistance in making the child hear, and thus teaching it to perfect its power of speech, and also to retain and improve its hearing, can be gained by the employment of either a good tin ear-trumpet or, what is in my opinion still better, a Maloney otophone.[1] This latter instrument conveys speech to the deaf ear not only with the most power, but also with the greatest purity of sound, because it is free from unpleasant resonance and the interference of sound-waves. Furthermore, it has the very good feature of not being inserted into the meatus, but its aural end is provided with a disk-like expansion three inches wide, which is held against the auricle, and therefore does not cause pain in the ear. If a child retains any hearing, this will be improved and the child's speech developed by the intelligent use of the above-named instrument, to convey the words of a parent or teacher to its ear.

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[1] Made by J. A. Maloney, Washington, D.C.

Adenoid growths and enlarged pharyngeal tonsils, as described on p. 33, may demand removal by crushing, evulsion, or cauterization. It should be borne in mind, however, that these enlarged glands and adenoid bodies undergo a spontaneous retrogression towards puberty, and therefore, unless they are productive of great aural irritation and deafness, a conservative course of treatment should be pursued, and violent surgical interference in the naso-pharynx avoided, for such irritation of itself may set up an acute otitis media and aggravate, rather than ameliorate, the condition of the ears.

Earache and Chronic Catarrh of the Middle Ear.—I am often asked, What should be done for earache? As earache is commonly due to subacute attacks of catarrh in a child who is the subject of chronic catarrh of the naso-pharynx, I will try to answer the important question here. In all cases of earache the proximate cause should be determined, as well as the seat of the inflammation. Sometimes earache is due to a myringitis, the middle ear being very slightly or not at all implicated, as shown on p. 15. But most cases are catarrhal, and a child who has one attack of earache is very apt to have another, unless the nares and naso-pharynx are treated, and the child is kept from imprudent exposures to bad weather and wet feet in winter-time and from cold bathing and exposure to draughts of air in summer-time. If, however, a child has earache in spite of all that is done for its nares and middle ear, it should at once be housed, or even kept in its room and in bed if the pain is severe or long continued and there are symptoms of an acute "cold." Nothing should be dropped into the ear, for that is generally worse than useless: it always irritates the inflamed tissues.

The bowels should be in a normal condition. If confined, a laxative or a purgative may be given, though this is not imperative unless the constipation is excessive. If the child is feverish, a mild febrifuge should be given. If the child seems to have simply a grumbling pain in the ear, in addition to keeping him in the house or in a warm room, dry heat should be applied to the painful ear, as set forth on p. 19. If in spite of this treatment the pain grows worse, the local abstraction of blood may be resorted to. But, as I have already stated, if the ear is not improperly treated by various drops at the beginning of the pain, which invariably make it worse at last, the very simple plans I have given, if promptly pursued, will give relief.

At the beginning of an earache in a child who is already the subject of naso-aural catarrh, an inflation of the tympanum, by Politzer's or other methods, will give relief by opening the middle ear and overcoming the retraction of the membrana tympani.

Hygiene of the Ear.—Prevention of earache and further ear-disease, in such cases as I have described, is far more valuable than any attempts at cure, just as it is elsewhere in the body. A child who has once been the subject of an earache from catarrhal causes has received a valuable warning; or, at least, its parents have.

Of course its nares and naso-pharynx must be carefully watched and treated until normal respiration through them is established and mouth-breathing prevented. A mouth-breather is always threatened with earache and deafness. Such a child must have plenty of exercise in the open air, must not be in a badly-ventilated school-room, nor must its study-hours be as long as those of the child free from such defects.

Its underclothing must be of wool—all wool, not merino—at all times excepting the very hottest weather. Even at such time, if it can endure very thin wool, so much the better. But, as children are very active even in summer-time, they may be permitted to wear merino at that time. The feet and ankles must always be kept dry.

In summer-time cold-water bathing should be indulged in with the greatest caution, while diving must be most strictly prohibited. Bathing in the tub in winter-time must be done not too frequently,—once a week is enough for cleanliness,—always in a warm room (not lower than 70° F.), and preferably just before going to bed. If the hair is washed, as it often is in girls, it must be carefully dried with a towel,—not before a fire nor an open window,—and it would be safer to remain in the house the rest of the day. A fire is too heating and congesting, and a draught from an open window is of course very liable to cause a cold in the head, even in summer-time.

The sleeping-apartments should be well ventilated, but no draught should be felt blowing on the head at any time of year. When in a perspiration, the greatest caution should be exercised at all seasons of the year regarding cooling. The child should be taught that this should never be done in a draught nor rapidly in any way.

In regard to blowing the nose, as it is termed, I would say that the nose should never be blown violently, but rather wiped, and sneezing should always be suppressed as far as possible, because both of these acts when done forcibly tend to further congest the nares and naso-pharynx and make the catarrh worse. I am sure many a so-called cold in the head could be—in fact, has been—prevented by avoidance of violent sneezing and blowing the nose at the beginning of the irritation. Hence catarrh snuffs and all forms of catarrh remedies of a sternutatory nature should be most carefully avoided by those afflicted with a catarrh in the nares or ears.

The shoes and clothing, like trousers in boys and skirts in young girls, should not become damp. But if these get wet from unavoidable exposure, they should be taken off and dried as soon as possible. Wet feet are especially injurious to the fauces, nares, and middle ears of children.

Mouth-breathing in children must be stopped as soon as possible. In those who are six years old and over it is often merely a *habit*, originating in past "colds in the head." In such cases an exercise of their volition will overcome it in the daytime and tend towards correcting it when they are asleep at night. All such efforts on the patient's part will be furthered by treatment of the nares by the remedies suggested for chronic hypertrophic and other forms of nasal and naso-pharyngeal catarrh. The nasal douche should never be used. The reader is also referred to the part of this work having especial reference to nasal and pharyngeal diseases and their treatment.

DISEASES OF THE INTERNAL EAR.

By the internal ear is meant the so-called labyrinth, composed of the vestibule in the middle, the cochlea in front, and the semicircular canals behind. These parts are all interconnected. The auditory nerve sends branches to all three of these divisions of the labyrinth, but most generously to the cochlea. The nerve-filaments in the semicircular canals have not been traced beyond the ampullæ. In the vestibule they distinctly supply the sacculi, the chief soft contents of that part of the labyrinth which is nearest the conductors of sound, being just behind the foot-plate of the stapes, where it is received into the oval window. The round window is practically the distal boundary of the cochlea, while the oval window may be considered the point of the initial impression made on the endolymph in the labyrinth by the oscillations of the conductors of sound-vibrations, the ossicula auditûs.

These labyrinth structures and their mechanism are extremely delicate and susceptible of derangement by force from within the cranium and also from without. Many of the processes of disease in the internal ear have their origin in catarrhal diseases in the mucous membrane of the middle ear. The vascularity of these two parts is intimately and directly connected, and hence a morbid process in the middle ear—the part of the ear most likely to be primarily affected—is very easily communicated, by vascular changes, to the labyrinth.

The deafness in such cases is slow in its approach, but permanent and incurable. Such are the forms of deaf-dumbness from chronic catarrh of the middle ear (see p. 32).

In other instances the disease of the labyrinth originates within that cavity, and in such cases the affection is characterized by sudden and permanent deafness. The cause of this form of labyrinth-disease is either an apoplectiform hemorrhage into the confined bony case containing the auditory nerves, producing a destructive pressure upon the nerve-tissues, or it is a sudden displacement and tearing of the nerve-structures by concussion, as from a fall or a blow on the head. Doubtless this latter cause is a frequent one in children.

This second variety is very important, from the fact that its prevention should be most carefully aimed at, as a cure in such cases is very difficult, if indeed it is at all possible. Hence all sports which include standing on the head, or hanging by the heels with the head down, should be prohibited, as tending to produce congestion and hemorrhage in the head. Furthermore, a child in this position, or in that of so-called walking on the hands, is exposed to the risk of suddenly falling on its head, or striking its head from its disturbed co-ordination. A blow on the head, which must be congested while in such a position, is very likely to bring about destructive concussion of the soft tissues in the bony labyrinth. Hemorrhage and effusion into this cavity ensue, the nerve is suddenly and hopelessly impaired, and the child becomes absolutely and permanently deaf. Such cases I have observed.

A third class of labyrinth-diseases is produced by intracranial processes, and also by the effect of some general systemic disorders upon the internal ear. In this division we find the effects of mumps, scarlet fever, typhoid fever, cerebro-spinal meningitis, meningitis in general, hydrocephalus, otitis labyrinthica, hemorrhagic otitis, etc.

MUMPS.—Under this third division we may first consider the effect of mumps on the ear. That the nervous apparatus of the internal ear is sometimes suddenly and permanently injured by the poison of mumps, the process being most probably a metastatic one, has been recognized and described by a number of writers, from Toynbee and Hinton to the present time. Usually the aural symptoms consist in a simultaneous tinnitus aurium, vertigo, and deafness. The two first-named symptoms disappear sooner or later, but the deafness remains. When no disturbance in equilibrium occurs, the metastatic process is probably limited to the cochlea, the semicircular canals escaping. These peculiar symptoms may not develop until the fifteenth day, and then only after subsidence of metastatic processes elsewhere. Usually there are no symptoms in the external nor middle ear to account for the sudden deafness following mumps. The diagnosis of the origin of the failure of hearing is aided by the tinnitus, nausea, vomiting, vertigo, and sometimes the altered gait, ensuing suddenly either during or after an attack of mumps. Sometimes there is pain in the ear and head, and in a day or two deafness, first in one ear and then in the other. It is supposed that in some instances the deafness and other aural symptoms are due to a serous exudation into the labyrinth. It is, fortunately, a rare sequel of mumps. No treatment has shown itself capable of either preventing or curing the deafness.

CEREBRO-SPINAL MENINGITIS is often attended with deafness during the fever, and this failure in the function of the ear very frequently is permanent. At the termination of the disease or during convalescence the patient is found to be profoundly deaf, usually in both ears. There is also in many cases an alteration in the walk, so that the little patient assumes a sailor-like gait. The lesion seems to be due to a neuritis descendens,—*i.e.*, a slow encroachment of the inflammation in the interior of the cranium, upon the labyrinth, along the perineural vessels of the auditory nerve. Moos (1881) shows that in sixty-four cases of ear-disease following cerebro-spinal meningitis, as observed by him during eighteen years, fifty per cent. showed disturbances of equilibrium and hearing, fifty-nine per cent. were totally deaf in both ears and hence became deaf-mutes, thirty-one and a half per cent. were totally deaf but retained speech, while one and a half per cent. escaped without an aural lesion. He also observed that those left with hearing for high notes, but with dulness of hearing or absolute deafness for low notes, stood a better chance of retaining hearing for spoken words. If absolute deafness persists for more than three months after the cerebral disease, the prognosis is unfavorable, without exception.

Treatment.—Charcot has proposed to treat the dizziness and staggering with large doses of quinine. His plan consists in beginning with thirty centigrammes of the sulphate of quinine in an adult, and gradually increasing until one gramme is taken daily. This method of administering quinine may be kept up for a month; then a pause for a fortnight should occur; the treatment may then be resumed for another month. Of course in children the dose must be proportioned to their age. Charcot's theory of the mode of action of the quinine in these cases is that it destroys any remaining function in the auditory nerve. This, however, is not accepted by Moos, who claims that quinine acts simply by antagonizing the inflammation. In cases where the hearing is not entirely destroyed, the constant electric current applied to the ear offers some hope of improvement, if made at once, as soon as convalescence sets in.

Otitis Labyrinthica.—In some respects closely resembling the otitis interna of cerebro-spinal meningitis is an acute inflammation of the membranous labyrinth, described by Voltolini as occurring in young children. He reported (1872) some cases of a disease closely resembling acute meningitis, which he claims is a disease of the labyrinth peculiar to children, and as specific as croup.

The symptoms may be briefly described as follows. A child five years old, with perfect hearing, may be attacked suddenly with vomiting, which lasts for several days, with intermissions, and there will be accompanying chill and fever. No cause can be assigned by the parents. On the first day of the illness the child still hears, but on the second day the hearing is entirely gone. The intellect remains clear during the entire disease, and there are no spasms, paralysis, nor opisthotonus. The urine and fæces are in no way abnormal. The child may complain of the subjective noises in its head. By the fourth day the appetite returns and the child begins to play. Upon attempting to walk, in the course of two or three weeks, the gait is very unsteady and the child has to be led about. An examination of the external auditory canal and membrana tympani reveals no alterations to account for these distressing symptoms. As death has never occurred in any of these cases, the precise lesion has never been determined. There are some points of differential diagnosis between this disease and the aural disease following cerebro-spinal meningitis. In the latter disease convalescence is slow and herpes labialis is an almost constant symptom, while it rarely appears in otitis labyrinthica. This latter disease is ushered in by vomiting, which is absent in cerebro-spinal meningitis. Then, too, the hearing is quickly and entirely destroyed in otitis labyrinthica, while in cerebro-spinal meningitis it is destroyed much less rapidly and partial audition is maintained for some sounds. Treatment is of no avail in otitis labyrinthica.

Closely related to the foregoing process in the labyrinth—probably identical with it—is the so-called

PRIMARY OTITIS INTERNA.—Toynbee, Moos, Steinbrügge, Politzer, and Gradenigo[1] have demonstrated changes in the labyrinth, of an osteoplastic form, consecutive to a destructive pathological process in the soft tissues of the labyrinth.

Toynbee found besides changes in the middle ear an osseous deposit near the vestibule, lying on the lamina spiralis, which entirely filled the scala tympani, and covered the inner surface of the membrane of the round window. Moos and Steinbrügge found, in a girl who had been deaf many years, inflammation of the labyrinth and the nerve-structures entirely wanting in the first whorls of the cochleæ; also partial ossification and formation of connective tissue. Politzer described a case of total ossification of the labyrinth, found in a boy who had become deaf at the age of two and a half years, after an attack of fever, accompanied by great restlessness, convulsions, and a discharge from both ears, the latter lasting until the child was seven years old. At the age of thirteen years the child died of acute peritonitis, and then the post-mortem examination of the ear was made. In Gradenigo's case the patient was a deaf-mute girl fifteen years old. She died of pulmonary phthisis, and the post-mortem examination revealed complete destruction of the membranous labyrinth of both ears and the elements of both labyrinth windows, with new formation of fibrous and osseous tissue, most marked in the left ear, where there was an accompanying chronic purulent otitis media. The new-formed bone-tissue was developed partly from the endosteum of the labyrinth cavity, and partly by direct metamorphosis of new-formed fibrous tissue. There were no traces of semicircular canals: the vestibule was narrowed. In the cochlea the new osseous tissue diminished in quantity as the cupola was approached.

Gradenigo[2] further shows that otitis interna may be due primarily to hereditary syphilis, and secondarily to cerebro-spinal meningitis, and sometimes, though rarely, to otitis media suppurativa (panotitis) in children.

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[1] Archiv für Ohrenheilkunde, 1887, Bd. xxv. p. 46.

[2] Ibid., 1887, Bd. xxv. p. 237.

HEMORRHAGIC INFLAMMATION OF THE LABYRINTH IN CHILDREN.—It has been demonstrated by post-mortem examination[1] that hemorrhages and hemorrhagic inflammation occur in the labyrinth of children. This may be best understood from the following history of a case.

A boy three and a half years old was affected with symptoms of a mild cerebro-spinal meningitis. On the tenth day he had begun to manifest symptoms of improvement, but the day after he became suddenly deaf. For three days preceding this event he had complained of intense tinnitus aurium. Symptoms of meningitis, with strabismus, now returned. Three weeks after the relapse the patient was emaciated, vomiting, and insensible. Eight weeks after the beginning of the illness the patient died, apparently in consequence of tubercular meningitis. The post-mortem examination revealed signs of internal hydrocephalus and a tendinous thickening of the pia mater at the base of the brain. The dura mater covering the petrous bone was reddish, blue, and green, especially in the region of the semicircular canals. The osseous semicircular canals were filled with fluid blood and dark-red coagula. The vestibules also were filled with blood; the cochleæ contained less. Pus was found in the canals and in the broken-down membranous parts of the labyrinth generally. The vessels of the lamina spiralis were greatly congested, and Corti's membranes in both cochleæ very much thickened. There were no evidences of tubercles in the labyrinth.

Careful examination seemed to show that the disease had reached the labyrinth by means of a reduplication of the dura mater, very rich in blood-vessels, which extended into the *hiatus subarcuatus*, the space beneath the superior semicircular canal, which exists in children, not yet having been filled up with bony tissue. There was also an osteo-myelitic condition of the spongy tissue of the bone about the labyrinth, which had induced the hemorrhagic inflammation of the membranous labyrinth. It seems probable that this is the way of transmission of inflammation from the meninges to the labyrinth in many cases, and it may explain instances of sudden deafness in children.

DISEASE OF THE LABYRINTH IN TYPHOID FEVER.—In typhoid fever the entire labyrinth is often affected upon one or both sides. The parts more usually affected are the utriculus, the sacculus, the ampullæ, and the lamina spiralis membranacea. The semicircular canals are not so likely to be affected. Histologically the affection consists in a small cell-infiltration,—*i.e.*, an infiltration of small lymphoid cells (Moos).

THE LABYRINTH IN SCARLET FEVER, ETC.—Ambilateral inflammation of the labyrinth has been found in cases of scarlet fever complicated with diphtheria, suppuration of the parotid gland, otitis media purulenta, and periostitis of the left squama, in which death occurred from secondary meningitis (Moos). The condition of the labyrinth in such cases is held to be an explanation of all cases of great hardness of hearing after scarlet fever, in which an analysis of the other clinical symptoms unconditionally excludes the supposition that a concomitant cerebral affection has caused the destruction of hearing.

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[1] Lucae, 1882.

Purulent inflammation of the labyrinth has been found in children who have died of variola.

Effects of Quinine and Salicylic Acid upon the Internal Ear.—Large doses of quinine produce congestion of the membrana tympani, the middle ear, and the labyrinth. Fifteen grains given at once to an adult will produce all the well-known symptoms of quinine-poisoning, and may be followed by permanent alteration in the function of hearing. Of course smaller doses will produce similar bad effects in children. It has been demonstrated by the experiments of Kirchner[1] that hyperæmia and hemorrhages in all parts of the labyrinth may ensue from large doses of quinine and salicylic acid. Thus, in a cat to which large doses of quinine had been given, a copious extravasation of white and red blood-corpuscles extended over large areas of the cochlea. In a preparation taken from a rabbit an extravasation could be seen extending from the semicircular canals to vessels of the surrounding osseous tissue. In a woman who had taken for a long time large doses of salicylic acid Kirchner found, in addition to symptoms of labyrinthine congestion, an exudation into the middle ear, which necessitated paracentesis of the membrana tympani. In cases of acute quinine-deafness it may be assumed that paralysis of the vessels and exudations ensue in various parts of the auditory apparatus.

Salicylate of sodium and salicylic acid do not seem to affect the ear as quickly nor as extensively as quinine. However, doses of fifteen grains every hour for five hours may induce permanent changes in hearing (Schwabach). To overcome the vessel-dilating effect of these two drugs it is recommended by Schilling that the vessel-contracting drug ergot be given shortly after these two drugs are administered. In eighty-seven cases in which salicylate of sodium was combined with ergot seventy-six per cent. of the cases were entirely free from effects of the drug upon the hearing. In nine cases in which ergot was combined with quinine no aural symptoms were observed. The antirheumatic and antifebrile effects of these drugs are not diminished by their combination with a controlling drug. In this connection it will be well to recall the suggestion of Finkler and Prior[2] that amorphous borate of quinine is an efficient antipyretic and antiperiodic remedy, and possesses, besides, the great advantage of not inducing tinnitus aurium to the same extent as the muriate of quinine. This was demonstrated by experiments upon themselves by the above-named observers.

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[1] Archiv für Ohrenheilkunde, Bd. xviii. p. 305; ibid., Bd. xx. p. 209.

[2] Deutsche Med. Wochenschr., 1884, No. 6.

Therefore, when the clinician resorts to large doses of quinine at a time, a controlling drug should be employed, in order both to give the patient comfort and to save his internal and middle ears from permanent injury.

Malformations of the Internal Ear.—For a detailed account of malformations of the ear and the literature of this subject the reader may consult Von Troeltsch's "Diseases of the Ear in Children," and Schwartze's "Pathological Anatomy of the Ear," both of which are translated into English by J. Orne Green, M.D., of Boston. It is sufficient to note here a condensation of facts as presented in the above-named works.

The entire labyrinth may be wanting, or it may be but partially developed; in the latter case some of its parts, most commonly the semicircular canals, are absent, or they exist only in a rudimentary form. Variations in the form and size of certain parts are very common, but these variations are said to be symmetrical. The possibility of a malformation or arrest of development being confined to the inner ear is explained by the development of the ear, the labyrinth being formed from its own so-called labyrinth-vesicle in the region of the cerebellum, while the middle ear and the auditory canal are formed from the first branchial fissure, and the ossicles from the two first branchial arches. The ossification of the labyrinth is said to be finished sooner than that of the external portions of the petrous bone. In some very rare cases congenital absence of the auditory nerve has been observed in connection with defects in the labyrinth.

DEAF-DUMBNESS: THE NERVOUS LESION.

The lesion in congenital deaf-dumbness may be an early periostitis of the petrous bones. This is shown by the sclerosis in the substance of the bones, the ankylosis of the ossicles, the partial hyperostotic condition of the bony walls of the tympanic cavity, and the closure of the fenestra rotunda. A colloid substance may be found in the labyrinth in such cases. In some instances the semicircular canals, the ampullæ, and the cochlea are wanting. The auditory nerves may be atrophic and contain concretions of phosphate of lime. In such cases the auditory vesicle alone is arrested in development. In other cases an examination of the labyrinth on both sides reveals a large quantity of otoliths and numerous colloid bodies in the ampullæ, in the sacculi, and on the membranous lamina spiralis. The abolition of hearing is often directly due to colloid degeneration in the labyrinth.

In some cases of congenital deaf-muteness some of the inner convolutions of the posterior lobes of the cerebrum are considerably atrophied, discolored yellow, and œdematous, as shown by post-mortem examinations (Moos, Steinbrügge, Luys, and others).

Aural Hygiene in Deaf-Mutes.—Deaf-dumbness may be either congenital or acquired; in some instances the two forms may be united. The congenital form of deaf-dumbness was once considered the commoner occurrence. But later investigations have shown that this is by no means the truth. Thus, in the Pennsylvania Institution for the Deaf and Dumb in Philadelphia, within three years one hundred and thirty-seven children were admitted who had lost their hearing from fevers and other known causes, and had thus become deaf. These constituted two-thirds of the entire number of admissions, thus demonstrating that, in this institution at least, congenital deaf-muteness is considerably less frequent than the acquired form.

Every physician may be called upon to decide whether a child is deaf and dumb, and, if it be, to suggest, if not a cure for the deafness, at least a plan for the proper care and education of the little patient.

Mute children are very apt to suffer from earache and sometimes from chronic discharges from their ears. These symptoms should be most carefully combated according to plans already given, in order to make the child more comfortable and to retain or improve any remaining hearing.

In very young children it cannot be readily determined, except by an expert, whether total deafness exists or not. But whether or not the child is totally deaf,—and very few mutes are entirely deaf,—it may be too deaf to learn to talk by hearing others speak. An opinion on this point is rarely sought for until, the child having reached the age when most children begin to use words intelligently, it arouses suspicion as to its defect by showing no evidence of learning to talk. The mere utterance of the words "mamma" and "papa" is no proof that the child hears, as these elementary sounds may be made quite involuntarily by very young children.

The child having reached the age of two years and given evidence of being a deaf-mute, it remains for us to consider what can be done for its education.

In all civilized communities there are provisions for the proper corporeal, moral, and intellectual training of the deaf and dumb. Deaf-mutes naturally communicate with one another by means of a sign-language, which in most respects is common to mutes of all nations. This method, scientifically elaborated, is termed dactylology, or finger-talking. Until within a few years it has been the only chief method of instructing deaf-mutes in England and the United States.

The system of educating mutes by teaching them to understand and to use articulate speech by observing and imitating the speech of others, in which method the pupils are not taught to use the sign-language at the same time, has been employed for a long time in most of the countries of Continental Europe. There are records which prove that in England at the beginning of the eighth century mutes were taught to understand the motions of the lips, and again in the seventeenth century Bulwer and Wallis, of the University of Oxford, induced some teachers to devote themselves to the instruction of deaf-mutes by means of lip-reading. In order to become educated by this manner the child must possess ordinary intelligence and normal vocal organs, and must begin its studies in this direction at not later than seven years of age. The average length of time demanded in the study of this method, before the pupil can understand and communicate with any one it may meet, is about eight years. But great attainments are thus made. English children thus skilled have learned to speak French and German. In Vienna I have conversed in German with mutes who understood me and replied in their own tongue. Their proficiency was such that they perceived I was a foreigner by my speech.

The writer feels very sure that many mutes retain more hearing than is supposed. This function remains, as it were, latent, because it is difficult to use it and hence develop it by ordinary vocal sounds of conversation. If, however, a speaking-tube is used, even in those who have long been mutes, more or less hearing is discovered. Of course what is said to them is often heard better than is supposed from their imperfect reproduction or translation of what is said. This is very much as it is when a foreign and unknown language is spoken to any one with good hearing. He hears what is said, but, being unaccustomed to utter the sounds of the foreign tongue, he cannot repeat them. So it is with the mute who hears a little: he cannot reproduce what he hears, or he does it imperfectly. If, however, a little patient labor be bestowed in speaking to him every day through a good ear-trumpet, his latent hearing can be developed, and it will become an immense aid in teaching lip-reading and articulation. For this purpose no better instrument exists than Maloney's otophone, already mentioned (p. 36).

HYGIENE OF DEAF CHILDREN.

1. There is a large number of children, all of whom have learned to talk, but who are too deaf to go to school or to learn anywhere by hearing the ordinary speech of other people. Many of these drift into institutions for the deaf and dumb. Those who enter these institutions soon lose their ability to speak or become very imperfect in it, from want of hearing others and from lack of practice. The inducement to talk soon goes when hearing what others say is no longer possible or is possible only with great difficulty.

2. There is also a large number of speaking children who hear very poorly, and will become semi-mutes or very imperfect users of speech unless carefully watched, both as to what they hear and how they speak. Such deaf children find it very difficult to keep up in their studies with children who hear well. They should, however, continue to go to school with hearing children, as it perfects their speech and their hearing if these are exercised as they must be in schools of hearing children. If they continue in such schools, however, they must be favored by seats near the teacher. The latter must be fully apprised of the amount of deficiency of hearing in any deaf pupils, and fully aroused to the importance of making some simple efforts at favoring hearing on the part of the deaf. It is a great advantage for the deaf child who hears some and knows how to speak to struggle on with children who hear and talk well, rather than to be consigned to a school for deaf-mutes, where it may lose all ability to hear, and will certainly lose the ability to talk.

In regard to the first class it may be said that at present there is no provision for their proper instruction. The poor of this class are forced to enter institutions for deaf-mutes, where they learn much and are also taught some trades. The rich of this class are now taught lip-reading and articulation, either by private tutors or in private schools. All of this class, both poor and rich, should be taught in a special way, or at least their instruction should be largely conducted, by making them hear through speaking-trumpets. This method should be begun early, as soon as the child gets too deaf to hear in the natural way. By doing so, the hearing may be not only kept from going entirely, but even improved.

In order that this plan of instructing the deaf child and improving his hearing and his speech may be appreciated, let me recall the mechanism of hearing and the anatomical basis of it.

Sound-waves falling on the membrana tympani force it inward and at the same time carry inward the malleus. The hammer bonelet, articulating with the incus, carries it inward also, and the latter forces the stapes into the oval window. Thus, then, we have sketched the mechanism of an inward excursion of the three auditory bonelets in the middle ear, articulated to one another in the so-called chain of ossicles. By such an excursion the labyrinth fluid and the auditory nerve in the labyrinth are impressed. It is now seen that hearing depends on the movement of joints, like those between bones in any other part of the body. If these grow stiff, as they do in catarrh of the middle ear, their mobility is impaired and hearing grows dull. If this impeded motion continues long, the nerve of hearing falls into disuse and fatty degeneration ensues. Just as in the case of ankylosis of any other joints, passive motion will overcome the impaired function. Instead of using our hands and arms to promote this passive motion, as in other large joints, in the ear the joints are movable only by sound-waves falling on the drum-membrane, upon the membrane of the round window, or upon the bonelets themselves if the membrana tympani be destroyed or perforated. In the deaf ear, the ordinary vocal sounds do not produce sufficient impression to overcome the ankylosis in the ossicles, and the patient is said to be deaf. If, however, ordinary sounds or louder ones be concentrated and conducted to the drum in more than ordinary quantity and intensity, the ankylosis is overcome temporarily and the child hears. If this is repeated systematically, the ankylosis, like such an impediment elsewhere, is overcome, and the hearing is made permanently better than it would be if allowed to remain unexercised.

I am fully convinced of such good result of a rational use of artificial means to convey sound to the deaf. It must be done patiently and systematically, but it has been done by placing the mouth close to the ear of the very deaf, by parents for their children, and by wives for their husbands. What may not be done for a number of deaf children brought together and taught by conducting the voice of the teacher to their ears by good ear-trumpets! They will be rescued from great deafness, and be improved in hearing and saved from being mutes.

In regard to the *second* class (p. 47) it may be said that, in order to make allowance for their defective hearing, a careful examination should be made in each case, in order to determine the extent of deafness.[1] This is done by finding out at what distance the voice of the teacher can be heard, in ordinary conversational tones. Tests should also be made to discover the distance at which consonant tones are heard by the deaf child. The child to be tested should be placed in front of the teacher, who should be in his accustomed place in the school-room. Then, with the ears alternately stopped, let the child be gradually brought to a point in the room where it hears and repeats the tests employed. Here the child should have its permanent seat. The tests to be employed are isolated words,—not sentences, because the latter can often be guessed by the context.

This test might be made by an expert, who can also examine the ears and discover whether any remediable disease be still present in the organs of hearing.

Dr. Samuel Sexton, of New York, has suggested that teachers often have defective hearing. This should be guarded against as far as possible by examining orally candidates for admission to the ranks of teachers in the public schools.

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[1] Dr. C. J. Blake, of Boston, found that in eight thousand seven hundred and fifteen cases of ear-disease two thousand one hundred and seventy-five, or twenty-five per cent., were children under fourteen years of age, all of them pupils in the public schools.

PART II.

THE EYE.

AFFECTIONS OF THE EYELIDS, LACHRYMAL APPARATUS, CONJUNCTIVA, AND CORNEA.

By G. E. de SCHWEINITZ, M.D.

AFFECTIONS OF THE EYELIDS.

Disorders of the eyelids naturally divide themselves into affections of the eyelid border and its tissues, new growths, affections of the muscles, affections of the cilia, vices of conformation, and congenital faults.

BLEPHARITIS.

Synonymes.—Many names are current for the various types of the disorder blepharitis. The late Prof. Frederic Horner[1] describes this affection under two headings: 1. Seborrhœa of the ciliary border, Blepharitis simplex, Blepharo-adenitis, Blepharitis ciliaris. 2. Eczema of the ciliary border (Blepharitis ciliaris, Blepharitis ulcerosa, Psorophthalmia, Lippitudo ulcerosa, Tinea tarsi, etc.). In a work by De Saint-Germain and E. Valude[2] the following classification is adopted: Scrofulous blepharitis and Eczematous blepharitis. The former variety is discussed under the two headings Hypertrophic scrofulous blepharitis and Ulcerative scrofulous blepharitis.

Definition.—Blepharitis is a general term which describes the various types of acute and chronic inflammation of the border of the lid.

Etiology.—Blepharitis is quite distinctively a disease of childhood, and often begins long before the age of puberty. It consists either in hyperæmia, a hypersecretion of the sebaceous glands, or an eczema of the lid-margin. Children with pallid complexion and light hair and of strumous constitution are particularly liable. It often follows an attack of measles. Lack of cleanliness and imperfect hygienic surroundings are exciting causes, while insufficient length of the skin of the upper lid, in some instances, according to Fuchs,[1] is a predisposing condition. Since Roosa's[2] investigations, refraction-error, especially hypermetropia, is known to originate the disorder in many instances, though certain writers, like Swanzy and Berry, while ready to believe that ametropia may keep up the affection if present, are disinclined to look upon this as a basal cause. Just as blepharitis is an independent disorder and may give rise to secondary changes in the other parts of the eye, so it may also originate from any long-standing hyperæmia of the conjunctiva, affections of the lachrymal apparatus, and coexisting nasal disease. Exactly what *rôle* bacteria play in the production of this complaint cannot be definitely stated. In the hands of Gallenga[3] cultures made from abscesses in a case of ciliary blepharitis gave origin to colonies of staphylococcus aureus and albus; and Widmark[4] found in the little abscesses at the roots of the lashes in cases of blepharo-adenitis staphylococcus pyogenes albus and aureus. Hirschberg,[5] under the name *vaccine blepharitis*, reports the appearance of vaccine vesicles on the middle free border of the lids of a man who played with children recently vaccinated; an inquiry instituted among nearly a thousand cases of vaccination done in this city discovered no similar instance.[6]

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[1] Handbuch der Kinderkrankheiten, C. Gerhardt, Tübingen, 1882.

[2] Traité pratique des Maladies des Yeux chez les Enfants, Paris, 1887.

Symptoms.—The disease may vary from a simple redness, the hyperæmia of the lid-border of some writers, to severe ulceration. In the milder types the ciliary margins are slightly thickened, red, and sore, while small scales and occasionally pustules appear, and if these be removed a yellowish sebaceous matter is seen beneath. The nutrition of the lashes is not seriously interfered with in this variety, which is often spoken of as *marginal eczema* or *blepharitis simplex*. To that form in which the eyelids under the slightest provocation grow red, the eyes weep and feel hot, the lids swell, and the conjunctiva is injected, while crusts are but scantily present, the term *vaso-motor blepharitis* has been applied. In another common and stubborn variety, which has its origin in the follicles of the eyelashes, the border of the lid is thickened, dusky, and congested; the edges are smeared with tenacious secretion; the lashes are gathered into little tufts by the collection of matter at their bases; scabs covering small ulcers and pustules appear, while the cilia are loosened and their removal is followed by bleeding; the lashes become misplaced, stunted, and deficient; cicatricial changes follow the subsidence of the swelling and cause slight eversion of the lids, as a result of which their borders are deprived of cilia, are raw and tender, and the appearance thus produced has received the name *lippitudo.* In the severest types, all four lid-borders may be attacked simultaneously, the lids are œdematous and highly congested, the margins beset with thick yellow crusts through which groups of lashes, glued together, push their way. Removal of these reveals beneath bleeding and ulcerated pits which extend inward as far as the tarsus; in short, the entire lid-border is lined with small crater-like abscesses. This *blepharitis ulcerosa,* as well as the less aggravated forms, not infrequently is associated with eczema of the auricle and nares, nasal catarrh, and diseases of the lachrymal apparatus, each standing in relation to the other either as cause or as effect, the whole forming what not inaptly has been described as a vicious circle.

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[1] Wien. Klin. Wochenschr., 1888, Nos. 38 and 39.

[2] Transactions of the American Ophthalmological Society, 1876.

[3] Annales d'Oculistique, xcviii. 51.

[4] Nord. Ophth. Tidsskrift, Nos. 1 and 2, 1888; Archives of Ophthalmology, December, 1888.

[5] Centralblatt f. prakt. Augenheilkunde, 1885, ix. See also another case of vaccine blepharitis, Hirschberg, Archives of Ophthalmology, xv.

[6] Under the title "Vaccinal Ophthalmia," S. T. Knaggs (Transactions of the Ophthalmological Society of the United Kingdom, i. 16) has described violent ophthalmia and later hypopyon-keratitis in a mother whose recently-vaccinated child inoculated her eye.

Prognosis.—The earlier the cases come under proper treatment, the more favorable the prognosis, and hence it is particularly in childhood that radical cures may be effected. Long-standing cases that have resulted in rounded, everted lid-margins, deprived of lashes, and with closed and misplaced lachrymal puncta, are rebellious to all forms of medication.

Treatment.—The local measures in the milder forms consist in the use of a lotion of boric acid and the application to the edges of the lids of a salve of the yellow oxide of mercury (gr. i–ʒi). In that variety, however, described under the term vaso-motor blepharitis the use of salves is contraindicated, and the best results, according to Koenigstein,[1] are reached by douching the closed lids with water at a temperature of 60° F. from a vessel raised a short distance above the head of the patient, the fluid being conducted through a small apparatus in the form of the rose ordinarily seen upon watering-cans. Whenever scales are present these must be taken away, either by means of warm alkaline solutions, of which bicarbonate of sodium (gr. viii.–f ℥i) and biborate of sodium (gr. iv–f℥i) are the best, with a five-per-cent. solution of chloral, as recommended by Gradle,[2] or with forceps. Gradle advocates a three-per-cent. mixture of milk of sulphur with vaseline and the addition of three per cent. of resorcin. During severe inflammatory attacks, and even in old cases, results surprisingly beneficial follow the removal of all the lashes with a cilia-forceps. In ulcerated blepharitis, after the removal of the crusts and loose cilia the ulcers should be painted with a solution of nitrate of silver, and may even be cautiously touched with the point of a mitigated stick. Michel advises that unguentum diachylon Hebræ with oil of sweet almonds be spread upon pieces of lint so shaped as to cover the lids, and containing apertures through which the cilia, if present, may pass, and allowed to remain overnight. Kroll recommends that Pagenstecher's ointment (hydrarg. oxid. flav., gr. i; cosmoline, ℨi) be rubbed in until the scales are removed, and then the lid-border pencilled with nitrate of silver, one to fifty. In the hands of Schweigger iodine on the edges of the lids has proved beneficial. In all cases the lachrymal passages and the nasal cavity should be examined, and, if epiphora exists, the canaliculus slit. Any error of refraction, after the eyes have been carefully atropinized, is to be corrected with suitable glasses.

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[1] Die Behandlung der häufigsten und wichtigsten Augenkrankheiten, Wien, 1889.

[2] Medical News, February 8, 1890.

The best constitutional measures are quinine, iron, especially in the form of the iodide, cod-liver oil and malt, together with appropriate nourishing diet. Fresh air and a daily salt bath are highly desirable.

PHTHIRIASIS PALPEBRARUM.

Synonymes.—Blepharitis pediculosa, Phthiriasis ciliarium.

The pediculus pubis (crab-louse), besides its seat of predilection, occasionally infests the eyebrows, and very rarely the eyelashes. The rarity of the affection is attested by the fact that Hirschberg[1] among forty thousand cases of eye-disease met with only three instances. Despagnet[2] during twelve years of Galezowski's service found but two recorded examples; and among more than ten thousand patients in the service of Prof. William F. Norris at the University Hospital only two instances have been observed.[3]

Symptoms.—On superficial examination the lashes appear to be covered with small, dark spots like grains of powder, which upon closer inspection resolve themselves into the lice clinging closely to the lids, while the eggs, darker in color, are fastened with great regularity along the roots of the cilia; in many instances the parasites are buried head-foremost in the hair-follicles. Their presence causes severe irritation, and the case may be mistaken for an ordinary marginal blepharitis. A magnifying lens will at once clear up the diagnosis.

Treatment.—Cleanliness, together with balsam of Peru, or mercurial ointment, or a cautious pencilling with a solution of corrosive sublimate, will remove the intruders.

HORDEOLUM.

Synonymes.—Stye, Hordeolosis.

Hordeolum is an inflammation of the tissues of the edge of the eyelid or of one of its sebaceous glands. Exposure to artificial light, to dust, and cold winds are the most usual exciting causes. Eyes that are subject to styes are often ametropic, hypermetropia being the most usually observed refraction-error. Styes tend to appear in crops, and occasionally produce great swelling and œdema of the lid until an appearance like the commencement of purulent ophthalmia is produced. By observing the circumscribed character of the swelling and the absence of purulent secretion from the conjunctiva, an error may be avoided. In like manner a stye may simulate an acute inflammation of the lachrymal sac. Usually the small inflamed swelling on the edge of the lid increases in size for a few days and then resolution or suppuration occurs.

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[1] Berlin. Klin. Wochenschrift, 1882, xix.

[2] Recueil d'Ophtalmologie, November, 1887, p. 674.

[3] University Medical Magazine, March, 1889.

Treatment.—Warm fomentations, especially in the form of hot-water compresses, give speedy relief. If suppuration occurs, the contents should be evacuated with a knife. Saturated solutions of boric acid, according to Abadie, cause a rapid cure. A stye occasionally may be aborted by painting its surface with collodion, and the vigorous application of a salve of the red or yellow oxide of mercury has produced the same result. The refraction of the eye should be examined in all cases, and if this is anomalous, as it usually is, the proper correcting glass is to be ordered. Associated conjunctivitis must be treated with a mild astringent or antiseptic wash, while internally iron and quinine, and, if the styes come in groups, the sulphide of calcium, as recommended by Webster, may be exhibited. Constipation must be relieved by suitable remedies.

CHALAZION.

Synonymes.—Meibomian cyst, Tarsal tumor.

A chalazion is a small tumor or retention-cyst due to a chronic inflammation of a Meibomian gland together with the surrounding tissue.

The etiology of these little growths is obscure. They may be associated with inflammation of the border of the lid, which aids in closing the duct of the Meibomian gland. Poncet and Boucheron[1] have described microbes in connection with chalazia, though Vassaux[2] and other observers have failed to find them except in such as already had undergone suppuration. They are more common in adolescence than in very young children or in old age, but are not infrequently found in infants.

Pathological Anatomy.—According to Felix Lagrange,[3] the chalazion has three periods of development,—retention of the products of the Meibomian gland; consecutive adenitis and periadenitis, with destruction of the cartilage; and passage of the tumor to the conjunctiva (internal chalazion) or to the skin (external chalazion). Lagrange believes that microbes play only a secondary part in the production of the affection. If examined, the cysts contain pus, puriform fluid, and cholesterine crystals, together with surrounding granulation-tissue. There is no true cyst-wall.

Symptoms.—The tumor grows slowly and forms a firm swelling, tightly attached by its under surface to the tarsus; over it the skin is usually freely movable; occasionally adhesions between it and the integument occur. On the conjunctival surface of the lid a bluish patch marks its position. Suppuration may take place in the cyst, and, like styes, these tumors tend to come in crops.

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[1] Bull. et Mém. de la Soc. Franç. d'Opht., Paris, 1886, iv. 88–91.

[2] Ibid.

[3] Archives d'Ophtalmologie, May–June, 1889.

Diagnosis.—A chalazion is to be distinguished from an ordinary sebaceous tumor by the firmness of its attachment to the tarsus, and, if it suppurates, from a stye by the more circumscribed character of the inflammation. It may be mistaken for a round-celled sarcoma of the lid, an interesting instance of which has been recorded by Randall,[1] where the microscope was necessary to settle the diagnosis.

Treatment.—If the growth points towards the conjunctival surface, it is to be removed from this side; if not, the incision should be made over it in the skin parallel with the muscle-fibres, and the mass carefully dissected out, care being taken not to perforate the conjunctiva. Operation is facilitated by having the lid steadied with a clamp (Desmarres, Snellen, or Knapp). The assiduous use of hot water and the application of yellow oxide of mercury salve are often resorted to with the hope of producing resolution. Removal by the knife or scoop is the only practical measure, though the local means above mentioned may be useful to aid in the dissipation of any inflammatory thickening which remains after the operation.

Post-variolous Ulcers of the Eyelids.—A favorite spot for the pustule of small-pox is the border of the lid. Not only may this result in the ordinary scar, the sequel of cicatricial contraction, with loss or faulty position of the cilia, but occasionally a long time after the eruption has disappeared ulcers remain which stubbornly resist treatment. Horner (*loc. cit.*) has seen such variolous ulcers ten years after the original disorder.

Furuncles are not infrequently seen in children, especially in the outer half of the eyebrow as well as within the tissue of the lid. They occur as a red swelling, move with the skin, are unattached to the bone or periosteum, and are soon capped with a yellow point of suppuration. They should be treated by warm fomentations or flaxseed poultices, and early incision shortens their existence.

Œdema of the Eyelids.—This is an affection characterized by an œdematous condition of the cellular tissue of the eyelids, which may be fugitive or persistent, and is not infrequently recurrent. R. W. Doyne[2] reports the case of a girl aged fifteen, the subject of migraine and myopia, with recurring swelling of the upper eyelids, the œdema sometimes standing out on a level with her brows. Mr. Gunn[3] believes these cases are all of the nature of urticaria. They may appear in connection with the establishment of menstruation, but sometimes occur in otherwise healthy children, spontaneously and without cause, and in boys, as reported by W. J.

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[1] Transactions of the American Ophthalmological Society, 1887.

[2] Transactions of the Ophthalmological Society of the United Kingdom, viii. 41.

[3] Ibid.

Collins[1] and others. Œdema of the eyelids, when present as part of a general condition—renal or cardiac—or from injury, should be distinguished from these cases of transient œdema by observing the history of the case. If the case calls for treatment, this may consist in bathing the parts with dilute lead-water and laudanum and the internal administration of Basham's mixture. Œdema of the eyelids when associated with general disease necessarily receives the same treatment as that directed to the relief of the constitutional disorder.

ERYSIPELAS may attack the eyelids, and in this situation does not differ materially from this disease in other portions of the body. It is much less seldom peculiar to this region than it is as part of an attack of general facial erysipelas. It is one of the causes of orbital cellulitis.

PHLEGMONOUS INFLAMMATION (Pseudo-Erysipelas) and the formation of *lid-abscesses* of a mild type is not an unusual disorder during the early months of childhood: it is confined almost exclusively to the upper lid, which becomes red and swollen, and in a few days palpation reveals the presence of pus. In other cases a much more severe type is manifest. It begins with the formation of a pustule, quickly followed by great swelling and accompanied by high fever. The skin and subcutaneous tissue may become sphacelous after the formation of one or more pustules of dark color (*œdème malin* of French writers). Hilbert[2] has described cases of a peculiar gangrene of the lid in children who were well nourished, rosy, and never before ill,—cases which bore no resemblance to noma, malignant pustule, œdema malignum, phlegmon, or multiple cachectic lid-abscesses.

Etiology.—If secondary lid-abscesses, and especially acute conjunctival processes, are excluded, the origin of these cases may be looked for either in a traumatism or as the result of an infectious process, although the causes are by no means always evident. Lid-abscess has been reported as a sequel of influenza by Landolt. I have observed similar instances.[3] In many cases contact with infectious disorders in animals gives rise to the disease,—an explanation not so readily applied to children as to adults. Michel, however, quotes a case in which a child was suddenly seized with this affection of the lids after coming in contact with straw that had been used for horses suffering with glanders.

Treatment.—This naturally directs itself to lessening the constitutional disturbance and to the local condition. The latter should be treated with warm fomentations, early incision, and antiseptic dressing. Subcutaneous injections of carbolic acid have proved useful in the hands of Horner, and Delens has reported success with similar injections of iodine. If deformity of the lid result from the disease, this must be restored by a plastic operation.

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[1] Transactions of the Ophthalmological Society of the United Kingdom, viii. 41.

[2] Vierteljahrschrift für Dermatologie, Wien, 1884, xi. 117–119.

[3] Medical and Surgical Reporter, March 15, 1890.

SYPHILIS OF THE EYELIDS.

Syphilitic affections of the eyelids may be divided into ulcerations and eruptions, and may exist as the primary sore or in the form of secondary or hereditary manifestations. A chancre upon the lids may have its seat on any portion, but the delicate skin of the ciliary border is the favorite region. Among one hundred and eighteen cases of all ages collected by Alexander,[1] the skin of the eyelid was affected sixty-five times, the tarsus sixteen times, and the conjunctiva thirty-seven times. Associated with the local lesion in the eyelids, the lymph-glands in front of the ear and at the angle of the jaw are much enlarged. Contagion usually occurs from an infected attendant, not infrequently the mother. J. V. Solomon[2] has recorded an instance of an indurated syphilitic ulcer at the inner end of the eyelid edge, which occurred in an infant eight months old, who had been inoculated by an aunt at that time suffering with specific ulceration of the tonsils. A primary syphilitic sore on the upper eyelid of a boy aged six is described by Mackay.[3] A papular eruption appeared six weeks after the initial lesion; the origin of contagion was probably from a diseased mother. The frequency with which chancres in the eyelids of children have occurred (in ninety-four cases collected by De Beck,[4] ten were found among infants or young children) led M. Baudry,[5] of Lille, to investigate their etiology. In one case, a female child twenty-two months old, an indurated chancre appeared on the free edge of the lower eyelid. Investigation showed that the infant was nursed by a woman who wiped its eyelids with her saliva, the woman being at the time the subject of syphilitic ulceration of the mouth. Inquiry elicited the fact that women among the peasant folk were accustomed to cleanse the eyelids of children in this manner when they were glued together with discharge from the conjunctival cul-de-sac.

Infants the subjects of hereditary syphilis are sometimes affected with an eruption of papules upon the external surface of the lids, which appear several weeks after birth. Hutchinson[6] describes a form of blepharitis in which sharp-bordered ulcerated plaques appear at the roots of the cilia, and instances in which absence or falling out of the eyelashes was a manifestation of hereditary syphilis in children.[7]

Treatment.—Locally the ulcer may be dressed with black or yellow wash, while internally the ordinary antisyphilitic remedies are to be exhibited. Especially efficacious is the employment of unguentum hydrargyrum spread upon flannel in the form of a binder.

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[1] Syphilis und Auge, Wiesbaden, 1889.

[2] British Medical Journal, 1863; ibid., 1885, ii. 62

[3] Edinburgh Medical Journal, September, 1888.

[4] Hard Chancre of the Eyelids and Conjunctiva, Cincinnati, 1886.

[5] Mémoires de la Société Française d'Ophtalmologie.

[6] Ophthalmological Hospital Reports, ii. 258–283.

[7] See also Barlow, quoted by Alexander, loc. cit.

TUMORS AND HYPERTROPHIES OF THE EYELIDS.

Milium.—Milia are small sebaceous elevations situated in the skin, varying in size from a pin-point to a pea, and find their favorite seat in the forehead and about the eyelids. They are common in children about the age of puberty. They consist in an accumulation of sebum within the distended but closed sebaceous glands; in this respect they differ from a comedo, in which the duct of the gland is patulous. They are caused by improper care of the skin, and may be connected with general constitutional disturbances, dyspepsia, and constipation. They should be opened with a knife or needle and the contents removed.

Sebaceous Tumors (Atheroma) are not usually found upon the eyelid, but occur especially in the outer portion of the eyebrow. (See page 69.)

Molluscum Contagiosum.—This is a disease of the sebaceous glands, or, according to some observers, of the rete mucosum, in which rounded papules, usually about the size of a pea, but often much smaller, appear in the skin of the eyelids. The common seat of the disease is upon the face, and especially the eyelids, but it often appears on the neck, breasts, or genitalia. Each little prominence may have the normal pinkish color of the skin, or it may be of a distinctly waxy hue, with a flattened summit which contains a depression. This disease, usually non-inflammatory, occurs chiefly among ill-nourished children, and may appear as an epidemic in homes and asylums. Two such epidemics have been well described by Mittendorf.[1] The contagious nature of the disorder has been largely entertained, but so high an authority as Duhring[2] denies that the evidence on this point is conclusive, while Neisser[3] believes that it is really an epithelioma contagiosum caused by a parasite belonging to the class of *coccidia*. The contents of the bodies is a yellowish material which consists of fat and fatty epithelial cells.

Treatment.—Each molluscum should be incised and the contents forced out. Nitrate of silver may be applied to the cavity, but this is not usually necessary.

Ptosis Lipomatosa is an affection of the lids in which these drop over the cornea, owing to an abnormal accumulation of fat in the connective tissue. The deformity is considerable, and gives the patient a disagreeable expression. The fat should be removed through a horizontal incision. In one case reported by Schell[4] seventy-one grains were thus taken away. A reasonably good result may be anticipated; but in a case recorded by Norris,[5] although the removal of the fatty tissue improved the appearance of the patient, the levator palpebrarum failed to regain its power, and full activity in the movement of the upper lid was not secured. Care must be taken not to mistake œdema of the lid for a circumscribed tumor, an error readily avoided by palpation, which will reveal a characteristic difference in the resistance of the two affections.

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[1] Transactions of the American Ophthalmological Society, 1886.

[2] Diseases of the Skin, 2d ed.

[3] Vierteljahrschrift f. Dermatol. u. Syphilis, 1888.

[4] Transactions of the American Ophthalmological Society, 1885.

[5] Ibid.

ANGIOMAS (Nævi) when they appear in the eyelids may exist either as simple bright red spots or in the form of cavernous growths. They are congenital tumors. It is important that they should be dealt with early in their existence, as they may take on rapid growth with the development of the child and extend far into the orbit. Such operative interference as promises the least subsequent deformity of the lid should be practised. When small, they may be readily excised; if of the larger variety, some measure which destroys their blood-vessel structure will be the proper procedure. The methods employed are the galvano-cautery, which yields the best results; injections of liquor ferri subsulphatis, a method which has been followed by sloughing of the lid; and cauterization with red-hot needles or with nitric acid.

PLEXIFORM NEUROMA is an unusual form of tumor occasionally seen in the eyelid, consisting of a convoluted mass somewhat corded beneath the skin. In a collection from the literature of seventeen such cases four were noted as occurring in the eyelid.[1] The growth is entirely benign in its nature, and its removal by an ordinary dissection with antiseptic precautions is unattended with difficulty.

MOLLUSCUM FIBROSUM is a connective-tissue new growth, either sessile or pedunculated, situated beneath the skin. The tumors may appear singly or in great numbers all over the body, and occasionally are seen upon the eyelids. They are found at any time of life, but especially in childhood. In a remarkable case of this kind described by Michel the whole body was beset with these tumors, the largest of which grew from the upper lid and hung down below the lower eyelid.

SARCOMA occurs as a primary tumor in both the upper and lower lids of children, and has been seen as early as the tenth month of life. It appears as a somewhat elastic growth, over which at first the integument is movable, but it tends to rapid growth, ulceration, and involvement of the orbit. As has already been pointed out (page 55), this growth may be mistaken for a Meibomian cyst. Indeed, Samelsohn[2] has reported one instance occurring in a child two years of age, which the microscope showed to be a sarcoma of the round-cell variety which had its origin in a proliferating tarsal cyst. The prognosis is grave. Thus, Van Duyse[3] has recorded a case of myxo-sarcoma in a seven-year-old child, which followed a contusion. After extirpation at the end of three months the local return required another operation. Four months later a second return developed, and the whole orbit was cleared out; in spite of which, a third return took place, and the tumor grew to the size of a child's head. Early excision, which is the only proper treatment, unfortunately, is not able in most instances to prevent the two chief dangers of this form of malignant tumor, especially in this region,—recidivity and metastasis.

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[1] Unpublished Inaugural Dissertation, "Painful Tumors, with Special Reference to Neuromas," Prize Thesis, University of Pennsylvania, 1881.

[2] British Medical Journal, 1870, ii. 706.

[3] Annales d'Oculistique, May and June, 1889.

LUPUS may secondarily attack the eyelids in its destructive march from a seat of origin in the face, and occasion ulceration of the lids, ectropion, or ankyloblepharon.

LEPRA, according to Michel, who quotes the observations of Carron du Villards, may appear in the eyelids among its earliest manifestations: Bull and Hansen[1] have observed the first symptoms in leprosy to be the falling out of the hair of the eyebrows, and E. Lopez[2] analyzed forty-five cases of leprosy and found absence of the eyebrows and lashes the sole ocular lesion in the youngest subject, ten years of age.

ELEPHANTIASIS ARABUM has appeared in the upper lid in consequence of an injury. C. du Villards saw this affection in a seventeen-year-old girl on the left upper eyelid as the result of a contusion. Elephantiasis may be congenital. Van Duyse[3] records an example in a girl of eight.

ELEPHANTIASIS TELEANGIECTODES, or a disease which consists in an hypertrophy of the skin and the connective tissue, together with fatty tissue and distended vessels, has been observed in the upper eyelid as a congenital affection. Cases are on record by Pauli,[4] Von Graefe,[5] Liston,[6] and other observers.[7] The growth should be removed by the knife in such degree as is permissible with the preservation of the form of the lid.

BLEPHAROSPASM.

Blepharospasm, or an involuntary contraction of the orbicularis, usually clonic, but sometimes tonic in its nature, is a constant symptom of diseases of the cornea and conjunctiva, and will be referred to again under these headings. According to Schubert,[8] it may arise in the manner of a reflex action in individuals whose susceptibility is increased by hereditary influence, anæmia, over-exertion, etc., so that a slight irritation sends a stimulation to the facial branches of the orbicularis. Fissure at the angle of the lids is given by Koller[9] as a cause of reflex blepharospasm. As has been known since Von Graefe's historic case, a persistent lid-cramp occurs in children, unrelieved for weeks at a time, and when the eyes are finally opened there may be blindness, temporary in its character and with normal ophthalmoscopic appearances, or loss of vision, permanent, and with gross changes in the eye-ground. In a case recorded by Silex,[1] a scrofulous child two and a half years old kept the eyes closed for twelve weeks, and was blind for twelve days, with normal ophthalmoscopic appearances, sight returning on the thirteenth day. The reporter argues in favor of the blindness being a species of cortical blindness, owing to the long absence of peripheral stimulation. Samelsohn,[2] who among sixty thousand cases of eye-disease has observed five instances of blepharospasm lasting for months and followed by loss of sight, seeks for an explanation of the blindness which follows this long-continued cramp-like shutting of the lids, in an example of the forgetting volition (*Vergessenwollens*) of the sensory perceptions analogous to the intentional suppression of the sense of sight of an eye in alternating strabismus. This explanation would apply only to such cases as have normal ophthalmoscopic appearances and recover, not to the instances of which Samelsohn has seen two, in one of which a true atrophy and in the other a glaucomatous atrophy was present. In these cases a peripheral cause must be sought for, and the theory of Von Graefe, which explained the blindness by the abnormal pressure of the lids, is applicable.

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[1] Leprous Diseases of the Eye, 1873.

[2] Archives of Ophthalmology, December, 1889, p. 404.

[3] Annales d'Oculistique, t. ii., 1889.

[4] Schmidt's Jahrbücher, xxi. 84.

[5] Klin. Monatsbl. f. Augenheilkunde, 1863.

[6] Canstatt's Jahresbericht, ii. 153.

[7] Consult Graefe u. Saemisch, Handbuch des Augenheilk., iv. 409.

[8] München. Med. Wochenschr., No. 28, 1887.

[9] Transactions of the American Ophthalmological Society, 1888.

Not infrequently children in their early school-days are affected with an undue winking of the eyelids, associated, perhaps, with jerky movements of the facial and other muscles. This is the form of nervous disorder which has been called habit chorea by Weir Mitchell,[3] habit spasm by Gowers.[4] Almost invariably blepharitis, follicular conjunctival catarrh, and anomalies of refraction, usually hypermetropia, will be found to be the exciting causes. In a series of cases in my own practice the correction of the refraction-error and the treatment of the conjunctival disorder were productive of the most happy results, when the ordinary antichoreic remedies had proved valueless.[5] In persistent forms of lid-cramp hypodermic injections of morphine will relieve the peripheral (trigeminus) irritation, and in stubborn varieties section of the supra-orbital nerve has been performed. The general condition must be relieved by appropriate remedies.

Lagophthalmos, or an inability to close the eyelids, as usually seen, is the result of paralysis of the facial nerve, but occurs also in connection with tumors of the orbit, in those rare instances of exophthalmic goitre which are seen in children, and with staphyloma. As a congenital defect, when the lids themselves are wanting and the entire orbit is divested of any covering for the bulb, the highest grade of lagophthalmos occurs. The cornea may suffer from ulceration, owing to exposure,—a danger greatly increased if with the affection of the facial nerve paralysis of the trigeminus also exists, but rendered less likely to occur in the single palsy, because, when the effort to close the lids is made and during sleep, the eyeball is rotated upward under the upper lid, owing to the associated action of the superior rectus.

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[1] Klin. Monatsbl. f. Augenheilkunde, February, 1888.

[2] Deut. Med. Zeitg., No. 15, 1888, and Centralblatt f. prakt. Augenheilkunde, February, 1888.

[3] Nervous Diseases, 2d ed., 1885.

[4] A Manual of Diseases of the Nervous System, 1888.

[5] Transactions of the County Medical Society of Philadelphia, 1888.

Treatment.—In paralytic lagophthalmos the primary cause of the disorder must be treated; in the non-paralytic varieties and in any form in which the vitality of the cornea is threatened, the operation of tarsorrhaphy, which consists in uniting the margins of the upper and lower lids in the neighborhood of the external commissure, should be employed.

AFFECTIONS OF THE CILIA AND CILIARY BORDER.

Trichiasis, Distichiasis, Entropion.—Trichiasis is that condition in which the lashes are turned inward against the eyeball; distichiasis, where incurved supplementary rows of cilia are developed from the intermarginal part close to the opening of the tarsal glands. The most usual cause for trichiasis in children is disease of the lid-border,—the various forms of blepharitis. Distichiasis in rare instances appears as a congenital affection; sometimes associated with other defects, as in the instance observed by Schweigger[1] where epicanthus and ptosis existed. The supplementary row of cilia is produced when the ordinary follicles are generated, although it is quite probable that in some instances the supernumerary lashes do not appear until the age of puberty, the extra hair-follicles having remained quiet until that time, now springing forth under the same impulse which the growth of hair elsewhere receives.

Entropion, or inversion of the lid, is conveniently divided into muscular, organic, and the so-called bulbar entropion. The former variety is seen occasionally at birth, owing to undue development of the orbicularis, and is present as spasmodic entropion in conjunctivitis, keratitis, and when foreign bodies lodge on the cornea. By far the most common causes of organic entropion are granular lids and essential shrinking of the conjunctiva. It also follows diphtheritic ophthalmia. The bulbar entropion appears when the eyeball is shrunken or even absent (anophthalmos), and there is consequent falling in of the lids.

Treatment.—*Trichiasis.*—If not too numerous, the faulty lashes may be removed with a cilium-forceps, and when they reappear the procedure repeated. Destruction of the hair-follicles with galvano-puncture is recommended by Mitchell[2] of Missouri, Benson[3] of Dublin, and other surgeons. Strangulation of the roots of the incurved lashes, when only a few are out of order, may be accomplished by means of a fine subcutaneous ligature (Snellen); or complete removal of them by excision of the corresponding portion of the ciliary margin, a practice to be deprecated if the trichiasis is at all extensive. Finally, in severe cases, the whole ciliary border must be transplanted by either the single or double transplantation operations. Those most in vogue are the methods devised by Jaesche and modified by Arlt, and the double operations of Spencer Watson, Dianoux, Gayet, and others, or the more recently advocated method of Van Milligen,[1] which consists in the transplantation of a piece of mucous membrane from the lip into the intermarginal space after the lid has been split.

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[1] Hand-Book of Ophthalmology, 1878.

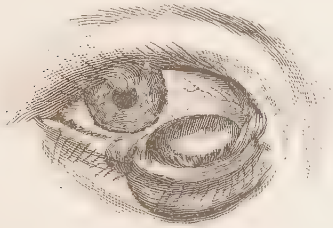
[2] Klin. Monatsbl., April, 1882, quoted by Swanzy.

[3] British Medical Journal, 1882.

Entropion.—In temporary entropion good results may be obtained by painting the lid with collodion, which by its contraction draws out the inverted border, or by simply fastening this with a strip of plaster. The same effect may be obtained by pinching up a longitudinal fold of skin and muscle with a serre-fine and keeping it in place, occasionally changing the position of the instrument to avoid irritation. In spasmodic entropion excision of a transverse fold of skin and muscle and stitching the edges together may be practised, while in the organic forms more decided measures are necessary, and those most frequently employed are such as have already been referred to in connection with trichiasis; in addition to which may be mentioned the Streatfeild-Snellen operation of grooving the tarsus, and the modifications devised by Green, Hotz, Berlin, Panas, and other surgeons. (For the methods of performing these and all operations upon the lids, see pages devoted to describing operations.)

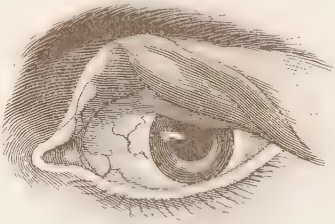
Ectropion, or eversion of the lid with exposure of the conjunctival surface, occurs most frequently in the lower, but is also seen in the upper lid. This may be partial, or there may be complete eversion with displacement of the puncta lachrymalia. The disorder is usually divided into the acute (e. musculare, e. spasmodicum) and the chronic form, or that which results from organic changes. The most usual causes of acute ectropion in children are ophthalmia neonatorum, and diseases of the cornea associated with blepharospasm, where the lids, during examination or in spells of crying, become everted and remain so until replaced. One form of muscular ectropion is always seen in facial palsy, during which the lower lid is partially everted. The common causes of the second form of ectropion are

Fig. 1.



Ectropion the result of a wound from the tine of a fork. (Children's Hospital.)

Fig. 2.



Ectropion of upper lid after injury to the brow. (Philadelphia Hospital.)

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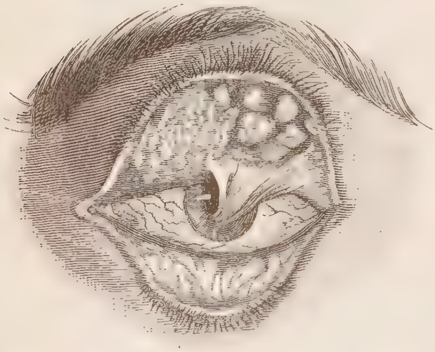
[1] Ophthalmic Review, 1887, p. 309; also Centralblatt f. prakt. Augenheilkunde, July, 1889.

wounds, especially such as are produced by the laceration of dog-bites or by the lid being caught upon a sharp instrument, by burns and subsequent cicatricial contraction, by ulceration of the lids, and by caries of the orbital border and the malar bone.

Treatment.—This must vary according to the character and cause. In the spasmodic forms it is sufficient to replace by manipulation the everted lid and treat the conjunctival or corneal disease which caused the trouble. If there be eversion of the punctum lachrymale, the canaliculus should be slit and the lachrymal passages probed. In the organic forms of ectropion a plastic operation which will relieve deformity and restore the lids to a normal position should be undertaken. A great variety of these procedures have been described and performed, but the plan adopted must be modified according to the existing deformity. Those commonly employed are such as include the vicious cicatrix in an excision, the remaining gaps being supplied by flaps taken from the surrounding tissue. Among these may be mentioned the methods of Adams, Wharton Jones, Arlt, and Richet. Complete destruction of the lid requires for its reformation a blepharo-plastic operation, which consists in the transplantation of a flap removed from some adjacent part to which it remains attached by its base, a method, however, which is being superseded by that introduced by Lefort and advocated by Wolfe, where a non-pediculated flap is taken from the arm to supply the defect. (See pages devoted to description of operations.)

SYMBLEPHARON, or a cohesion between the eyelids and the ball, may be complete or partial. It occasionally occurs as a congenital defect, owing to an imperfect separation of the cutaneous folds which form the eyelids, on account of failure in development of the ball or functioning of the eye-muscles. The most usual causes are injuries, especially burns with acids or lime. Symblepharon also follows diphtheritic conjunctivitis, trachoma, pemphigus,[1] and occasionally purulent ophthalmia.[2] The attachment may be merely slight bands between the conjunctival surface of the lid and ball, or the cornea also may be involved in the cicatricial union, in which case vision is materially disturbed. It is the lower lid which is most usually involved in the process; the upper may also participate (see Fig. 3).

FIG. 3.



Symblepharon of upper lid following purulent ophthalmia. (Philadelphia Hospital.)

ANKYLOBLEPHARON is that condition in which the borders of the two lids have grown together. This may be congenital or acquired, and, like symblepharon, either partial or complete. When merely the outer angles of the lids are involved, the disorder has received the name *blepharo-phimosis*, while sometimes only the middle portions of the lid-borders are attached to one another, as, for example, in a case recorded by Von Hasner,[1] where this attachment occurred in the form of a thread as a congenital defect. The same causes which operate in the production of symblepharon are here active. Arlt[2] and Dujardin[3] have described varieties in which the vicious union was not due to a growing together of the lids, but probably to the organization of a membrane the result of croupous conjunctivitis.

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[1] Czermak, Wien. Med. Wochenschr., 1888.

[2] University Medical Magazine, January, 1890.

Treatment.—After an injury or a disease which is likely to be followed by one of these results, scrupulous care must be exercised to avoid the complication. During the formation of granulation-tissue this should be repeatedly broken up with a probe, and adhesions occasionally may be prevented by introducing between the lids and the ball a piece of gold-beater's skin. If the attachments have formed and are slight, these may be cut through, and readhesion prevented in the manner just stated. In extensive symblepharon a formal operation must be done, and many methods have been devised, among the best of which may be mentioned Arlt's, in which the mass of adhesion is reversed, Teale's, where the raw surfaces left after the separation of the lid from the ball are covered by flaps from the neighboring healthy conjunctiva, and Prof. Wolfe's procedure of transplanting rabbit's conjunctiva. In ankyloblepharon the adhesion should be divided with a fine knife. Blepharo-phimosis is corrected by canthoplasty. (See pages devoted to description of operations.)

SUDDEN TURNING GRAY OF THE EYELASHES.—Sufficiently definite observations have shown that occasionally the hairs of the head can be deprived of their color suddenly, either universally or in places, forming locks of gray or white hair. Hirschberg[4] has recorded an instance in which the eyelashes of a girl, aged fourteen, turned white without apparent cause in fourteen days. The child formerly had been under his care for styes and phlyctenular disorders, but otherwise was in good health. The discoloration took place in the middle third of the upper lid, while in the under lid white bundles were commingled with dark ones. I have seen an exactly similar case in a healthy, dark-haired young woman of eighteen. Within one week the middle portion of the cilia of the right upper eyelid turned perfectly white, while single white lashes alternated with dark ones in the lower lid. There was no reason to believe that this sudden change in color had been produced by artificial means.[5]

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[1] Prag. Zeitschr. f. Heilkunde, 1881–2, ii. 429.

[2] Ophthalmological Society, Heidelberg, 1881; see Archives of Ophthalmology, vol. xvi.

[3] Rev. Clin. d'Ocul., November 5, 1886.

[4] Centralbl. f. prakt. Augenheilkunde, January, 1888.

[5] University Medical Magazine, March, 1889. Since writing this I have learned that the original color has returned to the eyelashes.

CONGENITAL DEFECTS OF THE EYELIDS.

Congenital Ptosis.—This is not an uncommon affection, and may be either unilateral or bilateral. The lid droops over the eyeball, and its edge covers the upper border of the pupil, its middle, or sinks still lower, but cannot be elevated above these points. Horner (*loc. cit.*) has observed this affection in the first days of life, and has seen it in three generations of a family. It may be associated with limitation of the movements of the superior rectus, as well as with vices of conformation in other organs of the body. In a case under my own care ptosis of the left eye was combined with divergent squint from paresis of the internal rectus. The patient was six years old. The defect had been present since babyhood, and when the child attained the age of four he became the subject of epileptiform convulsions. The fundus of each eye was normal. Gunn[1] has recorded a remarkable case of peculiar associated movements of the affected lid. When the jaw was moved to the right laterally, the left upper lid was raised, or, in other words, there was contraction of the levator palpebræ in connection with the external pterygoid.[2]

Under the name *ectopia tarsi* J. T. Streatfeild[3] has described, as a congenital defect, a sloped condition of the palpebral fissures, the lids being apparently drawn down wholly and evenly at the inner or nasal side.

Etiology.—Ptosis usually is divided into two varieties,—one in which a positive hypertrophy of the connective tissue exists, and one in which the drooping is due to absence or imperfect development of the levator palpebrarum, or to paralysis of this muscle. Its presence also has been attributed to the pressure of the forceps during birth, a cause which Horner denies, inasmuch as this affection is seen during the first days of life without any mark of the instrument upon the face of the child, and because it occurs through several generations of one family. It furthermore frequently is associated with other congenital defects. Ptosis the result of paralysis of the oculo-motor nerve is referred to on page 135 of this volume (article on diseases of the eye).

Treatment.—It is usual to attempt to remedy this defect by the removal of an elliptical piece of skin. To avoid the risk of shortening the lid which attends this operation, methods have been devised for producing cicatricial bands by means of subcutaneous sutures passed from the brow to the tarsus. Among these may be mentioned the plans of Bowman, Pagenstecher, De Wecker, and the more recently devised method of Panas.

Epicanthus.—Von Ammon[4] gave this name to a somewhat rare affection in which, owing to an excessive development upon the bridge of the nose, a fold of skin passes from the inner end of the brow to the side of the nose and covers the internal canthus. It is usually bilateral, and generally associated with congenital ptosis.

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[1] Transactions of the Ophthalmological Society of the United Kingdom, London, 1883, iii. 283–287.

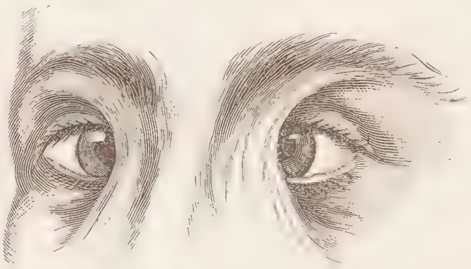
[2] See also a paper by O. Bull, Archives of Ophthalmology, 1888, vol. xvii., on "Synchronous Movements of the Upper Lid and Maxilla."

[3] Ophthalmological Hospital Reports, 1874–76, vol. viii.

[4] Klinische Darstellungen der Krankheiten und Bildungsfehler des menschlichen Auges, Berlin, 1838.

Horner (*loc. cit.*) has pointed out that an examination of this region in new-born children might readily lead to the belief that a low grade of epicanthus was very common. This, however, lessens and disappears as the child develops. The fact that the free border of the abnormal fold of skin nearly covers the sclera gives rise to an appearance as if convergent squint was present.

Fig. 4.



Epicanthus. (After Von Ammon.)

Etiology.—Von Graefe[1] believed that epicanthus depended not so much upon the development of an abnormal fold of skin as upon insufficiency of some of the twigs of the oculo-motor nerve, and Hirschberg[2] has demonstrated a connection in one case between epicanthus and ophthalmoplegia, in which the defect appeared to be due to a congenital aplasia of the gray nuclei below the aqueduct of Sylvius, while Manz[3] has sought for a common origin of this and similar anomalies in the development of the bones of the face which are concerned in this region. The abnormality may appear in several members of the same family. Under the name *epicanthus externus* a somewhat analogous affection has been described in which the fold of skin was observed to cross the outer angle of the eye.

Treatment.—The usual method for the relief of this deformity is to excise a piece of skin from the bridge of the nose, with or without a canthoplasty, according to the circumstances. It is important to obtain firm primary union, lest, as has been pointed out by Knapp, the subsequent stretching result in unsightly scars. Arlt has obtained good results from the excision of the vertical fold of skin itself. I have seen an interesting case of partial epicanthus associated with a mole growing over the bridge of the nose, in which Dr. W. F. Norris effected a good result by excising the mole and covering in the skin-deficiency with a flap taken from the forehead.

Coloboma of the Eyelids appears in the form of a fissure which may be confined to the upper lids, either one or both, but which also has been noted in the lower lids and even in both upper and lower lids.

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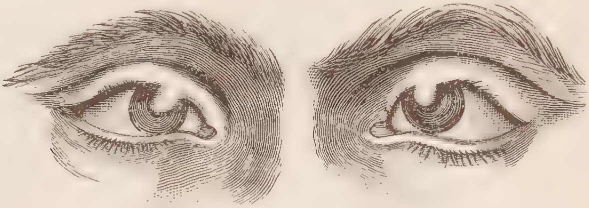
[1] Quoted by Manz, Graefe u. Saemisch, Handbuch der gesammten Augenheilkunde, vol. ii.

[2] Neurolog. Centralbl., 1885, No. 13.

[3] Graefe u. Saemisch, Handbuch der gesammten Augenheilkunde, vol. ii.

In the observations of Dor[1] of Lyons, and Jules Nicolin,[2] in twenty-seven instances a single eyelid was involved, twice the two eyelids of the same eye, sixteen times one lid of each eye, and once the deformity appeared on all four lids. In the majority of instances the defect is found in the upper lids,—according to D'Oench,[3] twenty-three times in thirty-three cases. Coloboma of the eyelids may exist as a single malformation, but more frequently has been seen in conjunction with hare-lip (fourteen times in forty-seven cases), absence of the lachrymal puncta, dermoid tumors of the cornea, and clefts of the iris, pharynx, lip, and nose.

Fig. 5.



Coloboma palpebrarum. (After Manz.)

Etiology.—Many theories have been advanced to explain this anomaly: a primordial defect of organization; the action of amniotic strands (Van Duyse); heterotopic tissue-formation (Manz); intra-uterine inflammation (Osio); an arrest of development, owing to the failure of the joining of the two halves of the first branchial arch together with the frontal prominence (Nicolin and Dor).

Treatment.—This consists in freshening the edges of the gap and uniting the opposing surfaces by sutures. The extent of each deformity and its relation to the cornea necessarily determine the character of operation which must be undertaken.

Absence of the Lids.—Sometimes children are born with complete or partial absence of the eyelids,—*ablepharia totalis* and *partialis,* or congenital lagophthalmos. At other times the eye is hidden, owing to an adhesion between the eyelids, and we have the condition to which Manz (*loc. cit.*) has given the name *cryptophthalmos,* a term which, as Van Duyse[4] has observed, should be preserved for those cases where the exterior integument passes in front of an eye more or less developed,—that is, where there is complete absence of the lids and palpebral fissure. It is a congenital anomaly of extreme rarity. Fuchs[5] has reported two cases of abnormal shortness of the lids so that the patients could close them only with the strongest pressure, and Pflüger has observed an instance of abnormal length of the fissure of the lids where complete closure was possible only by the greatest effort. Ectropion was not present. Tarsorrhaphy produced a cure in the first case.

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[1] Revue Générale d'Ophtalmologie, December, 1888.

[2] Du Colobome congénital des Paupières, Lyons, 1888.

[3] Archives of Ophthalmology, vol. xv.

[4] Annales d'Oculistique, January–February, 1889.

[5] Archiv f. Augenheilkunde, xv. 2.

Symblepharon, ankyloblepharon, and distichiasis also occur as congenital anomalies, and have been described. A rare defect is complete absence of the eyelashes.

DISEASES OF THE EYEBROW.

The eyebrow may be involved in any diseased process which attacks the neighboring skin or the scalp; and no separate description of injuries or diseases of the skin, especially seborrhœa and eczema, which are prone to attack this region, need be appended.

Two forms of cystic growth affecting this area are seen in children. Sebaceous cysts (atheromatous cysts) frequently appear as congenital growths upon the outer portion of the eyebrow, and may reach a considerable size. They are deeply situated, and not infrequently attached to the periosteum of the orbital margin. A cyst of this character takes its origin in the sebaceous follicles of the region. Dermoid cysts exist as painless, spheroidal growths, most frequently at the outer angle of the orbit on a level with the eyebrow, less usually at the inner angle above. When in the latter situation, it is possible to mistake the affection for a meningocele which may have a similar situation. As Juler[1] has pointed out, the meningocele can be emptied on pressure, has a slight impulse, and is not movable,—diagnostic points which do not obtain in the case of the dermoid cysts. The structure of a cyst of this kind is composed of the elements of the skin.

The treatment is the same for both classes of cysts, and consists in extirpation through an incision made parallel to the border of the orbit, care being taken not to rupture the sac-wall. It ought to be remembered in the excision of these growths that sometimes they are attached firmly to the periosteum, and that they may even erode the bone and extend far into the orbit.

AFFECTIONS OF THE LACHRYMAL APPARATUS.

Statistics show that affections of the lachrymal apparatus are less common among children than among adults. In Horner's list diseases of this system among children are set down as constituting one and sixteen-hundredths per cent. Among seventeen hundred and eight recorded cases at the Children's Hospital in Philadelphia one and one-tenth per cent. exhibited affections of the lachrymal apparatus. Diseases of the lachrymal structures naturally divide themselves into those which have their seat in the lachrymal gland, and those which affect the drainage-system,—*i.e.*, the puncta canaliculi, lachrymal sac, and nasal duct.

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[1] Ophthalmic Science and Practice, 1884.

DACRYOADENITIS, or an inflammation of the lachrymal gland, is comparatively a rare affection, and may be either acute or chronic. Hirschberg,[1] among twenty-two thousand five hundred cases, found only one instance of suppurative dacryoadenitis. He reports a case of acute, non-suppurative dacryoadenitis in a girl of fifteen, which on account of its analogy to bilateral parotitis he called "mumps of the lachrymal gland."[2] The chronic—especially, according to Knapp,[3] the monolateral—form is more common, and has been observed among scrofulous children, and may be caused by an injury or follow diseases of the conjunctiva and cornea. If the gland is chronically enlarged, palpation will reveal its lobulated border; if the inflammation is acute, there are pain, tenderness, and swelling at the upper and outer part of the eyelid, with chemosis of the conjunctiva beneath. This may go on to suppuration, and the abscess usually points upon the skin, but occasionally through the conjunctiva.

Treatment.—Warm applications and poultices to relieve the pain, and at the first appearance of pus early incision, either through the integument parallel to the eyebrow or through the conjunctiva, must be practised. If induration of the gland occur, this is to be treated locally with iodine or inunction of iodide-of-cadmium ointment.

FISTULA OF THE LACHRYMAL GLAND may remain on account of the rupture of an abscess, but has also been recorded as a congenital defect,—for instance, in Steinheim's[4] case, mentioned by Horner (*loc. cit.*), which was situated at the outer third of the upper lid, one-third of an inch from the ciliary margin, and surrounded by a tuft of hair. This may be closed by repeated cauterizations, by a plastic operation, or, in the event of failure, by extirpation of the gland.

DACRYOPS is an unusual affection caused by a cystic distention of one of the gland-ducts, and may be recognized by the presence of a bluish, translucent swelling beneath the conjunctiva at its upper and outer part.

HYPERTROPHY OF THE LACHRYMAL GLAND has been observed at birth, but usually is seen in later years, and consists in an indurated, lobulated tumor.

SPONTANEOUS PROLAPSE OF THE LACHRYMAL GLAND in the form of a soft movable tumor under the upper lid has been described. In a case recorded by Noyes[5] this condition was found in a girl of twenty, who for nine years had a swelling beneath the upper lid of this character, which on removal proved to be the lachrymal gland itself. The treatment is extirpation of the prolapsed organ.

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[1] Archives of Ophthalmology, 1879, viii. 370.

[2] See, also, Centralblatt f. prakt. Augenheilkunde, 1890.

[3] Transactions of the American Ophthalmological Society, 1884, vol. iii.

[4] Klin. Monatsbl., xiii. 302; also Ophthalmological Hospital Reports, vol. viii.

[5] Transactions of the American Ophthalmological Society, 1887.

Syphilis of the Lachrymal Gland.—The lachrymal gland is singularly free from syphilitic affections, but specific inflammation has been described by Streatfeild[1] and others in adults, the inflammation subsiding under antisyphilitic remedies. Albini[2] observed in a syphilitic young woman a tumor of the lachrymal gland which was composed of glandular elements, organized connective tissue, giant cells, but no Lustgarten bacilli.

Tumors of the Lachrymal Gland.—The several varieties of benign and malignant growths which have their seat in glandular tissue have been seen in the lachrymal gland. Samelsohn[3] found in a child three and a half years old a lithiasis of the gland, the concretion proving to be an osteochondroma. Snell[4] has seen an adenoma in a girl of eight, and I have recorded a similar instance in a young man, a patient of Dr. D. Hayes Agnew, who removed the growth.[5] Power[6] had a sixteen-year-old patient with encapsulated fibro-sarcoma. Tubercle has been discovered in this region, as, for instance, in a case reported by Abadie,[7] where the gland of a girl aged sixteen was found to be tuberculous. Excision of the growth in all instances is the only treatment.

Anomalies of the Puncta Lachrymalia and Canaliculi.—As congenital anomalies double puncta lachrymalia and canaliculi have been observed, as in the cases recorded by Mooren, Galezowski, Horner, and others, while Emmert and Fieuzal have seen congenital absence of these structures, and Von Reuss,[8] in a boy aged twelve, noted the absence of all four lachrymal points, while the papillæ were present and the canals were represented in the lower lid by furrows; in the upper lids they were wanting.

The slightest change in the natural relation of the lower punctum to the eye, against which it is directed backward, causes epiphora, or an overflow of tears.[9] The most fruitful sources of such abnormal relationship are the various chronic inflammations of the lid and conjunctiva,—blepharitis, granular conjunctivitis, and ectropion; and facial palsy and wounds of this region. In facial paralysis watering of the eye is sometimes the first symptom noted, and is caused partly by the loss of compressing power in the lid, especially in the fibres of Horner's muscle, and partly by the falling away of the punctum. Epiphora further results from the presence of a stye or tumor of the lid near the punctum, or, if the canaliculus is closed, by the presence of a foreign body like a hair, usually a cilium, a mass of fungus (leptothrix),[1] a so-called tear-stone, as in a case of Kipp,[2] or even, as Paul[3] has recorded, by the development of a polyp. Finally, an overflow of tears may follow an abnormal position of the caruncle, as in the observation of Horner (*loc. cit.*), where in a five-year-old child this was so misplaced as to be situated below the lower punctum upon the inner surface of the lid, which was pressed away from the eyeball. Enlargement of the caruncle, as Von Graefe observed, may produce a like symptom, and its removal has been followed by the disappearance of the difficulty.

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[1] British Medical Journal, 1882, ii. 633.

[2] Ann. di Ottalm., vol. xvi., 5–6, p. 501.

[3] Centralblatt f. prakt. Augenheilkunde, December, 1880.

[4] Ophthalmic Review, 1889.

[5] Transactions of the Pathological Society of Philadelphia, xii. 238.

[6] Transactions of the Ophthalmological Society of the United Kingdom, 1882, vol. ii.

[7] Archives d'Ophtalmologie, 1880, p. 432.

[8] Wiener Med. Presse, 1886, No. 7.

[9] Epiphora, strictly speaking, is an excessive secretion of tears, while stillicidium lachrymarum is an overflow from obstruction; but, as Mr. Nettleship remarks, no useful purpose is served by keeping the two names.

Treatment.—In cases of epiphora without disease of the lachrymal sac or stricture of the nasal duct, a simple slitting of the canaliculus is usually sufficient. If a foreign body is present, this should be removed. This treatment does not apply to cases of facial palsy.

ANOMALIES OF THE LACHRYMAL SAC AND NASAL DUCT.—Among children about one-third of the cases of lachrymal affections belong to the acute form of diseases of the sac; Horner (*loc. cit.*) states that this occurs in from thirty-six to forty-eight per cent. Dehenne[4] has reported a case of congenital tumor of the lachrymal sac which appeared in the form of an abscess. Terson and Galezowski[5] have observed similar examples, the latter surgeon having successfully treated his cases by injections of water.

Dacryocystitis.—The universal symptom in affections of the lachrymal sac and duct is epiphora. The eye swims in tears, and these are excited to overflow by exposure to dust, cold, or wind. The caruncle and plica are swollen, the neighboring conjunctiva hyperæmic and injected,—the lachrymal conjunctivitis of Galezowski,—the skin macerated, and the margins of the lids, especially nasal-ward, show signs of blepharitis. Pressure upon the region of the lachrymal sac, which may be distended (mucocele, lachrymal tumor), expresses through the puncta the retained fluid, which is a clear or semi-transparent viscid mucus (dacryocystitis catarrhalis), or turbid from admixture with purulent material (dacryocystitis blennorrhoica). The lachrymal sac thus chronically distended is liable at any time to take on a suppurative inflammation, producing *acute dacryocystitis*, which may be preceded by fever and chill; the lids and region of the nose become tender to the touch and tense with a red and brawny swelling, resembling erysipelas, for which it not infrequently has been mistaken. When there is added to disease of the sac a phlegmonous inflammation of the cellular tissue (dacryocystitis phlegmonosa) which surrounds it, the pus burrows in front of the sac, forms pouches in the connective tissue, and in most instances the *lachrymal abscess* points below the tendo oculi. If unmolested, the abscess ruptures externally, with the formation of a fistulous opening into the sac, the mouth of which is surrounded by pouting granulations.

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[1] Consult Goldzieher, Centralbl. f. prakt. Augenheilkunde, 1884, p. 33, and A. v. Reuss, Wiener Med. Presse, 1884.

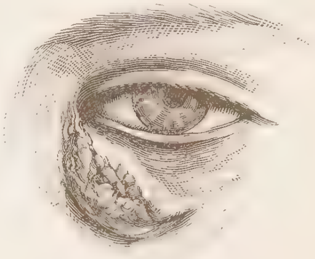
[2] New York Medical Record, xxiv. 289.

[3] Quoted by Schirmer, Graefe u. Saemisch, Handbuch, vol. vii.

[4] Recueil d'Ophtalmologie, 1883, p. 122.

[5] Ibid.; also Archives of Ophthalmology.

FIG. 6.



Phlegmonous dacryocystitis. (Children's Hospital.)

Obstruction of the nasal duct, which generally antedates the affection of the sac, may be situated at any part, but is usually found at its upper end. In the early stages of the catarrhal dacryocystitis there is probably no true stricture of the duct, but the flow from the sac into the nose is prevented by swelling of the mucous tissue; later, and in other instances, dense cicatricial strictures occur. The most impermeable obstructions follow injuries,—the rough use of bougies, and the like. It must not be forgotten that stoppage of the lachrymo-nasal duct may be caused by pressure from neighboring tumors,—*e.g.*, in the antrum of Highmore,—or by foreign bodies, as in the curious case recorded by Haffner,[1] where a lumbricoid worm three centimetres long and one millimetre thick occupied the left lower lachrymal canal.

Fistula of the Lachrymal Sac.—This occasionally has been observed as a congenital anomaly. It may be present only on one side, as in the case of Schreiber,[2] where in a child ten weeks old the orifice was directly under the internal palpebral ligament, or on both sides, as in the instance reported by Agnew.[3] Usually a fistulous opening into the sac is caused by the rupture of a lachrymal abscess, and Parinaud[4] has seen this result from a carious condition of the upper canine tooth. The opening may appear about one centimetre below the punctum, but also in various spots along a line which runs outward parallel to the lower orbital border. It usually communicates with the sac, but, as Rampoldi has reported, the opening may lead into the lower canal only, the sac above being shrunken. Pus or muco-pus, and later the tears, which should descend into the duct, exude from the opening, which for a long time persists as a fine orifice, at the mouth of which appears a drop of clear fluid. This is the so-called *capillary fistula*. The condition is to be differentiated from a buccal fistula below the margin of the orbit, which, according to Scheff,[5] may be done by observing that in the latter the situation is never accurately at the orbital margin, but from one-eighth to one-fifth of an inch below, that a sound never passes upward, but only downward, laterally or posteriorly, and that the secretion is always purulent.

Pre-lachrymal Abscess.—As has been especially pointed out by C. S.

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[1] Berlin. Klin. Wochenschr., No. 24, 1880.

[2] Jahresber. d. Augenheil.-Anstalt in Magdeburg, Nagel's Jahresbericht, 1885.

[3] Transactions of the Ophthalmological Society, 1874.

[4] Bull. et Mém. de la Soc. de Chir. Paris, ix. 180.

[5] Wien. Med. Wochenschr., No 12, 1888; Medical News, October 13, 1888.

Bull,[1] a swelling may exist above the internal palpebral ligament and a little external to the region of the lachrymal sac, associated with a fistulous opening from which pus flows, having no connection with the sac itself. In his case this pre-lachrymal abscess was caused by a blow from a cane at the inner angle of the eye, and was associated with caries and perforation of the lachrymal bone. The same condition I have observed in children without injury, the subjects of hereditary syphilis. The condition is to be distinguished from a true lachrymal abscess by the fact that there is no interference with the passage of the tears from the conjunctiva into the sac, by the pain on pressure, and by the absence of acute inflammation. The treatment is that of an abscess, together with such constitutional measures as may be indicated by the dyscrasia of which the patient is the subject.

Etiology of Diseases of the Lachrymal Sac and Duct.—Disease of the lachrymal sac rarely is primary. In young infants dacryocystitis, often double, arises without apparent cause. Kipp[2] found during two years three and six-tenths per cent. of lachrymal diseases, and six per cent. of these were under one year of age, the affection even having been seen shortly after birth. In the majority of cases blennorrhœa of the sac is caused by retention of the secretion from stricture or obstruction in the nasal duct and participation of the lining of the sac in an inflammation of the naso-pharynx. In other instances strictures result from, rather than cause, blennorrhœa. The proper appreciation of the pathological conditions of the nasal mucous membrane in relation to diseases of the lachrymal apparatus, and as an etiological factor, is of the utmost importance. This relationship has been especially dwelt upon by Harrison Allen,[3] Nieden,[4] Ziem,[5] Gruening,[6] and Grut.[7] Although it might seem natural that conjunctivitis, especially purulent conjunctivitis, should cause lachrymal disease, this is by no means frequently the case. Horner (*loc. cit.*) in one instance only was able to observe a blennorrhœa of the sac arise from a similar inflammation in the conjunctiva; and conjunctivitis and blepharitis, so constantly accompanying disorders, follow rather than cause the lachrymal affection. Obstruction of the duct and disease of the sac follow measles, scarlet fever, and especially variola, because these exanthemata are accompanied by inflammation of the nasal mucous membrane. Periostitis and caries of the lachrymal bone, the result of syphilis, are important causes. Gummy growths may block the sac (osteo-periostitis gummosa of Panas) and go on to rapid suppuration. The relation between asymmetry of the face and disease of the naso-lachrymal duct deserves mention. Finally, traumatism accounts for certain cases. Fistulas, especially those seen in infants, not infrequently depend upon disease of the bones, which in turn is the result of inherited syphilis.

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[1] American Journal of the Medical Sciences, 1880.

[2] Transactions of the American Ophthalmological Society, ii. 537.

[3] Medical News, February 6, 1886.

[4] Archiv f. Augenheilkunde, xvi. 381.

[5] Centralblatt f. prakt. Augenheilkunde, 1886, S. 222.

[6] Medical Record, 1886, xxix.

[7] Hospitalstid, Nos. 21 and 22, 1865,—abstract, Archives of Ophthalmology, vol. xv.

Prognosis in Lachrymal Disease.—The well-known fact that under the most skilful treatment affections of the tear-passages often resist healing may render a guarded prognosis necessary. This depends entirely upon the condition of the nasal chambers, the duration of the malady, the permeability of the stricture, and the cause of trouble. When the latter is the result of injury, the prognosis becomes especially grave, and the malady may be irremediable.

Treatment.—Manifestly, the success of all treatment centres upon the restoration of the calibre of the duct, if this be strictured, and the relief of the most important cause of the disease of the sac. Occasionally it suffices, especially in new-born children, to dilate the punctum and wash out the sac with a fine Anel syringe and an antiseptic solution. Usually three procedures are necessary,—slitting up the canaliculus, introducing the probe into the nasal duct, and washing out the sac and naso-lachrymal duct with an appropriate syringe.

The slitting of the caniculus is best done with a Weber's knife, which is introduced, the lid being drawn down and out with the thumb, until the probe point of the instrument touches the inner wall of the lachrymal sac. It is then raised to the vertical line with the cutting blade turned slightly inward, and the roof of the canaliculus thus divided. The lower canal is most frequently chosen. Some surgeons, as a rule, split the upper canaliculus; if there is much distention of the sac (mucocele), a good plan is to enter the upper passage and to incise both this and the wall of the sac. The probe (Bowman's probes are the best, though useful modifications have been devised by Theobald and Tansley) is now introduced along the canaliculus until its point touches the lachrymal bone. It is then raised to the vertical position and pushed into the duct, remembering that the direction is downward, slightly backward, and outward. Undue efforts should never be employed. If the stricture resist, recourse may be had to dividing this with a knife, either the one which has been employed in slitting the canaliculus, or, still better, the specially-devised instrument of Stilling. The duct and sac should now be washed out thoroughly with a lachrymal syringe and some antiseptic fluid, either a saturated solution of boracic acid, or a one to five-thousand solution of bichloride of mercury.

It is advisable to make the first trial with a No. 2 Bowman probe; if this fails, a smaller one may be tried. Either rapid or gradual dilatation is employed, the latter being the preferable method. The sound should be used at first every second or third day, but as the case progresses longer intervals may elapse. Theobald has recommended the introduction of very large lachrymal probes, a method not always applicable, owing to the great diversity in the size of the bony duct. The whole treatment often occupies months.

If a lachrymal abscess supervenes, and is seen early, the canaliculus should at once be slit, and, if possible, the secretion evacuated, with retention of the passage into the nose. Frequently the pain and swelling are such as to render this impossible, and the opening must be made upon the face about one centimetre below the palpebral tendon, cutting downward and outward. In the highly inflammatory stage, probing must not be employed, but the sac and abscess-cavity should be freely irrigated with a solution of bichloride of mercury. An excellent practice is to use hot compresses over the swelling, preferably of carbolized water of a temperature of 120° F., frequently changed, and applied for five or ten minutes at a time. Later, the restoration of the passage into the nose must be undertaken by probes in the manner already described, but if the patient is refractory, which is almost invariably the case in young children, excellent results will follow the introduction of a style made of lead wire, slightly hooked over the inner canthus to prevent its slipping into the sac.

Swelling over and around, together with fistulous connection into, the lachrymal sac, occasionally will subside under the judicious use of a compressing bandage. I have tried this method, again recently advocated by Bothen[1] and others, quite often in the Children's Hospital and in the Philadelphia Hospital, with very good results. My plan is to put a graduated compress of iodoformated cotton over the swelling, securing it firmly with a roller bandage.

In addition to the local measures already mentioned for the purpose of producing healing in case of lachrymal disease, weak solutions of nitrate of silver, salicylic acid, iodoform, and creolin, with which latter drug I have had but indifferent success, have been advocated. In case of acute inflammation with abscess-formation, quinine, and iron in the form of Basham's mixture, are indicated; in syphilis with disease of the bone and gummatous deposit, the usual remedies are to be exhibited, and the best of these in children is the mercury binder; in struma, cod-liver oil, hypophosphites, and iron in the form of the syrup of the iodide are the most trustworthy remedies. Scrupulous attention to the nose and naso-pharynx is necessary, and any local lesions which present themselves must be treated. In the absence of a special line of treatment for this region, I have achieved excellent results by simply spraying out the parts with Dobell's solution and listerine, while carrying on the regulation measures for the relief of the lachrymal disorder.

If a fistula remains, this may be healed at times, as already stated, by compression. In the event of failure, freshening the edges and the galvano-cautery may be tried, the surrounding pouting granulations being removed by scraping. The capillary fistulas are productive of no inconvenience, and may be allowed to remain undisturbed. In stubborn cases which have resisted all reasonable treatment, extirpation of the lachrymal gland has been done, as was originally recommended and performed by Lawrence, or, as more recently has been advocated by De Wecker, excision of its palpebral portion, or the lachrymal sac may be obliterated by means of caustics. Under judicious treatment, the necessity for these somewhat heroic measures ought not to arise.

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[1] Centralblatt f. prakt. Augenheilkunde, Supplement, 1887.

Character of the Lachrymal Secretion under Pathological Conditions.—The lachrymal sac is a reservoir for the fluid secreted by the conjunctiva, and, this fluid being more or less loaded with micro-organisms, if stopped by stricture of the duct the sac becomes stuffed with micrococci. Widmark[1] found in dacryocystitis streptococcus pyogenes, which by inoculation caused phlegmonous inflammation. If the cornea is abraded, or if a solution of continuity in this membrane is necessary by operation, the presence of pathogenic organisms in the fluid becomes a serious complication. These may turn a simple abrasion into a sloughing ulcer and an aggravated hypopyon keratitis; they may forbid the healing of an ordinary keratitis; and, finally, they may inoculate an operative wound and defeat the object of the operation. For this reason it is most important that in any of the three cases just quoted the permeability of the nasal duct should be ascertained; if it is strictured it should be opened, and the walls of the lachrymal sac, if inflamed, as speedily as possible brought into a healthy condition. The importance of this relation of the lachrymal apparatus to diseases of the cornea will be again referred to in the section devoted to the consideration of the latter affection.

DISEASES OF THE CONJUNCTIVA.

The great frequency of diseases of the conjunctiva and cornea during childhood—according to Horner, half of all the affections of the eye during this age of life belong to this group—sufficiently emphasizes the importance of the subject. Nearly sixty-three per cent. of the patients who have applied at the Eye Dispensary of the Children's Hospital during the last nine years have come on account of one or other of the types of corneal or conjunctival affection, in thirty-six and a half per cent. of the cases the lesion belonging to the conjunctiva alone.

The most important group of diseases of the conjunctiva includes the inflammations, to which the general term ophthalmia may be applied.

OPHTHALMIA NEONATORUM.

Synonymes.—Purulent ophthalmia, Gonorrhœal ophthalmia, Blennorrhœa of the conjunctiva, Purulent conjunctivitis.

Definition.—This is an inflammation of the conjunctiva characterized by great swelling of the lids, serous infiltration of the conjunctiva, and the free secretion of contagious pus.

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[1] Hygeia, 1887, abst. from Centralblatt f. prakt. Augenheilkunde, July, 1887.

Etiology.—The prevailing opinion is that this affection is caused by the introduction into the eye of the infecting material from some portion of the genito-urinary tract of the mother at the time of or shortly after birth. It is equally well ascertained that the majority of cases and all severe forms are due to the presence of the gonococcus of Neisser. Exceptionally inoculation appears to have taken place in utero. Magnus[1] reports an instance of ophthalmia neonatorum, with involvement of the cornea, of such origin, where the membranes were ruptured three days before birth and permitted the entrance of the gonococci. Fuchs[2] has observed in a child at birth perforation of the cornea as the result of congenital ophthalmia. A high degree of penetrating power is ascribed to this special micro-organism by Mules,[3] who has seen an infant born at the seventh month after an exceptionally easy labor with well-marked ophthalmia neonatorum. The child was brought eight hours after delivery, exhibiting the symptoms of the second stage of the disease: so that infection in utero must have occurred at least two days before birth or rupture of the membranes.

The gonococcus is generally, although, according to Widmark[4] and Weeks,[5] not invariably, present in the secretion, being specially numerous during the muco-purulent stage. According to Cohn, two varieties of ophthalmia neonatorum may be distinguished,—a severe type, supplied with the micro-organism, with a tendency to increase in severity and involve the cornea; and a milder type, non-specific, with a tendency to recover.

The presence of a virulent vaginal discharge in the mother is not necessary to produce this condition, as it probably may arise from the introduction of any muco-purulent discharge during the birth; while careless bathing of the child after birth and the use of soiled towels and sponges are fruitful sources of infection, and it is even possible that later contact with the lochial discharges may originate the disorder. Andrews,[6] Zweifel,[7] and others, however, have failed by inoculation of healthy lochia to produce the disease.

Opinions differ in regard to the exact time of the inoculation, which probably is more likely to occur in retarded labors and with face-presentations. Mules (*loc. cit.*) thinks the pus may be introduced into the eyes by the edge of the perineum, the anterior edge of which becomes an elastic curved cord, which, after slipping over the forehead, presses for a shorter or longer time on the eyelids, depositing thus vaginal secretion within them.

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[1] Klin. Monatsbl. f. Augenheilkunde, July, 1887.

[2] Die Ursachen und die Verhütung der Blindheit, p. 113.

[3] Medical Chronicle, 1888.

[4] Hygeia, 1884, p. 404.

[5] Medical Record, July 24, 1886.

[6] New York Medical Journal, October 25, 1885.

[7] Archiv f. Gynäkol., xxii. 329.

Boys are more frequently attacked than girls. Emmert,[1] of Bern, has demonstrated a relation between the temperature and this disease. In cold climates ophthalmia neonatorum is especially frequent in the summer months; in hot countries, in the spring and autumn.

Pathology and Pathological Anatomy.—Horner examined an eye which for forty-eight hours had been the subject of ophthalmia neonatorum, and found that the œdematous swelling limited a general laminated structure of the tarsal conjunctiva. The epithelium of the bulb was tolerably preserved; the superficial layer of that covering the tarsus was curled, irregular, and wanting; the swollen vessels were exposed and hemorrhages present; the papillæ were swollen, and there was much lymphoid infiltration.

The gonococci are seen in the nuclei and at the margin of the epithelial cells and on the surfaces of and within the pus-cells; later they penetrate the epithelium and enter the lymph-spaces. The infectious secretion introduced into an eye will produce purulent ophthalmia, so the pus from it can be in turn inoculated into the urethra with the production of a purulent inflammation.

Symptoms.—Ophthalmia neonatorum usually begins on the third day after birth, but may set in as early as from twelve to forty-eight hours after inoculation, or be delayed, when it is the result of a secondary infection from soiled fingers or sponges or cloths, to a much later date. Almost always both eyes suffer, the one being earlier and frequently more decidedly affected than its fellow. Four stages of the disease are common, but, as these vary in different cases and more or less rapidly shade the one into the other, no very sharp lines need be drawn. A slight redness of the conjunctiva, with a trifling discharge in the corner of the eye, is rapidly succeeded by great cushion-like swelling of the lids, with intense chemosis and congestion of the conjunctiva, accompanied by severe pain and discharge; the surface of the swollen lid is hot, dusky red, and tense; the upper lid overhangs the lower, and at first can only with difficulty be everted. The discharge, which in the beginning is slightly turbid, soon changes to a yellow or greenish-yellow pus, and is secreted in great quantities. If the lids are everted during the first day or two of the disease, the conjunctiva will be found to be swollen, red, and velvety, and that upon the eyeball intensely injected; upon the surface easily-detached flakes of lymph are found; later the conjunctiva becomes rougher, of a dark-red color, spots of ecchymosis appear, or it is succulent and easily bleeds. Marked chemosis and infiltration of the ocular conjunctiva succeed, forming a hard rim; at the bottom of the crater-like pit thus produced, the cornea may be seen; the thick cream-like discharge increases, and either flows out from beneath the overhanging upper lid on to the cheek, or is packed up in the conjunctival cul-de-sac.

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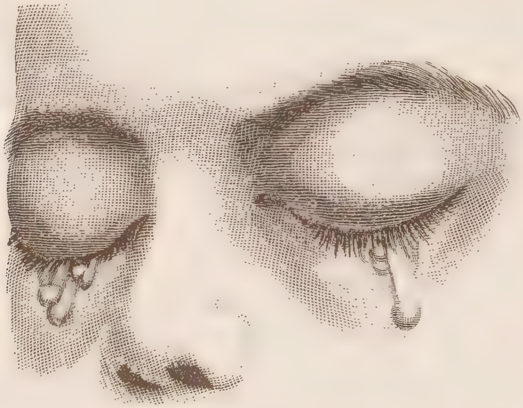
[1] Annales d'Oculistique, 1881, p. 63.

The lids now may lose much of their tense character, and can be more easily everted; the conjunctiva is puckered into folds and papilla-like elevations, and the discharge contains an admixture of blood and serum.

Fig. 7.



Ophthalmia neonatorum. (Philadelphia Hospital.)

Gradually the disease declines, and in from six to eight weeks the discharge ceases. The relaxed palpebral conjunctiva is thick and granular, looking like the granulation-tissue which surrounds wounds. The ocular conjunctiva is also thickened, and positive cicatricial changes may remain.

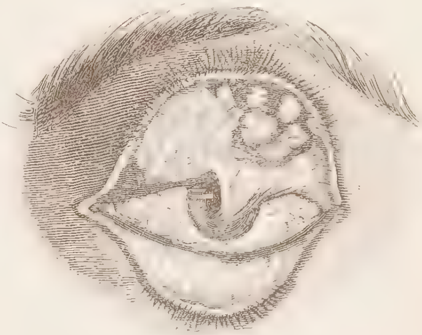
The chief danger is destruction of the vitality of the cornea, the danger of which is materially increased if this membrane becomes lustreless, dull, and hazy within the first day or two of the disease. Frequently small, oval ulcers form near the limbus, either transparent or surrounded by an area of cloudy infiltration. In many mild cases the cornea escapes without harm. The changes which take place in the cornea are due in part to strangulation of the vessels by the swollen tissue, but largely to direct infection of its substance by the presence of the discharge.

In the formation of a corneal ulcer, either its healing with regeneration of the corneal tissue takes place, or else perforation occurs. The result of perforation will depend upon the amount and character of the destruction of the corneal tissue. When the ulcer is central and perforates, the aqueous humor escapes, the lens is pressed forward against the posterior surface of the cornea, and the opening becomes closed with lymph. This renders the re-collection of the aqueous possible, or, when it occurs, returns the lens to its proper position, leaving upon its anterior capsule a little mass of lymph, and the formation of a *pyramidal cataract* results.

Perforation of an ulcer peripherally situated, especially below, is followed by adhesion of the iris to the opening. The aqueous escapes, and, as the iris and the lens fall forward, the former becomes entangled in the perforation and is fixed by inflammatory exudation. The adhesion is either on the posterior surface or in the cicatrix, and the resulting dense white scar receives the name *adherent leucoma.* If the region of the scar is bulged forward because it is unable to resist the intraocular tension and pressure, *anterior staphyloma* results. The effect of extensive necrosis and sloughing of the corneal tissue with total prolapse of the iris, matting together of the parts by exudation and protrusion of the cicatrix, is the formation of a *total anterior staphyloma.* In rare instances an adhesion between an ulcerated spot upon the cornea and the surface of the tarsal conjunctiva takes place, resulting in the production of a symblepharon, even in the absence of any diphtheritic processes in the ulceration. Hutchinson[1] has seen in a child of five years double ptosis which had followed an attack of purulent ophthalmia in infancy.

Finally, perforation may be followed by inflammatory involvement of the ciliary body and choroid, and the rapid destruction of the eye through panophthalmitis, or a slower shrinking of the tissue with atrophy of the bulb. Dense opacity occasionally appears in the cornea during convalescence, and may go on to softening and ulceration, or clear up perfectly. It may arise with great suddenness, and, when it occurs in the lower half of the cornea, a deep indentation, owing to the pressure of the margin of the lid, is likely to occur.

Fig. 8.



Symblepharon of upper lid following purulent ophthalmia. (Philadelphia Hospital.)

Ophthalmia neonatorum does not always follow the course just described. In many instances the inflammation is mild, and the secretion and general appearance of the eye are not far different from those of an ordinary case of catarrhal or muco-purulent ophthalmia. In these instances the cornea escapes injury.

The appearance of the conjunctiva materially differs in different cases. Its surface may be covered over, not merely with easily-detached flakes of lymph, but with a positive, gray, false membrane, and even, more rarely, with a deep infiltration like that seen in diphtheritic conjunctivitis. Constitutional disturbance is not lacking, with restlessness, fever, and distinct depression. Lucas[2] has seen an eighteen-months-old baby suffering from ophthalmia neonatorum have at the same time synovitis of the knee and wrists of the same character as such complications during gonorrhœa. Analogous cases have been reported by Saswornitzky,[3] Debierre,[4] and Darier.[5]

Diagnosis.—The onset and character of the disease, its symptoms and course, render any mistake in regard to its nature practically impossible. The only word of caution necessary is to avoid any indifference in regard to what at first may appear to be only a trivial inflammation in the eyes of a new-born child, remembering that with great rapidity a virulent and destructive inflammation may follow.

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[1] Ophthalmological Hospital Reports, vii. 43.

[2] British Medical Journal, 1885, ii. 57, 699.

[3] Abstract in Archives of Ophthalmology, vol. xv.

[4] Revue Générale d'Ophtalmologie, 1885, iv. 299.

[5] Archives d'Ophtalmologie, Mars–Avril, 1889.

Prognosis.—This is always grave, the gravity increasing in direct proportion to the violence of the inflammation and the condition of the cornea. Under the newer methods of treatment, more eyes are saved than was formerly the case, and still more would escape were it possible to impress upon the attendants of children thus afflicted the necessity of seeking capable medical advice at the very moment of the appearance of any trouble. If, as only too frequently is the case, treatment has been neglected until extensive sloughing of the cornea has occurred, no form of medication can do more than relieve the violence of the inflammation, which, when it subsides, leaves the child with sight hopelessly marred, perhaps destroyed.

Prophylaxis.—One of the most fruitful causes of blindness is this form of inflammation of the eyes of new-born infants. Thirty per cent. of the inmates of institutions in the United Kingdom have lost their sight from this cause. According to Prof. Magnus, of Breslau, 71.99 per cent. of all who become blind during the first year of life are rendered so by purulent ophthalmia; even of those who become blind before the twentieth year of life it constitutes 23.5 per cent.; in other words, of every ten thousand children under five years of age, 4.28 per cent. lose their vision by ophthalmia neonatorum. In the blind asylums of Switzerland the proportion is 26 per cent.; in those of Austria, Hungary, and Italy, about 20 per cent.; while in Spain and Belgium it falls to about 11 or 12 per cent.[1] In the face of these facts, and with the knowledge, as Howe's statistics show, that, owing to the carelessness of the emigrant population of this country, blindness is on the increase, the prophylaxis and treatment of this affection are of the highest importance. Credé's method of treating the eyes of the new-born child is the one which is followed by the best results. This consists in dropping into the conjunctival sac one drop of a two-per-cent. solution of nitrate of silver, the lids having been wiped dry. Occasionally decided reaction has followed this application, requiring the use of cold to allay the irritation, and Pomeroy[2] has related a case in which severe hemorrhage followed the use of nitrate of silver in an eye already inflamed. This method reduced the percentage of the disorder in Credé's service from 7.8 to 0.31. Other methods, like that first employed by Bischoff, in Basel, of washing out the vagina before birth with injections of carbolic acid, and the eyes of the newly-born with salicylic acid, or the later Kaltenbach method of washing the vagina with a 0.4-per-cent. bichloride solution after each examination, and cleansing the eyes with distilled water, have not compared favorably with Credé's procedure. L. Korn[1] has concluded, after a careful examination of this matter, that Credé's plan is absolutely safe and certain, although painstaking cleanliness during the birth and also in childbed may reduce the possibility of this disease to a minimum. Mules (*loc. cit.*) quotes from Fuchs the following statistics, which are interesting in this connection.[2]

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[1] See Lancet, July 20, 1889.

[2] New York Medical Record, August 20, 1887.

	Number of Children.	Ophthalmia Neonatorum.
No treatment	1092	19.26
One-per-cent. carbolic acid	1541	7.42
Credé's method	1250	5.44

Of not less importance is the necessity of searching for sources of infection in the hands of the mother and the child. Not only should all antiseptic precautions be taken during the labor and immediately afterwards, but, if infection is known to exist, the child should be removed from the immediate surroundings of the lying-in woman and the possibility of contamination by utensils and towels.

Treatment.—Naturally, this deals with three conditions,—the inflammatory swelling of the lids, the state of the conjunctiva, and the corneal complications.

1. During the earlier stages, when the lids are tense and the secretion lacking in its later creamy character, in addition to absolute cleanliness, local application of cold is the most useful agent. Kries[3] has shown that the coccus develops only slowly at a temperature of 90° to 92° F., and Weeks (*loc. cit.*) has demonstrated that the temperature of the conjunctiva may be reduced to 88° or 94° according to the amount of swelling of the lids. The cold should be applied in the following manner: upon a block of ice square compresses of patent lint are laid, which, in turn, are placed upon the swollen lids and as frequently changed as may be needful to keep up a uniform cold impression. This is far preferable to the use of small bladders containing crushed ice; indeed, the use of ice for infants is not advisable. The length of time occupied with these cold applications must vary according to the severity of the case. Sometimes they may be almost continuously used, and sometimes frequently for periods of half an hour at a time. On the other hand, hot fomentations are occasionally better than cold, especially when the corneal complications exist. These are employed in like manner with squares of antiseptic gauze wrung out in carbolized water of a temperature of 120° F. and frequently changed.

2. Constant removal of the discharge must be assiduously practised. The lids are to be gently separated, the tenacious secretion wiped away

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[1] Archiv f. Gynäkol., 1888, xxxi. 2, S. 240.

[2] For further statistics in regard to the comparative value of various forms of preventive treatment, see Peuch, Archives de Tocologie, des Maladies des Femmes et des Enfants nouveau-nes, Février, 1890.

[3] Wien. Med. Wochenschr., 1885, Nos. 30, 31, 32.

with bits of moistened lint or absorbent cotton, and the conjunctival sac freely irrigated with an antiseptic solution. For this purpose a saturated solution of boracic acid, or one of corrosive sublimate, a grain to the pint, inasmuch as a solution of one to ten-thousand will materially retard the vitality of the coccus, may be employed. Special and ingenious forms of lid-irrigators have been devised by Story and others for this purpose. The cleansing process must be repeated at least every hour, day and night, but, if necessary, should be much more frequently used. Many solutions other than those mentioned have found favor with surgeons; for instance, alum (gr. viii–f℥i), sulphate of zinc (gr. ii–f ʒi), carbolic acid in one-half- to five-per-cent. solution, weak solutions of nitrate of silver, solutions of quinine, as recommended by Tweedy, alcohol and bichloride-of-mercury solution, advocated by P. H. Mules,[1] iodoform ointment of four-per-cent. strength, creolin in one-per-cent. solution, and cocaine, either dropped frequently upon the conjunctiva or introduced in the form of a salve.

3. The local application of nitrate of silver to the conjunctiva must not be employed in the earlier stages before free discharge is established, nor in those cases, no matter what the stage, where the lids are tense and board-like and the surface of the conjunctiva is covered with a gray film or a positive false membrane. When the secretion is free and creamy, when the lids are relaxed, when the conjunctiva is dark-red and puckered into papilla-like excrescences, the time for its application has come. Once a day the conjunctiva should be brushed over with a solution, ten or twenty grains to the ounce, its surface first having been carefully freed from any adherent discharge, and all excess washed away with water. In severe cases the mitigated stick and even the solid pencil of nitrate of silver may be employed, great care being taken to neutralize the excess with a solution of common salt. All strong applications must be made by the hand of the surgeon himself. Ulceration of the cornea does not alter the treatment described, except that all pressure upon the globe while manipulating the eye is to be avoided. So long as the discharge is abundant the use of the caustic is indicated.

At the first appearance of corneal haze a solution of atropine is to be dropped two or three times daily into the eye. If, however, a marginal ulcer forms and danger of perforation is imminent, or even if this has occurred, good results will follow the use of a solution of sulphate of eserine. When the vitality of the cornea is threatened or the surface of the conjunctiva is covered with a gray film, better results follow the use of hot applications instead of cold, and I have more than once seen cases apparently very hopeless go on to recovery under the use of scrupulous antiseptic cleansing and the almost continuous application of hot compresses. Persistent swelling of the conjunctiva is sometimes treated by scarification. Division of the outer commissure to relieve pressure, leeching, and indeed any form of treatment followed by decided loss of blood, are hardly applicable to young infants, although they may be indicated in adults.

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[1] British Medical Journal, February 4, 1888.

If one eye alone is affected, suitable protection for the sound eye should be provided. This may be accomplished by antiseptic bandaging of the uninflamed organ (Buller's shield is difficult of application in infants). Fraenkel[1] has suggested the daily use in the unaffected eye of a drop of a two-per-cent. solution of lunar caustic.

On the whole, that treatment which has in view reduction of the inflammation with cold applications, for which under the conditions named hot affusions are substituted, absolute cleanliness, frequent irrigation with antiseptic solutions, and at the proper stage nitrate of silver, will meet with the best success. The attendants must be impressed with the fact that upon their faithful carrying out of directions and upon their unremitting care much, if not all, of the hope of bringing the case to a successful termination depends. The attendants must further be impressed with the contagious nature of the pus: all bits of rag and pledgets of lint used in the treatment must be destroyed, and after each treatment the hands of those engaged must be thoroughly washed and then disinfected with a solution of bichloride of mercury.

MUCO-PURULENT OPHTHALMIA.

Synonymes.—Catarrhal ophthalmia or conjunctivitis.

Definition.—This is an inflammatory disease of the conjunctiva characterized by congestion, dread of light, spasm of the lids, and free mucopurulent discharge.

Etiology.—The disorder is commonest in warm and changeable weather; it is markedly contagious, and will pass rapidly from one member to another of a household, varying much in severity with each. In the severe forms micro-organisms are found, which may be the cause of the contagion. Very troublesome ophthalmia follows or accompanies the exanthemata (*exanthematous ophthalmia*), especially measles and scarlet fever; scrofulous and anæmic children are most liable; neglected hyperæmias and the presence of follicular granulations increase the susceptibility to infection.

Symptoms.—There is at first redness of the edges of the lids, with increased vascularity of the conjunctiva and gritty sensation in the eye, some pain, and a free discharge, which glues together the edges of the lids, which are slightly swollen. Usually the cornea does not suffer, but in young children, especially those who have had measles, superficial ulcers form; the photophobia then becomes intense. The disease varies in type from a mere hyperæmia to a severity of such degree that it is not readily distinguished from *purulent ophthalmia,* into which type it may pass by neglect.

There is a large group of cases of *acute conjunctivitis* (simple or catarrhal conjunctivitis) which does not conform to the above description; those, for example, where there is more or less redness, little or only slight discharge, and where local irritants like wind and dust or the strain occasioned by neglected ametropia are evidently the causes of its existence,—cases, in short, which correspond to *hyperæmia* of the conjunctiva.

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[1] Klin. Monatsbl. f. Augenheilkunde, February, 1889.

In other varieties exposure and even rheumatism seem to be the causes: and among other factors catarrh of the nose and of the bronchia and eczema deserve mention.

In that form of conjunctivitis which is seen especially in the spring and fall, and to which the meaningless name "*pink eye*" has been applied, Weeks[1] has described and isolated a special bacillus which he considers the cause of the trouble; and, according to E. Schmidt,[2] *epidemic conjunctival catarrh* is due to a coccus identical with the staphylococcus pyogenes albus.

Children frequently suffer with marked muco-purulent ophthalmia coincidently with the appearance upon the face of the vesico-pustules of impetigo contagiosa. Muco-purulent ophthalmia of any type becomes a grave disorder if it breaks out in schools, homes, or any institution where numbers of children are gathered together. It is a markedly infectious disease, and is almost certain to run through the establishment; the importance of the trouble is rendered all the greater if granular lids are present.[3]

Diagnosis.—The diagnosis presents no difficulty. Inspection will reveal the characteristic congested, opaque, and velvety appearance of the conjunctiva, and the presence or absence of epithelial ulcers or phlyctenulæ, while the mobility of the iris and the preservation of its normal color and the character of the coarse bulbar injection (not fine and pericorneal) exclude iritis.

Prognosis.—This is good, but the cases following measles are sometimes very intractable. So, also, when the ophthalmia has existed for a long time, and if neglected, the papillæ of the palpebral conjunctiva become hypertrophied, and loops of vessels upon the ocular conjunctiva lie so closely together that an almost uniform red surface is the result, forming one type of chronic ophthalmia.

Knapp[4] saw pterygium superius as a sequel of an attack of purulent ophthalmia in a girl aged eleven years, an inmate of a home in which the disorder was epidemic.

Treatment.—The eye should be frequently and thoroughly cleansed with a weak solution of bichloride of mercury (one grain to the pint) or a solution of boracic acid (fifteen grains to the ounce). The lids may be everted and the surfaces brushed over with nitrate of silver (five grains to the ounce, or stronger if the discharge is copious). In the later stages, when the discharge is scanty, the nitrate of silver may be discontinued, and some other astringent collyrium substituted; zinc and alum answer very well. Dusting in calomel or iodoform or subnitrate of bismuth acts very well occasionally in stubborn cases. Photophobia calls for dark glasses or a large shade, but *the eyes must not be bandaged.* The pernicious habit of using poultices, tea-leaves, scraped potatoes, and the like cannot be condemned too strongly. Atropine drops, provided they cause no irritation, are useful if phlyctenulæ complicate the trouble.

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[1] Medical Record, May 21, 1887.

[2] Inaugural Dissertation, St. Petersburg, quoted in Medical Chronicle, June, 1888.

[3] In the event of an epidemic of purulent or muco-purulent ophthalmia in a school, workhouse, or similar institution, scrupulous attention to isolation of the affected inmates, strict cleanliness, and especially the use of separate utensils, towels, etc., are necessary. Touching the proper regulations under such circumstances, the reader is referred to a paper on "The Ophthalmic Isolation School at Hanwell," by Sydney Stephenson, Lancet, April 5, 1890.

[4] Archives of Ophthalmology and Otology, ii. 54.

Good diet, fresh air and exercise, iron, if there is anæmia, quinine, especially if the patients come from damp and possibly malarious quarters, and cod-liver oil, if scrofula is present, are all indicated.

FOLLICULAR OPHTHALMIA.

Synonymes.—Follicular conjunctivitis (Saemisch), Conjunctivitis follicularis simplex (Raehlmann), Trachoma folliculare (Mandelstamm).

Definition.—This is an affection of the conjunctiva characterized by the presence of small pinkish prominences in the conjunctiva, for the most part in the retro-tarsal folds, and usually arranged in parallel rows.

Etiology.—The disease arises under the influence of bad hygienic surroundings, and is especially engendered in pauper schools, asylums, and prisons, under which circumstances it may appear as an aggravated epidemic; but it is frequently seen in mild form among children generally. Much difference of opinion exists as to whether follicular conjunctivitis should be placed in a separate category from granular disease, as has been done by Saemisch,[1] Swanzy,[2] Meyer,[3] and other authors, or should be looked upon as an early stage of granular ophthalmia, as is taught by Nettleship[4] and other writers. Reich[5] looks upon this affection as a mild form and an early stage of trachoma, into the severe types of which it frequently passes, and Stilling[6] in endemic follicular ophthalmia has observed the disorder in school-children pass into the condition of granular lids. Bacteriologically, Kucharsky[7] considers trachoma and follicular disease identical. Admitting the not infrequent apparent transitional forms, the evidence, clinically at least, that this is a very distinct disease, widely different from granular ophthalmia, warrants a separate description.

Symptoms.—The children (for it mostly occurs in children and young people) complain of slight dread of light and inability to continue at close work, and inspection reveals numerous round elevations in the conjunctiva, chiefly along the fornix, which are tumefied lymphatic follicles. Their color varies from nearly white to a decided pink. They are usually unassociated with decided symptoms of catarrhal conjunctivitis, and the bulbar conjunctiva is not greatly reddened, although they sometimes give rise to ulceration of the margin of the cornea, decided hyperæmia, and swelling of the conjunctiva. Uncorrected ametropia aggravates the disorder. After the disappearance of the enlarged follicles the conjunctiva remains in its natural state.

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[1] Graefe u. Saemisch, Handbuch der gesammten Augenheilkunde.

[2] Hand-Book of Diseases of the Eye, 2d edition.

[3] Diseases of the Eye, translated by F. Fergus, M.B.

[4] Ibid., 2d edition.

[5] Wratsch, No. 7, Abst. in Archives of Ophthalmology, December, 1888.

[6] Berlin. Klin. Wochenschrift, No. 22, 1888.

[7] Centralblatt f. prakt. Augenheilkunde, September, 1887.

Diagnosis.—This disorder is to be distinguished from granular lids by observing that the small bodies are neither so prominent nor so highly colored as hypertrophied papillæ, that the mucous membrane is not affected more deeply than the lymphatic follicles, and that cicatricial changes are not present.

The prognosis is good, in so far that the disease will disappear and leave the mucous membrane in a smooth condition; but the affection is troublesome, in that it lasts for months, and under indifferent hygienic surroundings, especially in crowded asylums, is likely to prove a stubborn endemic.

Treatment.—The patient should be put in the best possible healthful surroundings, and given good food, iron, and quinine. Locally, boric acid, weak bichloride solutions, and occasional dusting in of iodoform and subnitrate of bismuth and calomel, equal parts, are the best measures. A salve of one-half grain of sulphate of copper to the drachm of vaseline has been highly extolled. If refraction-error exists, appropriate glasses should be ordered. Vossius[1] urges the necessity, especially if the disease is endemic in asylums, of excising the affected areas. Galezowski and Schneller have practised similar procedures.

GRANULAR OPHTHALMIA.

Synonymes.—Granular conjunctivitis, Egyptian ophthalmia, Trachoma.

Definition.—This is an inflammation of the conjunctiva in which this membrane loses its smooth surface, owing to the formation of rounded granulations, which, after absorption, leave cicatricial changes: it may be studied conveniently under two forms,—acute granulations and chronic granulations.

Etiology.—Acute granulations may arise primarily under the influence of bad hygienic surroundings, and appear endemically in institutions where the inmates are crowded together. The disease is contagious by the contact of the secretion from one eye with another, and also probably through the atmosphere. The chronic form may result from the imperfect disappearance of the acute granulations, but much more frequently appears as a primary disorder. Certain races are strongly predisposed to the affection,—the Irish, Jews, and Eastern races; children are attacked less commonly than adults. According to Burnett,[1] the negro race enjoys a comparative immunity from trachoma, and he believes the disease should be classed with the dyscrasias. Jackson[2] has seen granular lids in a negro boy of ten years. The Indians are frequent sufferers from granular lids. Overcrowding, bad ventilation, depressed health, are all factors in its production. Since the publication in 1881–82 of Sattler's work in which he attempted to prove that trachoma was due to the presence of a micrococcus, much work has been done in this line, but we are not yet in a position to state definitely the relation of micro-organisms to this disease, nor has the trachoma-coccus been positively isolated.

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[1] Therapeutische Monatsheft, June and July, 1889.

Pathology.—Two views have been prominently held in regard to the pathological anatomy of the "trachoma granules,"—the one that they are derived from the natural lymphatic vesicles, the other that they are to be looked upon as new growths of special pathological character. The latter view is the one which in recent times has received the widest acceptance. The presence of the granulations provokes thickening and vascularization of the conjunctiva, the cellular elements change into connective tissue, and cicatricial alterations take place, so that in the advanced disease the submucous tissue is involved, and finally fatty change in the tarsus arises. With the disappearance of each granulation in a cicatricial mass, shrinking of the conjunctiva takes place, resulting in contraction and atrophy, with hypertrophy and distortion of the lid.

Symptoms.—*Acute Granulations.*—The lids are swollen, the conjunctiva reddened, the papillæ hypertrophied, between which are found nonvascular roundish granulations. The dread of light is intense, the lids are spasmodically closed, and, on their forcible separation, scalding tears gush out. The bulbar conjunctiva is intensely injected, superficial vascularity of the cornea arises, and ulceration, especially of its margin, may appear. Severe eye-, temple-, and forehead-pain results. At first there is little discharge, but later a muco-purulent stage begins, and the process terminates either favorably by the absorption of the granulations, or unfavorably by running into a chronic form.

Chronic Granulations.—Often without antecedent inflammation these appear, usually first on the lower lid, in the form of grayish-white semitransparent bodies, which, accordingly as they resemble minute grains of boiled sago, or vesicles, have been called "sago-grain" or vesicular granulations. At first there is little discharge, perhaps only gluing together of the lids; later, with fresh development of new granulations and thickening and hypertrophy of the papillæ, the secretion becomes freer, and mucopurulent or purulent in character. Granular disease may at any stage take on an intense acute inflammatory reaction, with the production of a copious contagious discharge.

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[1] Medical Record, March 24, 1888.

[2] Polyclinic, January, 1888.

Sequelæ of Granular Lids.—The most important results of long-continued granular lids are trichiasis, distichiasis, and entropion, conditions already described (page 62), atrophy and shrinking of the conjunctiva from cicatricial changes (page 81), cloudiness of the cornea, and *pannus*. The latter is due to the formation of a vascular tissue beneath the epithelium of the cornea, and begins below the upper lid, but may in severe cases involve the entire membrane. The proper corneal tissue may be unaffected, or ulceration and softening may occur. Pannus is usually taught to be caused by the friction of the granulations; but, according to Raehlmann, it is a special implantation of the trachoma-process upon the cornea, a view which receives some support from the observation of Hansen Grut that with the granulations of spring catarrh pannus never occurs (page 93).

Diagnosis.—This presents no difficulties. Acute granulations must be distinguished from purulent ophthalmia, but the chronic form is made evident by the direct inspection of the everted lids.

Prognosis.—Under the best circumstances, granular disease of the conjunctiva, when well established, is a tedious disease and greatly endangers the useful vision of the patient. Relapses are frequent, and the disorder at any time is likely to assume an intense inflammatory action. Its contagious character renders the affection especially dangerous in pauper schools or any institution where large numbers of inmates are gathered together. The muco-purulent discharge, even when present in slight degree, is likely to be conveyed from one subject to the other by the careless use of towels or common utensils. Great caution is necessary under such circumstances to prevent a disastrous epidemic.

Treatment.—*Acute Granulations.*—These must be managed upon the principles which govern the treatment of acute ophthalmias generally, and in the inflammatory stage require soothing remedies rather than strong astringents and caustic applications.

Chronic Granulations.—When the height of the inflammation has subsided, or in the chronic stage, the treatment is directed to the absorption of the granular condition of the lids. For this purpose numerous caustic and astringent applications have been recommended. For routine treatment probably no better ones exist than nitrate of silver (ten grains to the ounce) and a crystal of sulphate of copper. Tannin and glycerin in a strength of twenty grains to the ounce is an excellent application in the milder forms, or after an impression has been made with more decided remedies. Liquid carbolic acid is praised by E. Treacher Collins. Betanaphthol, iodoform, hydrastin, and an ointment of the yellow oxide of mercury are all worthy of trial. Very satisfactory results follow the use of strong solutions of bichloride of mercury, one to one hundred and twenty or one to three hundred, applied to the everted lids with a mop of absorbent cotton, the patient at the same time using a tepid collyrium of the same drug of the strength of a grain to the pint.

More vigorous procedures are scarification of the conjunctiva, abscission of the granulations,[1] and squeezing them out between the thumb-nails, or with a specially-devised forceps as advocated by Hotz. Excision of the fornix conjunctivæ has been proposed and practised by some surgeons, and the operation of peritomy, or the excision of a ring of conjunctival tissue surrounding the corneal margin, is also advised. Any cases of stubborn pannus without ulceration of the cornea may be treated by the production of a violent conjunctivitis with a three-per-cent. infusion of jequirity applied twice a day to the everted lids, a method introduced by De Wecker to substitute the old-fashioned inoculation of the conjunctiva with blennorrhœic pus.

Much patience is required for the successful treatment of granular lids, together with frequent changing of the local applications, and care to discontinue the severe caustics and apply sedative lotions with atropine to prevent iritis if high-grade inflammatory symptoms should set in. The patients usually require a tonic, and must be placed in the best possible hygienic surroundings. If at any time severe swelling of the lids comes on, with dangerous pressure upon the cornea, this should be relieved by the operation of canthoplasty.

CHRONIC OPHTHALMIA (chronic conjunctivitis), a common disease in elderly people, is more rarely seen in children, either as the sequel of acute conjunctivitis or of independent origin. There are hyperæmia, thickening of the papillary layer of the tarsal conjunctiva, swelling of the caruncle, and soreness of the edges of the lids, especially at the outer canthus. If this results from a chronic blennorrhœa, the conjunctiva is thrown into velvety folds and involutions from one end of the lid to the other. Cleanliness, and the application of "lapis divinus," the alum crystal, or painting with a strong solution of bichloride of mercury (one to five hundred), are the best local measures.

TOXIC CONJUNCTIVITIS is a name applied to that form of inflammation of the conjunctiva caused by the prolonged use of certain drugs, prominent among which are atropine and eserine. The disease usually appears in the form of follicular granulations, sometimes associated with considerable swelling of the lids and an eczematous appearance of the surrounding tissue. Conjunctivitis produced by the prolonged use of cocaine has been described by Kipp[2] and Mittendorf,[3] and W. C. Ayers[4] reports granulations from the same cause. These cases occurred in adults.

LYMPHANGIECTASIS OF THE CONJUNCTIVA consists in the appearance of small blisters in the conjunctiva filled with semi-transparent fluid and usually gathered together in masses. They are situated superficially, and readily move with the conjunctiva over the subjacent tissue. The explanation of their appearance is the probable interference of the natural lymph-flow, and the consequent distention of the lymph-spaces. According to Berry,[1] this affection is most frequently met with in children, and, as it disappears spontaneously, it requires no treatment.

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[1] See, especially, observations of Vossius, loc. cit.

[2] Medical Record, October 6, 1888.

[3] New York Medical Journal, October 6, 1888.

[4] Archives of Ophthalmology, September, 1888.

CONJUNCTIVITIS ÆSTIVALIS.

Synonymes.—Frühjahrscatarrh (Saemisch), Phlyctæna pallida (Hirschberg), Hypertrophie pericératique de la conjonctive (Desmarres), Hypertrophia epithelialis æstiva (Emmert), Spring catarrh.

Definition.—This is a form of conjunctival disease usually seen in children, and characterized by photophobia, stinging pain, considerable mucous secretion, and an hypertrophy of the tissue surrounding the limbus of the cornea.

Etiology.—The present state of our knowledge does not yield definite information in regard to the cause of this peculiar disease. The characteristic behavior of the disorder is its return with the early spring, about April, and its subsidence in the fall and winter; although Hansen Grut[2] doubts if the spring and summer exacerbations are the most marked features, and points out how phlyctenular conjunctivitis flourishes in the summer. It is seen most frequently between the ages of five and fourteen years, usually in boys, although Burnett[3] says the largest contingent of cases comes from the female sex. Emmert[4] has observed a case in an individual aged fifty-one, and Saemisch another in a child of three. In Emmert's collection of twenty-nine cases seen during ten years, the greatest number occurred in June, then in May, July, and August; single cases occurred in all the other months except February, March, and April, when none appeared. I have seen one case which began in the child's fifth year, and has reappeared each spring for four years, until last year, when it began in February. It may be accompanied with the disorder known as hay fever. Some writers, like Adolph Bronner,[5] decline to consider vernal conjunctivitis as a distinct disease, but look upon it as a hypertrophic form of chronic conjunctivitis.

Pathological Anatomy.—Anatomically, spring catarrh is a chronic epithelial overgrowth, with simultaneous hypertrophy of the connective tissue, the deeper layers of the conjunctiva remaining tolerably normal. An analogy between it and psoriasis has been pointed out.

Symptoms.—The affection begins like an ordinary conjunctivitis, and is always bilateral. There are photophobia and more or less mucous secretion, with circumscribed pericorneal injection and the formation in this region of small, gray, semi-transparent nodules, which swell up and overlap the cornea, which is usually unaffected, although its limbus may become thickened and corneal opacity result. The disease is most strikingly observed in the bulbar conjunctiva, but three varieties are described,—the limbus, palpebral, and mixed forms. The conjunctiva of the bulb is injected, and from the outer and the inner commissure superficial vessels run and empty into the swelling at the limbus. The conjunctiva palpebrarum is slightly thickened, and of a pale, dull color, as if brushed over with a thin layer of milk (Horner). In severe cases the tarsal conjunctiva is covered with flattened granulations with deep furrows between; the lids droop and give the eyes a sleepy look. A peculiarity of this disease in the negro, as pointed out by Burnett (*loc. cit.*), is the brownish pigmentation of the scleral base of the hypertrophied masses.

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[1] Diseases of the Eye, Edinburgh and London, 1889.

[2] Nordisk Ophthalmologisk Tidsskrift, No. 1; American Journal of the Medical Sciences, September, 1888.

[3] Archives of Ophthalmology, x. 416.

[4] Centralblatt f. prakt. Augenheilkunde, March, 1888.

[5] Lancet, July 14, 1888.

Diagnosis.—The disease is to be distinguished from trachoma by the flattened appearance of the granulations, and the absence of infiltration and of pannus, a fact which tends to prove that the pannus of granular lids is not of mechanical origin. The peculiarity of the disease and its tendency to return with the early spring and subside in the fall and winter are further points to aid in a diagnosis.

Prognosis.—The prognosis of the disorder is not unfavorable, except in so far as the recurrence is concerned. The cornea is not usually affected, although a slight opacity of the border may occur. Its course is a long one, and may last from eight to ten years.

Treatment.—This appears to be efficacious only in so far as relief of the symptoms is concerned, and not in preventing the recurrence. The eyes may be protected with dark glasses,—a procedure, however, deprecated by Horner. Locally, the use of a mild astringent lotion and dusting in calomel are recommended. Dr. L. Webster Fox, of this city, informs me that he has obtained good results with the use of boro-glyceride. Hansen Grut employs the actual cautery to destroy the granulations. Internally, Fowler's solution should be exhibited.

PHLYCTENULAR OPHTHALMIA.[1]

Synonymes.—Phlyctenular conjunctivitis, Scrofulous ophthalmia, Eczema of the conjunctiva.

Definition.—This is a form of inflammation of the conjunctiva characterized by the appearance of one or more white-topped vesicles situated chiefly upon the bulbar portion.

Etiology.—The disease is believed to be of constitutional origin, and has for its subjects strumous and badly-nourished children. Errors of diet, the over-indulgence in pastries and unwholesome foods, the use of tea and coffee, often act as predisposing causes. It frequently follows in the wake of the exanthemata, especially measles. Micro-organisms have been described; Gifford[1] found seven varieties of micrococci, three of which seemed to bear some causal relation to this disease; and E. Schmidt (*loc. cit.*) has described five varieties of microbes in connection with phlyctenular conjunctivitis, but inoculations with them were negative.

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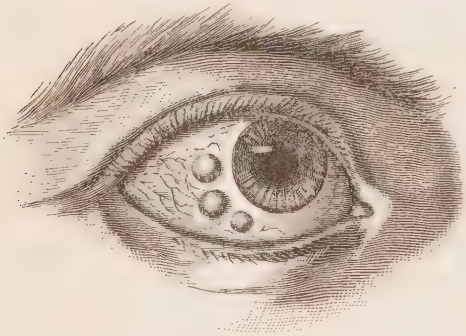
[1] This disease is so closely allied to phlyctenular keratitis that further discussion of it will be found under Diseases of the Cornea.

Symptoms.—The pimples or phlyctenulæ often lie near the corneal margin. They are usually from one to three millimetres in diameter. At first clear, the summit soon becomes turbid and may break down. The disease may exist in a single or a multiple form; usually each phlyctenula is supplied by a leash of dilated vessels.

FIG. 9.



Phlyctenular ophthalmia. (Children's Hospital.)

Sometimes the vesicles are large and yellow, and the disease receives the name *pustular ophthalmia.* Under any circumstances, it is accompanied by pain, dread of light, and increased lachrymation. It is not infrequently associated with muco-purulent ophthalmia, especially when one or other of the exanthemata has preceded its appearance. In a multiple form numerous minute phlyctenulæ are scattered over the conjunctiva, and are accompanied by decided red injection, irritation, and photophobia. When the phlyctenulæ border on the cornea, they frequently invade its substance and form the so-called phlyctenular ulcers, and larger ones are often the starting-point of severe marginal corneal ulceration (see page 104). The disease ordinarily runs a mild course, but tends to recur, just as do the relapsing corneal ulcers. The phlyctenulæ generally break down and disappear in ten days or two weeks.

Diagnosis.—Direct inspection will reveal the characteristic lesions of the disorder.

Prognosis.—This is perfectly good under proper treatment, but neglected cases or such as have been treated by injudicious applications may become the starting-point of severe inflammations.

Treatment.—Locally, the mild antiseptic washes previously described are to be employed, the most generally applicable being a lotion of boric acid. Much irritation calls for the use of atropine drops and the occasional instillation of cocaine. The eyes may be protected with colored glasses. Most important is attention to the condition of the alimentary canal. An excellent regulation treatment is a mild course of mercurial laxatives. Simple nourishing diet, good air, exercise, and internally quinine, iron, arsenic, and, in cold weather, cod-liver oil, complete the therapeutic measures.

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[1] Archives of Ophthalmology, xv. 180.

CROUPOUS OPHTHALMIA.

Synonymes.—Membranous or croupous conjunctivitis, Croup of the conjunctiva.

Definition.—This is an inflammation of the conjunctiva characterized by a soft, usually painless, swelling of the lids, a membranous exudation upon the surface of the conjunctiva, and a scanty sero-purulent discharge.

Etiology.—No distinct cause is known; some relation exists between the disorder, scrofula, and eczema, and a definite age of childhood. The affected patients may at the same time be suffering from a similar condition of the respiratory tract (Knapp). The contagiousness of the disease has not been proved. In forty-five per cent. of the cases collected by Arnold Lotz,[1] one eye only was affected. According to Horner (*loc. cit.*), between birth and four years, among eight thousand cases of eye-disease, only one pure instance was seen. The rarity of the affection is further shown in that eighty-two cases only were seen during twenty years of the Basel Clinic. It is never found among the new-born, and never among grown-up people. In Lotz's analysis, two of the eighty-two cases were under one-half year, seventy-four per cent. between one-half year and three years, and very few among older subjects.

Pathology.—This consists in the formation of a pseudo-membranous deposit of fibrinous character interspersed with lymphoid cells, which is deposited upon the conjunctiva and does not infiltrate the deeper tissues. The formation of the membrane is like that seen in tracheal croup; with this there is a proliferation of the papillary body of the conjunctiva.

Symptoms.—These usually begin with an acute ophthalmia, succeeded by swelling of the lids, which, however, remain soft and pliant and usually not painful to the touch. In a few days the deposit of a characteristic false membrane takes place. This membrane, composed of coagulated fibrin, is rather translucent and porcelain-like in appearance, and begins upon the retro-tarsal folds coating the inner surface of the lids, but does not invade the bulbar conjunctiva. The exudate is often in layers and can be removed easily. After the first removal the conjunctiva beneath is only catarrhal and does not bleed, but later becomes dark, granular, and bleeds freely. The membrane is quickly reproduced, and later there is proliferation of the papillary layer of the conjunctiva. The discharge, which may have been at first profuse, grows scanty. The cornea, except in severe cases, always escapes.

Diagnosis.—The disease may be confounded with ophthalmia neonatorum and diphtheritic ophthalmia. From the former it is to be distinguished by the absence of profuse purulent discharge and the age of the patient, and from the latter by the soft swelling of the lids and the superficial character of the membrane.

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[1] Inaug. Dissert, Basel, 1887.

Prognosis.—It is often a light affection, and in the absence of corneal involvement the prognosis is good. Severe cases, however, occur. Healing takes place in from ten to thirty days. Occasionally the membrane is formed again and again, and the course of the disease may continue for months.

Treatment.—Caustics like nitrate of silver must not be used: Horner states that the only case in which he saw corneal ulceration was where this drug had been employed. The eyes should be cleansed with a solution of boric acid, the membrane removed, and the swelling reduced by the application of cold compresses saturated with plain water, or, better, with dilute lead-water. After the removal of the membrane, subnitrate of bismuth has been dusted upon the surface and yielded good results. Quinine has been used in the same way,—a doubtful expedient. The presence of corneal complications calls for the same treatment as that described under ophthalmia neonatorum.

DIPHTHERITIC OPHTHALMIA.

Synonymes.—Diphtheritic conjunctivitis, Diphtheria of the conjunctiva.

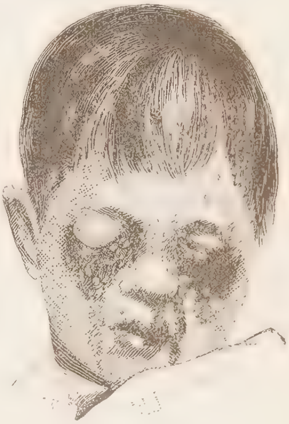
Definition.—This is characterized by a board-like, very painful swelling of the lids; a scanty sero-purulent or sanious discharge; an exudation within the layers of the conjunctiva, which leads to death of the invaded tissues, and tends by spreading to the ocular conjunctiva and by pressure to destroy the nutrition of the cornea.

Etiology.—The disease is contagious, and may originate from a similar case or arise in the course of a purulent conjunctivitis: it has occurred, though rarely, with ophthalmia neonatorum. In certain localities in the south of France and the north of Germany it is endemic. It appears at times in connection with eczema of the face and borders of the lids, and, especially in the discrete form, is an occasional accompaniment of some acute illness like scarlet fever or measles, when the diphtheritic type of the inflammation becomes engrafted upon the conjunctiva. Finally, the most severe forms of the disease are seen during epidemics of diphtheria, and it may be part of a process which passes from the nose to the conjunctiva, or be due to direct inoculation with the diphtheritic poison. The cause of the disease is to be sought in the presence of the micro-organism which is the probable origin of the diphtheritic process. The disease is commonest between the ages of two and eight years, and is rare in young infants.

Pathology.—The condition varies accordingly as the inflammation is superficial, or deep and parenchymatous. In the former class the damaged blood-vessels pour out an exudation rich in albumen, which saturates the dead epithelial cells, forming a coarse mesh-work, while the subepithelial layer is invaded by fibrin and leucocytes. In the parenchymatous form a greater extent of tissue is affected, and there are coagulation and death not only of the epithelial but also of the deeper structures. After the extrusion of the membrane, healing occurs through granulation-tissue, with the formation of cicatrices. Horner has compared the process to the destruction produced by a severe lime-burn. The invasion of the mucous membrane in this disorder is associated with bacteria, and clusters of micrococci, as in diphtheria elsewhere, are found.

Symptoms.—The patches either appear in a discrete form, or the membrane covers the whole inner surface of the lids, and more rarely, in the severe confluent varieties, extends to the ocular conjunctiva. The lids are swollen, very painful, board-like in hardness, and eversion is well-nigh impossible. The false membrane is of a dull-grayish appearance, is torn off only with difficulty, leaving beneath a raw and bleeding surface if the process is superficial, but if it is deep the subjacent structure is pale and infiltrated, and when cut into may be anæmic and lardaceous. If the diphtheritic inflammation has been engrafted upon a case of purulent conjunctivitis, the abundant secretion ceases, or becomes thin, irritating, and sanious. More than in any other disease of the eye the nutrition of the cornea is threatened, and all the destructive tendencies described with ophthalmia neonatorum are apparent. In the severe cases sloughing of the cornea is almost inevitable, coming on with such rapidity that destruction may take place in twenty-four hours. The diphtheritic inflammation may be primary on the conjunctiva, or be part of a process which is seen also in the nose and the nasopharynx. If the skin of the face is the seat of eczematous ulcerations, these also are attacked and covered with patches of false membrane.

FIG. 10.



Diphtheritic ophthalmia. (Children's Hospital.)

Restlessness, fever, generally higher in the evening, alimentary derangements, and nervous phenomena are the usual constitutional disturbances; even fatal cases are on record. When healing occurs, the cornea is found almost invariably to have suffered, and leucomata, adherent or otherwise, may be expected. Conjunctival cicatrices form, and even extensive symblepharon. In a remarkable case which I have described, in addition to the damage to the cornea the patient exhibited a series of nervous phenomena resembling catalepsy.[1]

Diagnosis.—This disorder should be distinguished from croupous ophthalmia and from cases of purulent ophthalmia in which coagulation of the secretion takes place, with which latter affection it has nothing in common. In croupous ophthalmia the lids are supple and painless, the exudation superficial and easily peeled off, the surface soft and congested, the cornea usually free from injury. In diphtheritic ophthalmia the lids are hard and painful, and the exudation removed with difficulty, leaving beneath a pallid and ragged surface, while the whole process tends to mortification of the invaded tissues and destruction of the cornea. The presence of an abundant secretion is the distinguishing feature in cases of purulent conjunctivitis.[1]

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[1] Pepper's System of Medicine, v. 316, article on "Catalepsy," by Charles K. Mills, M.D.

Treatment.—During the earlier stages the best local measures are cold compresses applied in the manner already described. If, however, the corneal involvement is imminent, or already at hand, hot compresses are to be employed frequently for ten to twenty minutes at a time, or even, as I did in one of my own cases, well-nigh continuously. The eyes should be frequently cleansed with a solution of boric acid or bichloride of mercury (one to eight thousand), and atropine drops instilled three times a day, for which, if the ulceration of the cornea is peripheral, eserine may be substituted. Scarification of the conjunctiva, on account of the speedy infection of the spots, is not advisable, and bleeding from the temple in young children is to be avoided. Besides the collyria mentioned, solutions of salicylic acid and carbolic acid have found favor. Vossius[2] has recommended a four-per-cent. solution of salicylic acid in glycerin to be painted every half-hour upon the conjunctiva. Fieuzal[3] uses the simultaneous application of lemon-juice, which is then washed away, and a two-per-cent. solution of nitrate; and Abadie[4] speaks of the application of citric-acid ointment as preferable to the antiseptics. Galezowski[5] has employed oleum cadini (one to ten), Tweedy quinine, and Bergmeister the flowers of sulphur. I tried in one case powdered boric acid, but the result was not favorable. Internally, the most useful remedies are quinine, iron, and mercury; the former should be given in suppositories, the iron as the tincture of the chloride, and mercury either as calomel or the bichloride. Of the latter, half a grain daily may be continued for days, and should be exhibited in milk or water hourly in the dose of one-sixtieth to one-fortieth of a grain to children from three to six years of age. Milk punch may be added if there is depression, and if naso-pharangeal diphtheria coexists the appropriate local measures are to be used, especially as Jacobi[6] has advised nasal injections of the bichloride of mercury, one grain to the pint. If one eye only is attacked, its fellow should be guarded by a bandage in young children, by Buller's shield in older cases. The patient must be isolated, especially if other children are at hand who are suffering from facial eczema or any form of catarrhal ophthalmia.

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[1] I have described croupous and diphtheritic ophthalmia as separate forms of conjunctival inflammation, although many modern writers, like Nettleship, are disinclined to maintain the classical distinction of A. von Graefe, which has also been insisted upon by De Wecker, Tweedy, Knapp, and others. It is perfectly true that croupous inflammation and superficial diphtheritis, here as elsewhere, are closely related, and Juler has examined microscopically cases of diphtheritic and membranous ophthalmia and in each found closely similar appearances; nevertheless, although cases intermediate between the two classes occur, a sufficient number of each class distinctly marked arise to render the maintenance of the differentiation scientifically worthy.

[2] Klin. Monatsbl. f. Augenheilkunde, November, 1881.

[3] Bull. de la Clin. Nat. Ophth., vol. vi., No. 2, p. 57.

[4] Ibid. [5] Ibid.

[6] Proceedings of the Philadelphia County Medical Society, 1888.

TUBERCLE OF THE CONJUNCTIVA is a manifestation of a similar affection of the lymphatic system of the same side, and by preference has its seat upon the palpebral conjunctiva, and rarely upon that of the globe (Horner). Examination of the yellowish masses reveals a granular-like tissue with giant cells, and Fuchs[1] has observed the presence of tubercle-bacilli. The chief symptoms are a somewhat resisting thickening of the lids, and upon the conjunctiva, especially between the tarsus and the retro-tarsal folds, yellowish or gray-red masses, and sometimes, as Rheim[2] has noted in four cases, ulceration with a lardaceous floor. A decided secretion is present, and occasionally swelling of the tear-sac and of the neighboring lymphatic glands; the nose may also be involved; general tuberculosis is sometimes absent. The treatment consists in excision of the masses, the galvano-cautery, or the application of iodoform in powder or salve. A general treatment of tonics, and especially arsenic, is indicated.

XEROSIS CONJUNCTIVÆ.—**Synonymes.**—Keratomalacia, Necrosis corneæ, Infantile ulceration of the cornea, with Xerosis of the conjunctiva.

Definition.—This disease is characterized by drying of the conjunctiva and destructive ulceration of the cornea, and usually appears in infants under one year of age.

Etiology.—Von Graefe[3] taught that this disorder was dependent upon encephalitis, a position which is no longer tenable since the researches of Jastrowitz[4] and Friedlander.[5] It occurs only in anæmic, badly-nourished individuals. It has been seen accompanying measles and variola, and is especially common among sickly children with diarrhœa, and among those inmates of homes whose hygienic surroundings are bad. Leber found and described a double bacillus, but the researches of Weeks,[6] Sattler, and others have failed to confirm this microbe as the cause of the disease.

Symptoms.—In the beginning there is conjunctival congestion, with lachrymation, but the peculiarity of the disorder is the dryness and lack-lustre appearance of the conjunctiva, with the formation of cheesy flakes, while the ocular conjunctiva becomes dry, greasy, and is thrown into folds. A gray haze rapidly turning into ulceration appears in the cornea, followed by involvement of the iris, with the formation of hypopyon. Perforation and destruction of the eyeball may result. Both eyes, as a rule, are affected, —one earlier than the other.

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[1] Abstract in Archives of Ophthalmology, 1887.

[2] Münch. Med. Wochenschr., 1886, Nos. 13 and 14.

[3] Archiv f. Ophth., Bd. xii. Abth. ii. p. 250.

[4] Archiv f. Psych. u. Nervenk., Bd. ii. S. 389; Bd. iii. S. 162.

[5] Berlin. Klin. Wochenschr., 1883, No. 6, S. 90.

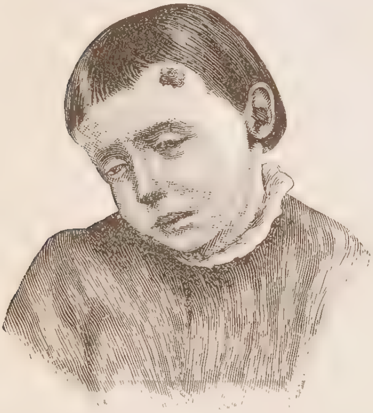
[6] Archives of Ophthalmology, 1886, vol. xv. p. 332.

Prognosis.—This is very unfavorable, not only in so far as the eye is concerned, but also in regard to the lives of the patients: they usually die of the wasting disease which has occasioned the trouble, or of an intercurrent pneumonia.

Treatment.—This resolves itself, besides the local measures of cleanliness, with antiseptic washes and the use of atropine, into the administration of such internal remedies as are called for by the general state of the patient.

Essential Shrinking of the Conjunctiva is a rare disease, in which this membrane atrophies and undergoes contraction until the conjunctival cul-de-sac disappears and the free borders of the lids are fixed to the ball; through exposure the cornea becomes dry and opaque. The process has been mistaken for granular lids, with which, however, it has no association. Some have believed this to be a form of pemphigus of the conjunctiva, and Nettleship (*loc. cit.*) has seen this condition accompany an outbreak of general pemphigus. In other cases no association of this kind was found, and Juler[1] thinks essential shrinking of the conjunctiva and pemphigus quite distinct processes; there is occasional coincidence of the two affections in the same patient. I have observed an instance of this character in a child the subject of hereditary syphilis, who died of phthisis.[2]

Fig. 11.



From a photograph of a patient suffering from essential shrinking of the conjunctiva. (Children's Hospital.)

Treatment.—This avails but little. It has been attempted to keep the conjunctiva moist with glycerin, and rabbit's conjunctiva has been transplanted, but without result.

Pemphigus of the Conjunctiva is a rare affection, characterized by the formation of bullæ associated with pain and lachrymation, and, after succeeding attacks, degeneration and cicatrization of the conjunctiva. It is doubtful whether this occurs as an independent disorder; it is usually seen in connection with pemphigus of the rest of the body. The course of the disease, which tends to recur from time to time, is destructive to the nutrition of the conjunctiva, and later of the cornea. The former undergoes cicatricial change and may grow fast to the ball; the latter becomes opaque and staphylomatous. Interesting examples of this affection in children have been reported by Cohn,[1] by Baeumler,[2] and, in this country, by Tilley,[3] who has well described one of the three American cases.

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[1] Transactions of the Ophthalmological Society of the United Kingdom, 1886.

[2] Transactions of the Pathological Society of Philadelphia, vol. xiii.

Treatment.—This is practically of no avail. Sight, if lost through this disease, cannot be restored, and the best that can be accomplished is relief of the local irritation.

XEROSIS (Xerophthalmos) is the name employed by systematic writers to describe the dry, lustreless, and shrunken appearance of the conjunctiva which may appear either in the parenchymatous or in the epithelial form. The former variety occurs under the influence of trachoma and pemphigus, or in the form of a primary disease just described as essential shrinking of the conjunctiva. The epithelial type of the disorder is seen with infantile necrosis of the cornea (page 99), and in debilitated subjects, accompanied by night-blindness (which see).

LUPUS OF THE CONJUNCTIVA occurs as a primary disease or extends to the membrane from the surrounding integument (page 60). It appears in the form of red, granulation-like patches. In a case reported by Grandmont,[4] inoculation experiments with the excised growth resulted in appearances similar to those produced where tuberculous human lung was employed.

AMYLOID DEGENERATION OF THE CONJUNCTIVA is a rare disorder, in which pale-yellowish masses appear chiefly in the palpebral conjunctiva. It has been supposed to arise from granular ophthalmia, but, according to Raehlmann, these growths are independent of trachoma. They disappear after extirpation, which is the proper treatment, and their structure is the same as that of lymphoid tumors in which a hyaline degeneration may be found, which is in all probability an antecedent condition.

CHEMOSIS OF THE CONJUNCTIVA occurs when the connective-tissue layer is distended with serum, and is often associated with inflammatory exudate. It is mostly a symptom of some other disease,—for example, acute conjunctivitis, choroiditis, iritis, or orbital cellulitis. Severe œdema of the conjunctiva, with great swelling and hyperæmia, may appear without any apparent cause and with marked suddenness. In paralysis of the external straight muscles the overlying conjunctiva is often decidedly œdematous and may be an early symptom of such an accident. I have seen acute chemosis[5] in young adults follow the administration of ascending doses of iodide of potassium, and in one instance succeed a general outbreak of urticaria.

Treatment.—The swelling may be reduced by the application of cold, possibly by pricking the tissues, and later by the use of some astringent, like alum.

HEMORRHAGE FROM THE CONJUNCTIVA.—This usually occurs as an ecchymosis beneath the conjunctiva scleræ, the meshes of the connective tissue being filled with blood-clot as the result of some violent straining during a paroxysm of whooping-cough. It may arise from injury or without obvious cause, and has been seen occurring spontaneously about the menstrual epoch in young girls. Pomeroy[1] has recorded a well-nigh fatal hemorrhage following the instillation of nitrate of silver, and Schmidt-Rimpler[2] has seen death follow hemorrhage from this membrane, but the cause of the bleeding was not determined. Ordinarily, subconjunctival hemorrhage will subside by absorption, and requires no treatment.

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[1] Abst. in Archives of Ophthalmology, 1886, p. 120.

[2] Klin. Monatsbl. f. Augenheilkunde, vol. xxiii. p. 329.

[3] American Journal of Ophthalmology, June, 1887.

[4] Annual of the Universal Medical Sciences, 1889, vol. iv.

[5] American Journal of Ophthalmology, December, 1887.

PTERYGIUM is a hypertrophic fold of conjunctiva extending from the periphery of the globe towards the edge of the cornea. It varies in color and thickness according to the amount of hypertrophy and the presence of blood-vessels. Its most frequent situation, corresponding to the course of the recti muscles, is at the inner side of the eyeball, more rarely at the outer, very exceptionally at the upper or lower. The disease, never of frequent occurrence, is rare in children. Individuals whose occupation exposes them to slight injuries of the eye are predisposed to its formation. Poncet has described microbes in connection with the formation of pterygia. They occasionally arise as the result of a blennorrhœa, during which the conjunctiva has become attached to a corneal ulcer. Knapp (*loc. cit.*) has reported one such case where the pterygium had the superior situation after purulent ophthalmia. Thickenings of the conjunctival membrane in all particulars resembling pterygium have been described as congenital affections. In one such case, recorded by Strawbridge,[3] the growth sprang from the outer commissure and covered the corneæ to fully one-half their surface. The treatment consists either in excision, transplantation, strangulation by means of a ligature, or evulsion as recommended by Prince.

TUMORS OF THE CONJUNCTIVA.—As congenital forms, angiomas, lymphangiomas, dermoid growths, and pigmented spots have been described. Although the latter may be congenital, it should be remembered that they appear after the healing of variolous pustules when they occur upon the conjunctiva. The other tumors which have their habitat upon the conjunctiva are lipoma, fibroma, osteoma, papilloma, and sarcoma. Lipoma, according to Von Graefe, appears to be most common in the region between the superior and the external rectus, and may be mistaken for the dermoid growth (Horner). Osteomas appear as small nodules of bone surrounded by fat and firm connective tissue (Snell, Schweigger). Loring[4] saw a case of this nature in a child eight months old. Papilloma arises from the limbus conjunctiva. I have seen one instance apparently follow a burn of the conjunctiva.[5] Cysticerci have been extracted from the subconjunctival tissue of children by Makrocki,[1] Hirschberg,[2] and others. Sarcoma, both of the pigmented and the unpigmented variety, occurs, usually the former, and generally arising from the bulbar conjunctiva. Fielchenfeld[3] has described an unpigmented sarcoma of the conjunctiva palpebrarum in a girl of sixteen.[4]

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[1] New York Medical Record, August 20, 1887.

[2] Klin. Monatsbl. f. Augenheilkunde, October, 1887.

[3] Transactions of the American Ophthalmological Society, 1873–1879, vol. ii. p. 386.

[4] Archives of Ophthalmology, 1883, p. 523.

[5] Transactions of the Pathological Society of Philadelphia, 1886.

Treatment.—Excision of these growths is the only treatment, either alone or in addition to cauterization of their bases. In sarcoma of the conjunctiva it will be necessary to discuss the propriety of enucleation of the entire eye.

AFFECTIONS OF THE CORNEA.

Equal in importance to the group of diseases just concluded are the affections of the cornea. In Horner's statistics they constitute twenty-seven and two-tenths per cent. of the whole number, and among one thousand seven hundred and eight recorded cases in the Children's Hospital of this city twenty-five and six-tenths per cent. were treated for the various disorders of the cornea. Many types of corneal inflammation exist, and, although it is customary to divide these diseases into suitable groups, it is by no means possible to refer in each instance to one or other of these divisions. Under the general term keratitis we include the divers forms of inflammatory affections of the cornea, and to all of them, if ulceration is present, certain well-marked symptoms belong,—pain, congestion of the vessels of the circumcorneal area, photophobia and blepharospasm, and loss of the substance or transparency of the cornea.

PHLYCTENULAR KERATITIS.

Synonymes.—Strumous keratitis, Pustular keratitis, Vesicular keratitis.

Definition.—This is characterized by the formation upon some portion of the cornea of small papules or pustules, and is accompanied by dread of light and blepharospasm.

Etiology.—The disease is quite constantly seen in strumous subjects. It often follows in the wake of an attack of measles or other acute exanthem. It is distinctly under the influence of climate, and is usually aggravated by warm, moist weather. Micro-organisms probably play a *rôle* in its production, and Burchardt[5] has described cocci which greatly resembled the coccus flavus desidens (Fluegge) and to which he attributed a causative action. There is a close connection between this form of keratitis and inflammatory lesions existing in the nasal fossa which are the source of an infectious pus. Thus, in twenty-six observations Augagneur[1] found the phlyctenules on the cornea succeeded a rhinitis of this character. Martin[2] has attempted to demonstrate a relation between keratitis and astigmatism.

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[1] Abst. in Archives of Ophthalmology, 1884, p. 484.

[2] Centralblatt f. prakt. Augenheilkunde, June, 1879.

[3] Ibid., April, 1888.

[4] For syphilitic diseases of the conjunctiva, see Diseases of the Eyelids, page 57.

[5] Centralblatt f. prakt. Augenheilkunde, February, 1887.

Symptoms.—The phlyctenules, which consist in the early stage of minute subepithelial collections of round cells, appear upon the cornea usually at or near the corneo-scleral junction. They vary in size from a poppy-seed to a millet-seed; their tops, at first gray, speedily grow yellow, break down, and form superficial ulcers. They are accompanied by decided local congestion, increased lachrymation, and photophobia. The palpebral conjunctiva, always hyperæmic, may remain translucent and bathed in tears, or the disorder is not infrequently accompanied by muco-purulent discharge and a velvety condition of the conjunctiva, under which circumstance it is spoken of as *phlyctenular keratitis with catarrh.* When the photophobia is severe, the child buries its head deeply in the bedclothes; the lids are spasmodically closed, rendering inspection of the eye difficult, at times well-nigh impossible. The dread of light and the blepharospasm are probably due to direct irritation of the corneal nerves, as Iwanoff found the cellular infiltration situated along the course of the nerves. The pustule, when it breaks down, forms a *phlyctenular ulcer,* which may remain at its original seat near the margin, or creep towards the centre of the cornea, followed by a bundle of thickly-crowded blood-vessels (fascicular keratitis). These, when the ulcer heals, disappear, but a stripe of opacity remains. Under the name *marginal keratitis* a variety of this inflammation exists where numerous phlyctenules extend along the rim of the cornea, giving rise to a process which may cease here, or may by further invasion produce vascular ulcers. More dangerous than any of the other varieties is the formation of a single pustule just at the corneal border, which speedily ulcerates and is surrounded by a yellow area of infiltration, with a strong tendency to perforate. If these inflammations constantly recur, the cornea becomes clouded, uneven from loss of epithelium, and covered by numerous superficial vessels, the whole forming the so-called *phlyctenular pannus.*

Diagnosis.—This presents no difficulties, the appearance by direct inspection rendering the nature of the disease evident.

Prognosis.—The course varies greatly; in mild cases healing takes place with only a slight loss of substance, and the resulting scar is scarcely discernible. Not so with the severe forms, in which there has been decided loss of substance and a distinct scar-tissue remains, or in which deep ulceration with perforation occurs, or where constantly-recurring vascular ulceration leaves an uneven and roughened surface. In children of the strumous type, especially if their surroundings are unfavorable, phlyctenular keratitis may be one of the most stubborn of corneal diseases.

Treatment.—In order to make a thorough application of the local remedies, the child's head should be taken between the surgeon's knees, while the attendant holds the hands and body, and the lids are separated; the cornea will usually roll out of sight, but may gradually be coaxed into view. Sometimes a lid-elevator is useful, and a few whiffs of ether or of chloroform may be necessary. If much secretion is present, boric acid solution is to be employed, and atropine drops sufficient to maintain mydriasis. Cocaine, judiciously used, will allay the photophobia, but its continuous application when corneal ulcers exist is to be deprecated. Later, an ointment of the yellow oxide of mercury, either with or without the addition of atropine, or calomel, provided no form of iodide is being exhibited, dusted into the conjunctiva, may be employed. The eyes should be protected with goggles, and the child encouraged not to bury its head in the bedclothes. The best possible hygienic surroundings must be obtained, with fresh air and wholesome food. Cod-liver oil, iron, quinine, often suitably given with pepsin, and arsenic, are the most acceptable internal remedies. Douching the eyes with cold water will subdue the dread of light, and touching the ulcerated external commissure, which almost invariably exists in these cases, with a crystal of bluestone, as Koller (*loc. cit.*) has suggested, helps to relieve the blepharospasm. The urine should be examined in all these cases; and scrupulous attention to the condition of the alimentary canal is an important factor in the treatment. If rhinitis is present, this must be attended to: for this purpose Augagneur employs a powder composed of equal parts of pulverized camphor, boric acid, and subnitrate of bismuth. I can recommend this treatment, especially if the nasal fossæ are thoroughly cleansed with Dobell's solution before the insufflation of this powder. In stubborn forms of recurring vascular ulcer and deep ulceration, the use of the thermo-cautery in the manner later described is productive of excellent results. After healing, any refraction-error should be corrected.

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[1] Recueil d'Ophtalmologie, October, 1888, p. 631.

[2] Annales d'Oculistique, tom. xc. pp. 14, 176; tom. xci. pp. 44, 209.

CORNEAL ULCERS.

In addition to the varieties of corneal ulcers just described, others remain which may be classified under four headings:

1. *Small Central Ulcer.*—This appears as a gray or gray-white opacity in the centre of the cornea, and is not accompanied by much vascularity or dread of light. The elevation is slightly cone-shaped until the whitish top breaks down into a shallow depression. Usually single, this form of ulcer may be multiple, and under any circumstances it tends to recur. It is seen in young children who have been poorly nourished and are of a strumous habit. While healing generally occurs with promptness, the tendency to recurrence leaves permanent opacity, which, from its central situation, may seriously impair vision. If neglected, and in patients of bad nutrition, this ulcer occasionally forms an abscess of the cornea.

2. *Excavated or Gouged-out Ulcer.*—This form of ulcer, often seen in children, most troublesome because it is so rebellious to treatment, has its

seat near the corneal margin. Its presence may be entirely overlooked, on account of the absence of congestion, and because in appearance it is a small punched-out excavation, with transparent bottom and free from any opaque surrounding infiltration. The disease probably depends upon some failure in the nutrition of the cornea due to nervous disturbance. When healing is about to take place, the floor of the ulcer loses its translucency and a few vessels of repair pass to its margin.

3. *Shallow Central Ulcer.*—In a certain number of cases a shallow nearly central ulcer appears, with a slightly turbid base, unattended with any considerable pain or photophobia. It is essentially chronic in its course, and when healing finally takes place a faintly opaque facet remains. It is found in anæmic or scrofulous patients, and is occasionally seen in subjects of long-standing granular lids.

4. *Infecting or Sloughing Ulcer* (*Purulent Keratitis*).—Ulcers unattended by vessels of repair, which spread widely from one border and become readily complicated with hypopyon and iritis, and which are often the result of a trifling injury, usually affect elderly people and those whose nutrition is depressed. Most important among these are the *serpiginous* or *creeping ulcer* of Saemisch, and the *circular ulcer*. But sloughing ulcers are not confined to aged subjects: the small central ulcer, as already noted, may spread and form an abscess. Usually in the early stage a nearly central gray area forms, over which the epithelium may be unbroken, though discolored. This speedily becomes yellow, notches laterally, bulges forward, and finally bursts entirely, leaving a more or less ragged ulcer covered with tenacious pus and forming the condition spoken of as *abscess of the cornea;* or it may evacuate posteriorly and cause a collection of pus in the lowest part of the anterior chamber, or an hypopyon. This combination of suppuration in the cornea with pus in the anterior chamber is often called *hypopyon-keratitis,* a name originally suggested by Roser, while *onyx* is the term applied to that condition when the suppuration passes between the layers of the cornea and settles in its most dependent portion. Purulent keratitis is usually accompanied by severe pain in the brow, the eye is intensely tender, and the vision is reduced to mere light-perception. The iris becomes hyperæmic, inflamed, and posterior synechiæ form if unchecked; the entire cornea is reduced to a softened mass, which, when it separates, allows the iris to fall forward and become adherent in the lymph which ultimately closes the aperture, and all the results of perforation follow. (See page 80.)

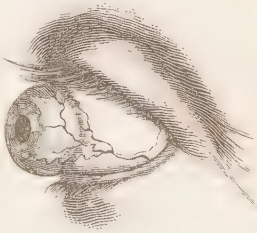
Etiology.—Sloughing ulcers of the character described are dependent upon local infection, and most frequently result from an injury to the cornea from a chip of stone, a chestnut-burr, or the like, which of itself may cause an insignificant wound, but, in an individual unfavorably disposed to kind healing, may terminate, through the microbic infection, in this dangerous form of inflammation. A certain number of cases have been ascribed to cold, and in still others no definite cause can be ascertained. Most violent forms of suppurative keratitis occur during attacks of small-pox; the pustules, however, rarely form upon the cornea. Indeed, it has been said that they are never so situated, though Horner in one instance observed a single ulcer the origin of which he believed to have been a corneal pustule.

Abscess of the cornea occasionally accompanies scarlatina, measles, and typhoid fever; its association with violent types of conjunctival inflammation has already received attention. A variety of abscess of the cornea, non-inflammatory in character, has been described by Von Graefe[1] as occurring in scrofulous children under eight years of age, without any healing tendency and with almost an entire absence of subjective symptoms. The character of the inflammation and the constitution of its subjects have led some to consider it a form of tuberculosis of the cornea, the possibility of which the experiments of Panas and Vasseaux[2] have demonstrated.

Results of Corneal Ulceration.—Opacities more or less permanent follow all ulceration of the cornea. If the opacity is slight, it is spoken of as a *nebula* or *macula*; if dense, as a *leucoma*, which, accordingly as the iris is or is not attached to its posterior surface, is described as *adherent* or *non-adherent*. It is evident that upon the position of the opacity in the cornea depends its influence upon vision. The more central it is, or rather the more directly it encroaches upon the pupillary region, the greater will be the disturbance of direct vision. Irregularities in the curvature of the cornea distort the retinal images and are fruitful sources of mixed astigmatism. When perforation has followed ulceration and the iris has remained entangled in the aperture, an anterior synechia results. An eye thus afflicted may become quiet, and even retain, either with or without operative interference, useful vision; but it may equally well be a continual source of annoyance, subject to recurring attacks of inflammation, and breed sympathetic irritation in the fellow-eye.

The distention of a cicatrix to whose inner surface the iris is attached constitutes a *corneal staphyloma*, which is called *total* when the entire cornea is involved, and *partial* when only a portion is included. The mechanism of this formation is briefly as follows. A perforation takes place, and the iris falls forward and attaches itself to, or protrudes through, the opening, becoming fixed there by the lymph thrown out in the process of repair. The scar-tissue which remains fails to withstand the intraocular tension, and that portion of the cornea is pushed forward beyond its normal curvature, forming a pouch-like deformity, or perchance including its entire surface. The protrusion may flatten down, and under the influence of fresh inflammation bulge forward again, or may extend between the palpebral fissure and prevent the lids from closing.

FIG. 12.



Staphyloma of the cornea.

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[1] Archiv f. Ophth., vi. 2, 135.

[2] Arch. d'Oph., 1885, v. 193.

Treatment of Ulcers of the Cornea.—It is manifestly impossible to lay down hard-and-fast lines for the treatment of corneal ulceration; this must be governed by the exigencies of each case; but certain principles of local treatment are common to the various types.

Pain, photophobia, and congestion are to be relieved by the plans already suggested in treating of phlyctenular keratitis. In mild cases atropine, combined, with due caution, with cocaine, a lotion of boric acid, and a pair of smoked glasses usually suffice. In chronic cases a seton in the temple has been advised.

After the subsidence of the acute symptoms, or when the ulcer from the beginning is unaccompanied by these, local stimulation should be practised. This is best done with an ointment of the yellow oxide of mercury, a small portion being introduced between the lids morning and evening. Calomel dusted into the eye is likewise of excellent repute, provided the patient is not taking iodide of potassium. Direct stimulation of the ulcer with nitrate of silver (five grains to the ounce) has been recommended,—an application of great value if cautiously and properly used. Whenever corneal ulceration is accompanied by conjunctivitis, the inner surfaces of the lids should be daily brushed over with a weak solution of nitrate of silver, and the cul-de-sac carefully cleansed with a boric-acid solution or the collyrium of bichloride of mercury.

In all forms of severe corneal ulceration, in sloughing and spreading ulcers, either with or without hypopyon, other and more decided methods are applicable. It has been and is a universal practice to instil atropine drops, because of their anodyne effect, and because they lessen the liability to iritis, mitigating at the same time the severity of the inflammation through their power to contract the vessels of the ciliary region and diminish the supply of nutritive material to the cornea. In many cases, however, eserine is the better drug, either because it has the power of stopping the migration of white blood-corpuscles, or promotes absorption through dilatation of the ciliary vessels, or acts locally upon the ulceration, limiting the sloughing process. Furthermore, if the tension is raised, it lowers this, but not otherwise. The solution employed may be from one-half to two grains to the ounce. Deep ulcers near the margin of the cornea are those most suitable for its application. Pain is relieved and the process of repair encouraged by the frequent application of hot compresses in the manner already described. (See page 83.)

When by extension of the ulcer perforation of the cornea is imminent, an antiseptic compressing bandage should be applied, to be removed when the necessary local applications are made. Long-continued use of the bandage is often followed by the appearance of an eczematous eruption upon the skin of the lids. This should be treated by dusting the parts with calomel. Catarrh of the conjunctiva contra-indicates the use of the bandage unless the danger of perforation is imminent.

If, in spite of such treatment, the local infection continues to spread, this must be checked by scraping or by the use of the actual cautery. The latter may be either a small Paquelin or galvano-cautery: when neither of these is at hand, a knitting-needle or platinum probe, as recommended by Gruening, heated white-hot in the flame of a Bunsen burner, will suffice. The edge and afterwards the floor of the ulcer should be well burned, and, as Mr. Nettleship has suggested, the burn may be extended a trifle beyond the edge. In like manner an ulcer may be carefully scraped with a blunt curette and thus stimulated to healing. Cocaine renders either of these little operations painless; but in young and restless children a few whiffs of ether are necessary. I have had the most satisfactory results with the actual cautery, and recommend the treatment. After both of these methods iodoform should be dusted into the conjunctival cul-de-sac. The direct application to the floor of the ulcer of a solution of nitrate of silver has already been referred to, and has many advocates.

The formation of an abscess of the cornea or of an hypopyon is the signal for the evacuation of the pus. This may be done by a simple paracentesis of the cornea in its lower portion, or by the more formal procedure of Saemisch, in which a cataract-knife is entered on one side of the cornea with its cutting edge upward, carried across the anterior chamber to the other side of the ulcer, and the section made directly through the diseased area, evacuating thus at the same time the collection of pus in the layers of the cornea and at the bottom of the anterior chamber. A great objection to this operation in children is the difficulty of keeping them quiet after its performance, and thus increasing the liability, always present, of prolapse of the iris. Moreover, it is surprising how in them absorption of the products of an hypopyon keratitis will follow the non-operative treatment already described. The use of the actual cautery has largely substituted the operation of Saemisch.

If perforation occurs, and the vigorous use of atropine or eserine, according to the situation, fails to restore the prolapsed iris, this should be drawn forward through the aperture and excised, or, in the event of a failure, a later iridectomy may be made through an incision in another part of the cornea. The most useful antiseptics during corneal ulceration are boric acid, bichloride of mercury (one to eight thousand), and iodoform in the form of a salve. Stimulating drops of laudanum or chlorine-water are occasionally employed. According to Dehenne[1] and others, irrigation of the lachrymal canal with a four-per-cent. solution of boric acid is of material aid in treatment.

Constitutional Treatment.—Attention to hygiene, diet, and judicious internal medication are of paramount importance. The child should not be penned up in a dark room, but, with the eyes properly protected with goggles, should go out into the fresh air every day. The diet must be nutritious and easily digested: tea, coffee, candies, and pastries are to be strictly forbidden. If struma is present, cod-liver oil, lacto-phosphate of lime, and iodide of iron are indicated; anæmia is best treated with the tincture of the chloride of iron; any suspicion of malarial taint requires the use of quinine and arsenic, while the syphilitic heritage calls for the iodides and mercury, especially in the form of the bichloride; the best laxative is calomel. The urine should be carefully examined for albumen and for the products which indicate imperfect assimilation, and the remedies directed according to the findings. The teeth should always be inspected, and, if faulty, the case turned over to a competent dentist. Bad teeth and proper mastication and digestion of the food are not compatible, and the occasional relation of carious teeth to diseases of the eye is too well established not to render their examination in all cases of corneal ulceration most necessary. In young children the irritation of a new dentition has caused abscess of the cornea, and in the hands of Galezowski[1] the simple lancing of the gums in an eighteen-months-old child was followed by a cure of the corneal ulceration. I have more than once obtained valuable results by such methods.[2] Careful inspection of the naso-pharynx is necessary here, as well as in diseases of the lachrymal apparatus and conjunctiva. This is especially true in the cases of phlyctenular keratitis which are so often accompanied by an irritating rhinitis. For this purpose I have found the powder recommended by Augagneur (*loc. cit.*) very serviceable. (See page 105.)

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[1] Recueil d'Ophtalmologie, 1887, pp. 205–210.

Treatment of Results of Corneal Ulceration.—Opacities, especially in young children, will often clear up in a surprising manner. By far the most satisfactory results follow massage of the cornea, as originally introduced by Pagenstecher and recommended by Snell,[3] Pfalz,[4] and others. The massage movement should be made upon the closed lid of the cornea after the introduction of a small piece of the yellow oxide of mercury salve. Some irritation accompanies the method, which may be allayed by the occasional use of a boric acid and cocaine wash. I have employed massage of the cornea with excellent results.[5]

Dense leucoma cannot be influenced by such practice. Here vision may be improved by an iridectomy for a new pupil, and the appearance of the eye improved by tattooing the cornea with India ink, or, as has been recommended by Vacher, De Wecker, and Levis, the colorings of the iris may be imitated by using for this purpose many colored pigments. In recent years attempts have been made at transplantation of the rabbit's cornea for the relief of these central opacities, and the results of Von Hippel have in one or two instances been encouraging. Martin of Bordeaux, and Strawbridge of this city, have proposed under similar circumstances to trephine the sclera, and thus create a new pupil.

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[1] Journal d'Ophtalmologie, tome i. p. 606.

[2] University Medical Magazine, October, 1888.

[3] Ophthalmic Review, 1888.

[4] Editorial in Medical News, February 23, 1889.

[5] University Medical Magazine, September, 1889.

In partial staphyloma of recent date a compressing bandage is to be applied and eserine drops used daily. If, in spite of this, the bulging continues, paracentesis of the anterior chamber, or an iridectomy opposite the clearest part of the cornea, may be performed. When the staphyloma is complete and unsightly, or if it is the seat of pain, is a source of danger to the fellow-eye, and its vision is destroyed, excision of the globe is indicated, an operation which in children is likely to be followed by a good deal of deformity, owing to the shrinking of the orbit and a failure of development of the bones upon that side. Various substitutes for the excision of the eyeball are practised. Abscission, or the removal of the staphylomatous cornea, leaves a movable stump for carrying an artificial eye. Evisceration is highly recommended by Mules of Manchester and Graefe of Holland. Optico-ciliary neurotomy is an operation not free from danger, and not to be recommended.

VASCULAR KERATITIS.

Vascular keratitis is a superficial vascularity and opacity of the cornea, and is seen in pannus caused by granular lids (page 90), and in the phlyctenular pannus the result of many relapses of phlyctenular keratitis (page 104). Another and the true form of vascular keratitis is characterized by the formation of two opposite vascular areas at the upper and lower margins of the cornea, which approach each other until the vascularization is complete. This disease is met with in young adults and in unhealthy and underfed children. The second eye is usually attacked, and, as Mr. Carter[1] has pointed out, the anatomical disorder indicates a perverted action of the nerves which govern the areas affected, and places it in an analogy with herpes. The symptoms begin insidiously with slight intolerance of light, preceding the appearance at the upper margin of the cornea of a crescent of closely-arranged blood-vessels, which as they advance push before them a border of corneal opacity. Simultaneously the same appearances become manifest at the lower margin. Clearing begins at the borders, and the whitish opacity which remains leaves the centre last of all. The diagnosis is readily made by the appearances pointed out, but in its early stages the lesion may be mistaken for an ordinary conjunctivitis. All the cases must be regarded with anxiety, and some do not clear up entirely.

Treatment.—The principles already laid down with reference to proper diet and exercise should be practised. All local irritants are contra-indicated, but atropine and cocaine and warm fomentations are indicated during the acute stages; later, the yellow oxide salve and calomel may be tried. The best internal treatment is a prolonged course of iron and bichloride of mercury. Iridectomy for a new pupil may be necessary, and, as Mr. Carter has suggested, the convex side of the vascular crescent may be touched with the galvano-cautery.

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[1] Ophthalmic Surgery, by R. B. Carter and W. A. Frost.

INTERSTITIAL KERATITIS.

Synonymes.—Syphilitic, Inherited, Specific, Parenchymatous, Strumous, and Diffuse Interstitial keratitis.

Definition.—This is a diffuse keratitis in which a chronic inflammation of the whole thickness of the cornea takes place, until, without ulceration, the cornea passes into a condition of universal thick haziness.

Etiology.—The majority of cases of interstitial keratitis, as was originally pointed out by Hutchinson,[1] are due to inherited syphilis; in rare instances, to acquired syphilis. In spite, however, of the not infrequent occurrence of this affection, which composes, according to Horner (*loc. cit.*), one-half per cent. of all gathered eye-diseases, and which Hirschberg[2] has found six times in each one thousand cases among sixteen thousand eight hundred observations in eye-disorders, the proof of the cause is not always of ready demonstration. This must be searched for in the family history, the accompanying symptoms, and the affected eye.

Nettleship,[3] writing concerning this point, says, "I have found further personal evidences of inherited syphilis in fifty-four per cent. of my cases of interstitial keratitis, and evidence in the family history in fourteen per cent. more; total, sixty-eight per cent.; and in most of the remaining thirty-two per cent. there have been strong reasons to suspect it." The percentage of cases in which inherited syphilis is the cause is given by Saemisch as sixty-two, Horner sixty-two, Michel fifty, and Hirschberg sixty-one, and according to the latter observer the percentage would probably be higher if the separation of typical cases was made from such as were similar in appearance.[4] A. Trousseau,[5] among forty cases of interstitial keratitis, found three only in which syphilis could be positively excluded. On the other hand, the influence of hereditary syphilis in the causation of this disease has been called in question by a number of observers, and Panas, because of the configuration of the teeth, has sought to bring the disorder in association with rachitis.

Poncet,[6] Javal,[7] and Landolt[8] have seen interstitial keratitis result from malarial cachexia, and Sedan[9] of Toulon analyzed thirty-four cases, finding sixteen times syphilis of the parents, nine times scrofula of the patients, and twenty-seven times malaria. I have seen two cases of this disorder of quite typical course, in one of which inherited syphilis was present, but in the other this could not be demonstrated. In both malaria was evident by direct history and by periodical temperature-ranges. Struma, however, as

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[1] Ophthalmic Hospital Reports, vol. i.

[2] Centralblatt f. prakt. Augenheilkunde, July, 1888.

[3] Diseases of the Eye, 1887, p. 142.

[4] These figures are taken from Hirschberg, loc. cit.

[5] Progrès Méd., May 14, 1887; Centralblatt, May, 1887.

[6] Soc. franç. d'Ophth., 1887, abst. in Archives of Ophthalmology, 1888, vol. xvii.

[7] Ibid.

[8] Ibid.

[9] Recueil d'Ophtalmologie, September, 1887.

Nettleship and others have shown, probably does not in any way originate this disease, because its subjects are not oftener scrofulous than other children, nor do strumous children suffer more from this form of keratitis than from others, while the well-recognized scrofulous eye-diseases are seldom associated with this form of diffuse keratitis.

It is most frequently seen between the ages of five and fifteen, occasionally as early as three years, but rarely after thirty. Among one hundred observations Hirschberg (*loc. cit.*) found thirty-one cases in the first decade, thirty-seven in the second, and sixteen in the third. Power[1] states that an examination of the ward-books of the Ophthalmic Department of St. Bartholomew's Hospital shows interstitial keratitis to be more frequent in females than in males: the average age for males to be attacked is $17\frac{9}{25}$ years, while the average for females is $15\frac{31}{38}$ years; whence it is seen that women are attacked a year and a half earlier, the average being reduced by the greater number of cases occurring about the supervention of menstruation. The greater immunity of the male sex from this disease does not appear in the statistics of A. W. W. Baker and J. B. Story,[2] where in a list of forty-eight cases there were twenty-four instances of each sex. Power has seen interstitial keratitis improve by the development of menstruation, but Mooren[3] has observed the same disease greatly aggravated by the menstrual epoch. The possibility that the affection occasionally may arise in utero has been raised. R. L. Randolph[4] reports a congenital clouding of the cornea affecting two sisters which he looked upon as a congenital form of interstitial keratitis, not differing from the ordinary, or what he called, in contradistinction, the post-natal, form of the disease; and Saltani[5] describes diffuse corneal opacity in three brothers and sisters which he considered the remnant of an intra-uterine interstitial keratitis.

Symptoms.—After a few days of slight ciliary congestion and watering, a faint cloudiness, usually, but not always, near the centre of the cornea, appears. The spots of haze, if carefully examined, will be found to be interstitial opacities,—that is, within the structure of the cornea itself, and not on either surface. In two or three weeks they spread until the whole cornea is invested with a diffuse haziness, veiling or completely hiding the iris, except perhaps through a narrow rim at the margin of the cornea. The steamy surface has often been compared to ground glass. Careful inspection, however, will reveal that the opacity is not uniform, but contains saturated whiter spots scattered through it, or, as Mr. Hutchinson remarks, "centres, as it were, of the disease." There are always at this stage ciliary congestion, some pain, and dread of light. Blood-vessels derived from the ciliary vessels, and formed in the layers of the cornea, are thickly set, and produce a dull-red color, or the "salmon patch of Hutchinson." These patches may be small and crescent-shaped, or large and sector-like. In one type, already described under vascular keratitis, the vascularity creeps from above and below until the entire cornea is cherry-red, a type seen probably when the patient is strumous as well as syphilitic. The subjective symptoms of irritability and photophobia are more pronounced in strumous children. Ulceration rarely occurs. Hutchinson in only one or two cases was able to discover ulcers of distinguishable size. Collins[1] saw four instances, all of which commenced with opacity of the cornea, followed by intense vascularity, which extended over the entire surface except at the central part, which ulcerated, and in one of the subjects perforated. Caudron[2] has recorded examples presenting the appearance of pus in the layers of the cornea; and I have seen in the Children's Hospital of this city, in the eye of a child eight years of age, a dense central opacity with a yellowish collection in the anterior chamber below, resembling an hypopyon.

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[1] Transactions of the Ophthalmological Society of the United Kingdom, vol. viii.

[2] Ophthalmic Review, 1885, vol. iv. p. 321.

[3] Quoted by Power, loc. cit.

[4] American Journal of the Medical Sciences, December, 1888.

[5] Bull. d'Ocul., vol. x., Nos. 10 and 11; also Archives of Ophthalmology, December, 1888.

Iritis and the formation of synechiæ are not uncommon; indeed, Hirschberg (*loc. cit.*) thinks they are practically always present, and that the fundus is more frequently involved than is supposed,—sixteen times in his one hundred cases. Not only may posterior synechiæ form, but Schweigger[3] has reported a case of interstitial keratitis in which, owing to the swelling of the iris, punctiform attachments (anterior synechiæ) formed between it and the cornea without any perforation of the latter membrane. Inflammation of the ciliary region is occasionally encountered; secondary glaucoma and shrinking of the eyeball may follow.

In the course of time, varying in accordance with the treatment, the eye begins to clear, usually from the periphery. Perfect recovery of the transparency must be rare, but often the remaining haze is so slight as to cause but little interference with vision. In bad cases a dense central opacity remains, but even this in time may clear up in a surprising manner. Years after an attack of interstitial keratitis, minute vessels, nearly straight, branching at acute angles and short bends, may be detected in the cornea. According to Hirschberg, the vessel-formation never subsides entirely, and with the aid of a corneal loup he has seen this thirteen years after an attack. The presence of these vessels and the deposits in the retina after the disease may be utilized for the diagnosis of inherited syphilis.

The subjects of typical forms of this disease often present a remarkable combination of physical defects. The dwarfed stature, the coarse flabby skin, the sunken nasal bridge, the scars at the angle of the mouth and the alæ of the nose, the malformed permanent teeth, in which especially the central incisors have vertically notched edges (Hutchinson's teeth), indelibly stamp the inheritance of the patient. Baker and Story (*loc. cit.*) found this character of teeth present thirty-one times among forty-eight cases. The presence of deafness, cicatrices in the pharynx, chronic periostitis of the tibia, and indurated post-cervical and epitrochlear lymphatic glands, still further emphasize the syphilitic taint.

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[1] Royal London Hospital Reports, January, 1887.

[2] Revue générale d'Ophtalmologie, April, 1889.

[3] Archiv f. Augenheilkunde, xvii. 4, 1887.

Diagnosis.—A mistake in diagnosis cannot readily occur. The course is usually quite typical, and the associated symptoms characteristic. The tension of the ball and the age of the patient help to exclude primary glaucoma, while the history and character of the inflammation differentiate it from old corneal maculas and from the diffuse infiltration of the cornea which is sometimes seen as the result of injury. The presence of the minute straight vessels is always good evidence of former interstitial keratitis. These vessels must be distinguished from those which remain after pannus: in the latter they are more superficial and pass into the anterior conjunctival vessels, there are well-formed anastomoses, the broader veins are accompanied by finer arteries, and there are peculiar ramifications of the smaller deep vessels. The vessels seen in corneal scars after ulceration are confined to these; the rest of the cornea is free.

Prognosis.—The duration of the disease is always lengthy; from six to eighteen months are usually consumed in the development of its various stages. The second eye is almost certain to be attacked in from a few weeks to two months; in rare instances the interval is many months, or even a year, and, according to Hirschberg, may be delayed from five to six years. The patient or his friends must be warned of this fact. A return to perfect transparency is unusual; the vessel-formation in the cornea probably never subsides entirely, but even long-continued opacity may often, in the course of time, markedly lessen, and reasonable vision eventually be restored. The occasional onset of deep-seated inflammation in the ciliary region, and the fact that, after the cornea has cleared, the ophthalmoscope may discover evidences of former choroiditis or of glaucomatous cupping of the disk, must not be forgotten in rendering a prognosis. Relapses are frequent, and, as Hirschberg has pointed out, occur not always of the corneal disease, but in inflammations of the iris and retina. Abadie[1] and De Wecker[2] consider the disease more severe than it was formerly thought to be.

Treatment.—All irritating applications are harmful. Atropine, to maintain mydriasis, prevent iritis, and allay inflammation, is to be systematically employed; if the irritation is great, this may be cautiously combined with cocaine. Any high grade of inflammation calls for the frequent use of hot fomentations, and tenderness in the ciliary region will be relieved by a leech placed upon the temple. The eyes may be protected from dust and light by goggles or a dark shade. The best general medication is a long-continued course of mercury. Certainly in children, and probably in all instances, the most satisfactory method of administration in the early stages is by inunction,—one drachm of the ointment rubbed into the skin once or twice a day, according to the circumstances. It is a good plan to order the mercurial ointment put up in one-drachm masses, thus securing the inunction of a definite quantity. The usual precautions in regard to changing the spots for the rubbings must be observed. Whenever slight tenderness of the gums is apparent, the remedy should be discontinued, and the patient put upon a course of iodide of potassium. During the administration of the inunctions, cod-liver oil may be advantageously exhibited; later, a long-continued course of bichloride of mercury is the most valuable remedy, and, as many of the patients are anæmic, this is readily combined with tincture of chloride of iron. A suspicion of malaria calls for quinine and arsenic, and in any event they are useful adjuvants. When all irritation has subsided, absorption of the remaining opacity is facilitated by the use of a salve of yellow oxide of mercury, together with massage of the cornea. Iridectomy, if the tension rises and glaucoma threatens, may be followed by excellent results: that it should be employed for new pupil when stubborn central opacity remains is manifest. Any line of tonic treatment, and due precaution in regard to nourishing diet, exercise, and healthful surroundings, in short, all measures which elevate the standard of the sufferer's general health, are indicated. Abadie and others have recommended that the mercury be given in the form of hypodermic injections. A trial of this plan has not caused me to abandon the older methods.

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[1] Soc. franç. d'Ophth., 1887.

[2] Ibid.

KERATITIS PUNCTATA.—This affection is almost always secondary to disease of the iris, choroid, or vitreous, and is characterized by a precipitate of opaque dots, generally arranged in a triangular manner, upon the posterior elastic lamina of the cornea (Descemet's membrane). The same name is also applied by some writers to those cases in which isolated whitish spots surrounded by a cloudy area appear in the parenchyma of the cornea. The disease is seen in children before puberty, and is probably syphilitic in origin. Inflammatory evidences, the appearance in the cornea of the white dots, and the later development of iritis with more diffuse corneal infiltration, characterize the disease. Iodide of potassium and bichloride of mercury are the proper internal remedies. A continued atropine mydriasis should be maintained; later, iridectomy may be required to check the iritis, or for optical purposes.

MALARIAL KERATITIS.—Any form of keratitis may be aggravated and sustained by the presence of malaria. The relation of this cachexia to inflammatory diseases of the cornea has been especially studied in this country by Kipp[1] of Newark. E. van Milligan[2] has described an essential form of keratitis in association with intermittent fever similar to the keratitis dendritica of Hock, Grut, and Emmert. This appears as a superficial erosion of the cornea, with local pain, photophobia, pericorneal injection, and anæsthesia, together with the evidence of malaria in the spleen, etc. Milligan's cases occurred in adults. In Kipp's cases the disease was observed chiefly in persons between twenty and fifty years of age; five occurred in persons under five years, and one in a nursing baby whose mother had intermittent fever. The treatment of such cases, in addition to local sedative measures, resolves itself into the management of the malaria which is their cause.

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[1] Transactions of the American Ophthalmological Society, 1889.

[2] Centralblatt f. prakt. Augenheilkunde, 1888.

HERPES CORNEÆ.[1]—In herpes zoster of the first division of the trifacial, the eye may become affected, especially if the eruption occur upon the parts supplied by the nasal branch. The pain and swelling of the affected area are so great as often to resemble erysipelas, and ulceration of the cornea and iritis develop. *Herpes zoster ophthalmicus* prevails for the most part among adults, but, as Horner has pointed out, the cornea may also suffer in connection with herpes labialis when this appears in childhood.

Symptoms.—The disease begins with a series of transparent vesicles upon the cornea, mostly near its margin, with pain and marked lachrymation. After a time the vesicles burst, and an irregular area of corneal opacity remains. Iritis may occur. The disease is slow in progress, and, after recovery, opacities remain. Horner observed the disorder in conjunction with labial herpes associated with pneumonia, bronchitis, and catarrhal conditions of the respiratory tract.

Treatment.—This consists in relieving the general condition, together with the local application of atropine and antiseptic washes.

CONICAL CORNEA (KERATOCONUS).—This consists in a cone-shaped bulging forward of the cornea, and is rarely congenital. It is mostly seen in young women, and usually does not develop until after the age of fifteen. Exhausting illness and especially chronic dyspepsia have been assigned as exciting causes, the immediate cause being a disturbance in the relation of the intraocular pressure to the resistance of the cornea. The eye becomes myopic and highly astigmatic. Although cylindrical lenses may not avail in advanced cases, certainly, as Thomson[2] and Wallace[3] of this city have well shown, in some instances their employment increases markedly the visual acuity. If the apex of the cone appears to be thinning, the use of a weak solution of sulphate of eserine and of a pressure-bandage is indicated. Iridectomy and the substitution for the apex of the cone of a contracting cicatrix are the operative measures which have been employed.

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[1] This term is often, as Horner observes, incorrectly used as synonymous with phlyctenular keratitis.

[2] Transactions of the American Ophthalmological Society, 1874.

[3] University Medical Magazine, January, 1889.

Buphthalmos.—**Synonymes.**—Hydrophthalmos congenitus, Keratoglobus, Megalocornea, Glaucoma congenitum.

In this rare affection there is a slow but progressive enlargement of the eye in all its diameters; the cornea is flattened, and the anterior chamber much deepened; the tension is raised. In the course of time the cornea may become cloudy, although this participation is not always present. The affection appears at birth or shortly afterwards, and its incipient stages are believed to be intra-uterine. The precise cause is not accurately determined; it has been ascribed to an intra-uterine irido-keratitis with increased intraocular tension,—in other words, a form of congenital glaucoma. In megalophthalmic eyes, according to M. Durr,[1] the oblique muscles present a greater obliquity than is ordinarily the case, and produce a noticeable compression upon the emergent veins. The prognosis is unfavorable: the affection usually progresses to blindness. Eserine and iridectomy are recommended methods of treatment.

Arcus Senilis, or a circle of fatty degeneration just within the margin of the cornea, is, as its name implies, almost invariably found in old persons. Occasionally, however, a genuine example of this affection appears to have been noted in children. H. F. Hansell[2] reports an instance in a mulatto boy three and a half years of age, resembling in all particulars the arcus of adult life. Canton[3] doubts whether this has ever been seen at birth, and thinks it likely that instances so reported have been due to arciform opacity the result of ulceration. The affection requires no treatment.

Tumors of the Cornea.—These are very rare, and include such growths as develop from the epithelium, as epithelioma, or invade it by extension from neighboring tissues, as sarcoma. Benson[4] has reported an instance of fibroma of the cornea in a girl aged nineteen.

Dermoid tumor occurs as a firm, hemispherical, yellowish-white growth lying partly upon the cornea and partly upon the conjunctiva. The apex, often paler than the rest of the growth, is covered with short hairs. These, however, occasionally grow to an unusual length, as in Wardrop's[5] case, where they protruded through the fissure of the lids and hung down upon the cheeks. If undisturbed, the tumor may slowly enlarge; and Graefe has recorded one instance where the size of a walnut was attained. Bilateral dermoids have been recorded, as in Wallenberg's[6] patient, a child of eight years, the point of origin being the conjunctiva in the neighborhood of the external rectus. It is a congenital growth, and is sometimes associated with

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[1] Annales d'Oculistique, July and August, 1888.

[2] Medical News, April 4, 1885.

[3] Quoted by Hansell, loc. cit.

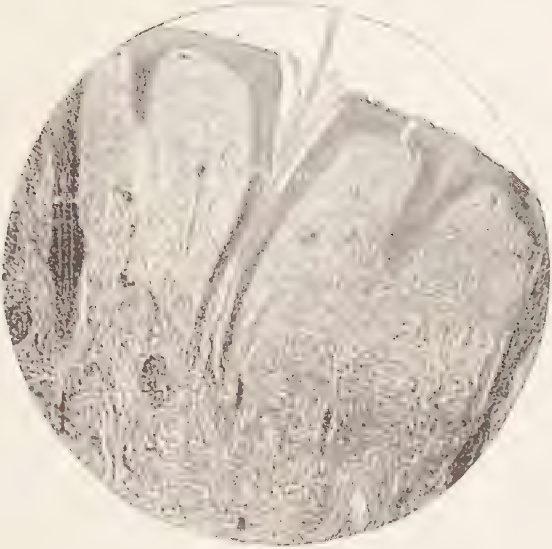
[4] Ophthalmic Review, 1887, p. 5.

[5] Essays on the Morbid Anatomy of the Human Eye, Edinburgh, 1808.

[6] Inaug. Diss., Königsberg, 1889, Schmidt's Jahrbücher, No. 10, 1889.

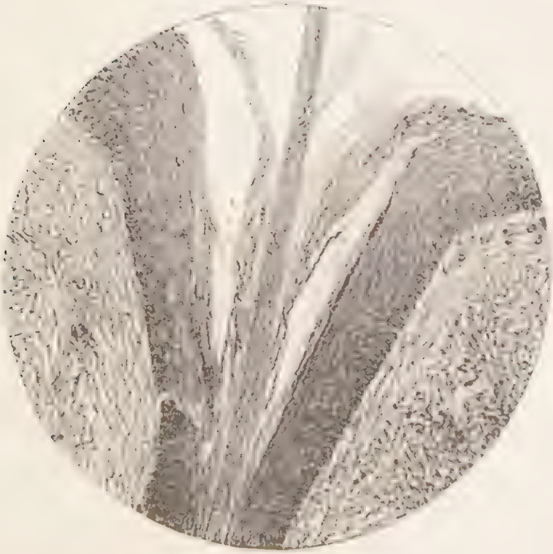
PLATE I.

Fig. 14.



Micro-Photograph from a Section of Dermoid Tumor of the Cornea, showing the General Structure.

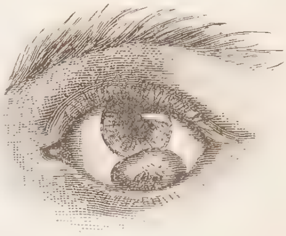
Fig. 15.



Micro Photograph from the same Section, more highly magnified.

other anomalies of the lid and eyes. Picqué[1] has collected ninety-four cases of dermoid tumors of the cornea and conjunctiva, eighty-five of which were observed in human eyes. These dermoids have been ascribed by Van Duyse to the remains of amniotic adhesions; but Picqué, basing his view upon the fact that the structure of the growths very often agrees with that of the lid-border, thinks they result from the coalescence of the eyelids in such a way that at the moment of separation one lid attracts to itself a portion of the other.

FIG. 13.



Dermoid tumor of the cornea. (Philadelphia Hospital.)

Microscopically, the growth represents the structure of the skin and its appendages. The presence of striped muscle-fibre and acinous glands analogous to those in the conjunctiva has been described in dermoid tumors growing from the caruncle.[2]

CONGENITAL ANOMALIES OF THE CORNEA.—Microphthalmos is that condition in which the entire eye remains in a more or less rudimentary state, and in which the cornea is too small in all its diameters. Pure cases of microphthalmos, according to Manz, are among the greatest of rarities; usually one or other of the component portions of the globe is wanting. Numerous theories have been expressed in regard to the etiology,—retarded growth of the cerebellum (Kundrat), incomplete closure of the fœtal ocular cleft (Arlt), fœtal illness *in orbita* (Wedl and Boch), intra-uterine sclero-chorio-retinitis (Deutschmann). This affection has also been ascribed to the influence of heredity.

Megalophthalmos has been described on page 118.

Sclerophthalmia is that condition in which the opacity of the sclerotic encroaches upon the cornea in such a manner that only the central portion remains transparent. It is due to an imperfect differentiation of the cornea and sclera at an early period of fœtal life.

Congenital opacities of the cornea are seen either in the form of milky spots which may clear up in later life, or as dense leucomas. They are due either to intra-uterine inflammation or to an arrest of development.

Congenital staphyloma of the cornea appears in the form of a true staphyloma, and is a rare affection. The abnormality depends not so much upon a malformation, or an arrest of development, as upon a fœtal inflammation which, according to Pincus,[3] takes place in the second half of fœtal life. Heredity probably plays some *rôle* in this and similar affections of the cornea. Bernheimer has seen congenital staphyloma of the cornea associated with dermoid formation.

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[1] Anomalies de Développement et Maladies congénitales du Globe de l'Œil, Paris, 1886.

[2] Wallenberg, loc. cit.

[3] Beitrag zur Lehre vom Staphyloma Corneæ Congenitum, Inaug. Dissert., Konigsberg, 1887.

DISEASES OF THE EYE:

THE ORBIT, SCLEROTIC, IRIS AND CILIARY BODY, VITREOUS, LENS, EXTERNAL OCULAR MUSCLES; REFRACTION; WOUNDS AND INJURIES; SURGICAL OPERATIONS UPON THE EYE.

By CHARLES S. TURNBULL, M.D.,

AND

GEORGE M. GOULD, M.D.

THE ORBIT.

Anophthalmus, or Congenital Absence of the Eyeball.—Although this and other congenital anomalies of the globe of the eye, considered as a whole, may not always be connected with orbital abnormality, they are sometimes so related, and may therefore be conveniently classed in this place. Collins[1] has gathered the details of all the cases of this affection that have been published. They number thirty bilateral and twelve monolateral instances,[2] of which there were post-mortem examinations in nine. As to the primary cause, neither hereditary influences nor consanguinity of the parents existed in a large majority of the cases. There was a maternal impression of fright in a number of the cases, and Collins is inclined to allow this as a possible cause. However well the appendages of the globe are developed, no trace of the globe itself is usually to be found, and the defect seems due to an early developmental cessation, the primary optic vesicle failing to bud from the anterior primary encephalic vesicle. Ten cases are reported as dying within two months from birth, and bodily and mental defects were more or less frequent.

Cryptophthalmus, a congenital union of the eyelids, usually over imperfect eyes.—In this defect[3] the developmental failure seems to be arrested at the formation of the lens: the lids, conjunctival sacs, etc., are wanting or are malformed.

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[1] London Ophthalmic Hospital Reports, vol. xi. p. 429.

[2] Several other cases have since been reported: see Ann. Univ. Med. Sci., 1889.

[3] Studied by Hocquart, Arch. d'Oph., vol. i. p. 289.

MICROPHTHALMUS, OR ABNORMAL SMALLNESS OF THE EYEBALL.—This abnormality is not infrequently coincident with unusual smallness of the head, or defective cerebral and mental development, and is still more commonly associated with such imperfections as cataract, nystagmus, strabismus, corneal, retinal, or nerve disease, subnormality of vision, and even blindness. Deutschmann thinks the cause of these anomalies is to be sought in fœtal inflammatory processes; Hesse[1] inclines to Arlt's theory of a non-closure of the optic cleft; Kundrat supposes a cerebellar failure of development; and Becker concludes that there was a failure of the ectodermal invagination. It is, of course, only in the case of the cataractous complication that any therapeutical or surgical procedure promises to be an advantage. When it is certain that a functional retina and nerve exist, the cataract should be needled as early as possible,—before vision has been lost or deteriorated, and before nystagmus and squint have become fixed habits.

CYCLOPIA.—Fusion of the two orbits into a single cavity in the median line of the forehead, with misformed and functionless remains of one or both globes, is a rare vice of development.

CONGENITAL ABNORMALITIES OF THE ORBITAL WALLS.—These are usually the result of a general asymmetry of the head, and may be so extreme as to interfere with the development of the eye. In some cases the fissures are abnormally placed, confluent, or constricted, and cases have been reported wherein the optic foramen was wanting. Failure in the lamina cribrosa or upper part of the orbital wall may result in a cerebral hernia into the orbit, the sac being composed of the dura mater.

ACQUIRED ANOMALIES OF THE ORBIT.—These may arise from osteomatous and other tumors, from tuberculous disease, or from hereditary syphilis of the walls of the orbit. The symptoms of osteoma will depend upon the position of the neoplasm. If it attain a considerable size, the globe will be pushed either forward or to one side, and, if the pressure upon the globe become so great that the circulation and function of the eye are hindered, visual failure consequent upon atrophy will speedily follow. This is especially true when the tumor extends towards or about the optic foramen. From the uncertainty as to the diagnosis and the impossibility of any therapeutic measures except surgical ones, it follows that blindness usually precedes enucleation, just as enucleation must precede removal of the tumor.

In reference to *orbital tuberculosis*, the coexistence of foci of tubercular deposits or processes elsewhere should lead to the earliest possible diagnosis of the retrobulbar affection. When the diagnosis is certain, no delay must be allowed, and complete surgical removal of every infected tissue is necessary to avoid what is not infrequently a sequel of delay,—thrombo-phlebitis of the orbital veins, and meningitis.

Syphilitic disease of the orbital walls has been rarely observed. The well-known general symptoms of syphilis should, in case of ocular affections of this kind, lead one to the diagnosis.

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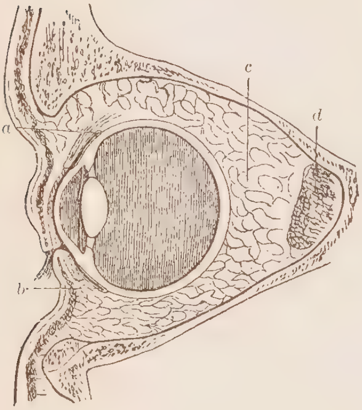
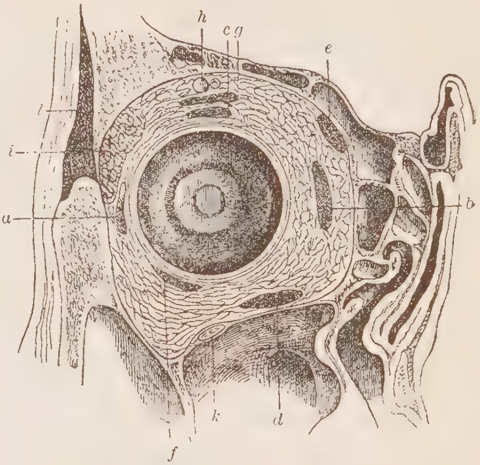
[1] Graefe's Archiv, Bd. xxxiv. Heft 3.

FIG. 1.



ANTERO-POSTERIOR VERTICAL SECTION THROUGH THE GLOBE AND ORBIT.—*a*, *b*, superior and inferior conjunctival cul-de-sacs; *c*, adipose and other tissues of the orbit; *d*, tendons of the external ocular muscles.

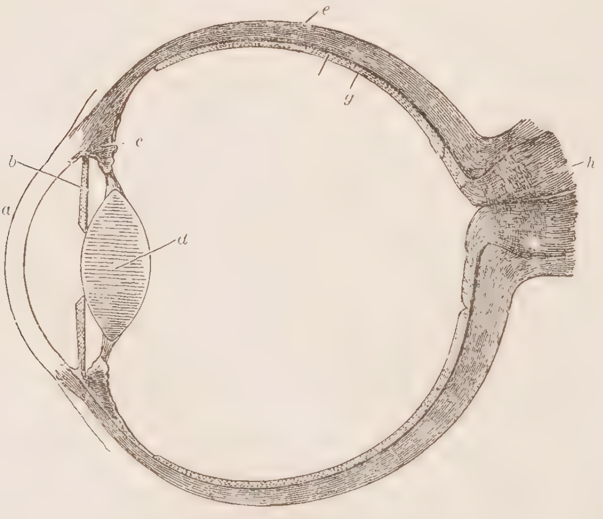
FIG. 2.



FRONTAL SECTION OF THE EYE AND ORBIT.—*a*, external rectus muscle; *b*, internal rectus; *c*, superior rectus; *d*, inferior rectus; *e*, superior oblique; *f*, inferior oblique; *g*, levator palpebræ; *h*, supra-orbital nerve; *i*, lachrymal gland; *k*, infra-orbital nerve; *l*, temporal muscle.

To aid in a more exact comprehension of the relations of the orbital walls and the contents of the orbit, three illustrations are inserted, the first (Fig. 1) showing a perpendicular median section through the globe and orbit from before backward; the second (Fig. 2), a similar section made laterally; and the third (Fig. 3), a schematic section of the globe of the eye.

FIG. 3.



SCHEMATIC SECTION OF THE GLOBE OF THE EYE.—*a*, cornea; *b*, iris; *c*, ciliary body; *d*, crystalline lens; *e*, sclerotic; *f*, choroid; *g*, retina; *h*, optic nerve.

TUMORS AND CYSTS OF THE ORBIT.—These in children are far less frequent than in adults, and consist, so far as tumors are concerned, almost exclusively of sarcomata. Cases of metastatic myxo-sarcomata and of cerebral sarcomata growing into the orbit have been reported, also echinococcus and cysticercus cysts. The symptoms will depend upon the location and growth of the tumor. Exophthalmus and papillitis are the more probable ones, and, where they are not too deeply located, prompt surgical interference may save the eye.

VASCULAR DISEASES OF THE ORBIT.—These in the young are rare affections, and almost without exception secondary to traumatism, to concussion of the brain, or to wasting diseases. Orbital aneurism following some injury will produce pulsating exophthalmus, and ligation of the carotid may become necessary. Venous thrombosis with septic infection is rare and of difficult diagnosis. The exophthalmus of exophthalmic goitre may be classed among vascular affections, but the local abnormality requires no attempt at local therapeutics.

ORBITAL CELLULITIS.—This severe affection may occur in the young as a result of traumatism, following surgical operation, secondary to septic phlebitis, or accompanying panophthalmitis. There will be exophthalmus, inflammatory swelling of the lids, pain, etc. Unless the products of inflammation find an outlet, the pressure upon the globe may endanger its function or existence, so that upon any indication of a pointing of the abscess it should be carefully but speedily evacuated. In lancing Tenon's capsule or the œdematous lids, caution is requisite not to injure the globe. The eye should be kept dressed with hot bichloride dressings, frequently renewed, and the patient's general strength supported by the free administration of tonics, a liberal diet, etc., while the kidneys, skin, and bowels should be kept active.

DISEASES OF THE SCLEROTIC.

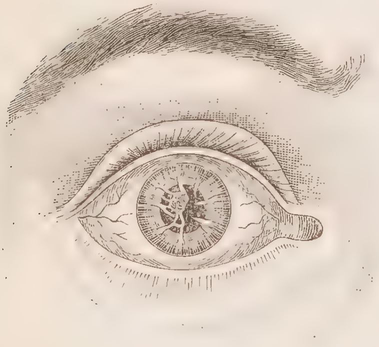
The only primary or idiopathic disease of the sclerotic is scleritis or episcleritis, and this, infrequent in adults, is yet more so in the young. Perhaps it is most commonly met with accompanying keratitis scrofulosa (inherited syphilis) with conjunctivitis lymphatica (Arlt), and is often mistaken for phlyctenular conjunctivitis. Iritis and cyclitis are rarely, if ever, seen in these cases, unless of traumatic origin. Scleritis and episcleritis begin with a localized focus of congestion near the corneal margin. The inflammation is differentiated from conjunctival affections by making traction upon the conjunctiva or lid, and the violet-red, inflamed structures are seen to be beneath the normal conjunctiva. The affection shows a curious tendency to change its location and move circle-wise about the cornea, the former seat becoming normal as the advance is made to a new position. There is little pain, but tenderness to the touch. The greatest danger consists in the liability of implication of other organs,—the cornea, iris, and ciliary body,—and to avoid this the treatment should be prompt. It is better to prevent the irritation arising from the functional activity of the iris and ciliary muscle by using a mydriatic solution of atropine, without a bandage. This will usually serve to cut short the affection. But it will commonly be found that these children have some systemic dyscrasia, and if syphilitic or scrofulous taint exists, or if there is other evidence of defective nourishment, special emphasis should be laid upon the correction of these things. With coincident or resultant iritis, cyclitis, or keratitis the same treatment is almost entirely directed to the important structures threatened.

DISEASES OF THE IRIS AND CILIARY BODY.

Coloboma of the Iris.—Congenital defects of the iris from developmental failure are occasionally met with, and such gaps or breaks in its continuity do not usually interfere with good vision, unless they extensively involve the choroid. They are beyond operative treatment.

Persistence of the Pupillary Membrane.—De Wecker and Landolt,[1] Collins[2] and Wickerkiewicz,[3] have described this congenital anomaly, consisting of fibrillar bands or filaments stretching across the pupillary area and inserted into the anterior surface of the iris. It is chiefly of importance from the fact that it might be mistaken for synechiæ or old iritic affection. Vision is slightly impaired. An illustration of this interesting remnant of fœtal ocular life is given in the accompanying cut (Fig. 4).

Fig. 4.



Corectopia, or Displacement of the Pupil.—In many cases the pupil is congenitally misplaced, so that it is not accurately behind the centre of the cornea nor in front of the lens. In rare instances this ectopia is so great as to become a source of imperfect vision and of unsightliness. As a consequence of traumatism and of operations upon the cornea, the pupil may likewise be drawn to one side, greatly distorted, or even obliterated. In such cases an artificial pupil or an optical iridectomy is advisable, though the results are often unsatisfactory.

Iritis and Irido-Cyclitis.—The iris and the ciliary body are in the young exceptionally exempt from acute or primary inflammations. Almost the only exception to this rule is the rare iritis of hereditary syphilis and of tubercle, and in such cases the existence of other symptoms will be sufficiently pronounced to give the clue for diagnosis. The choroid is almost certain to be implicated in the inflammatory process, and the existence of the eye itself greatly endangered. The somewhat obscure form of iritis called serous iritis, or irido-choroiditis serosa (the keratitis præcipitata of Arlt, and aquo-capsulitis or punctate keratitis of the older writers), is occasionally observed in the young. There is little or no tendency to plastic exudation, and the chief signs consist in minute punctate flecks upon Descemet's membrane, turbidity of the aqueous, increased tension, impaired vision, pain, and a sluggish, somewhat contracted pupil. To these symptoms Horner adds hyperæmia of the papilla and fulness and tortuosity of the circumpapillary retinal veins. The affection is usually a product of some systemic dyscrasia, occurring most frequently in girls approaching puberty, in the scrofulous, the anæmic, etc. The general indications for treatment will therefore depend upon counteracting the systemic abnormalism. If the pressure in the anterior chamber becomes so great that atropine does not produce mydriasis (the pressure preventing its absorption), paracentesis is urgently indicated. The incision should be made with a needle, and sudden or extensive drainage carefully avoided. This may be repeated as often as the tension or pain again becomes extreme. Moderate catharsis, diuresis, and, in severe cases, artificial diaphoresis (by pilocarpine) are advisable.

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[1] Traité complet, tome ii. p. 381.

[2] Royal London Ophthalmic Hospital Reports, July, 1888.

[3] Graefe's Archiv, Band xxxiv., Abth. 4.

Septic or metastatic irido-cyclitis may arise in infants, and has been called puerperal panophthalmitis. It is almost absolutely fatal, and is properly to be classified as an embolic or suppurative choroiditis.

Horner observed one case of *tuberculosis of the iris* in four thousand cases.

Plastic iritis, common in adults, is of great rarity in children, and, when it exists, is secondary to traumatism or affections of the cornea or other neighboring tissues.

As regards the *treatment of iritis*, there are few exceptions to the rule that it is of primal importance to prevent adhesions of the iris to the lens-capsule, or, if they have already formed, to break them up. For this purpose atropine-instillations are demanded. Should these not succeed, or if persistent atropinization produce local or general irritation, mercury by the mouth or by inunction may be pushed until found useless or no longer advisable. We must not neglect to be on the lookout for possible atropine-poisoning in certain idiosyncrasies.

GLAUCOMA.—This is a disease characterized by abnormal increase of intraocular tension or pressure, and, though occurring extremely rarely in children, a few cases have been met with, and a mention of the fact may be made here. The diagnosis depends principally upon the *tactus eruditus*, a sensation of unusual hardness being imparted when the globe is delicately palpated by the fingers. When this excess of tension has reached a considerable degree, there will be pain of the eyeball, radiating to deeper parts, anaesthesia of the cornea, dilatation of the pupil, a shallow anterior chamber, etc., and, with the ophthalmoscope, a cupped disk. The etiology of the affection is obscure. The treatment consists first in repeated instillations of a solution of salicylate of eserine (gr. ¼–½ ad ℥i). When this has been proved to give no relief, a broad, large iridectomy should be made at the superior part of the iris.

DISEASES OF THE VITREOUS.

Persistent hyaloid artery is almost the only abnormality of the vitreous in children, and that needs but a mere mention, since the anomaly admits of no treatment. Indeed, in the majority of cases it does not greatly interfere with the visual function, and is more an ophthalmological curiosity than a disease. Ophthalmoscopically it appears as a translucent mobile fibril extending from the central artery of the retina, or from a branch of the same, through the vitreous to the posterior capsule of the lens. It is the remnant of the artery which in fœtal life nourished the lens and capsule.

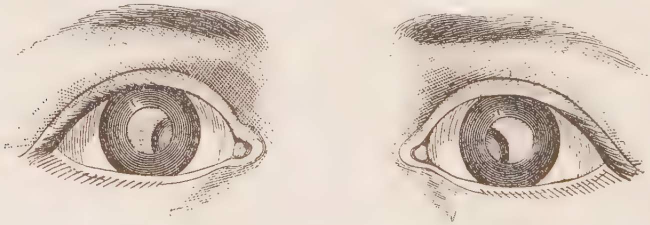
DISEASES OF THE LENS.

The diseases of the crystalline lens consist in defects of form,—colobomæ, etc.,—defects of position,—luxation,—and defects of transparency, or cataract. The lens, not being nourished by blood, and being without nerves, is not subject to true inflammatory action.

Defects of Form.—These are, of course, congenital, and, as the resultant impairment of vision is usually slight, and the condition itself is not remediable by operation, except by extraction of the lens, it follows that but a passing mention is required. Several cases of asymmetry of the lens, of coloboma, etc., have been reported, caused by developmental defect either of the ligament of the lens or of the lens itself. The condition called *anterior lenticonus* or *crystalloconus* consists in a (transparent) pyramidal exaggeration of curvature of the anterior portion of the lens, the anterior chamber thus being more shallow and partially filled by the lens. The obverse of this condition may sometimes exist, and is called *posterior lenticonus.*

Defects of Position.—The lens may be congenitally displaced, and this class of positional defects is called *ectopia lentis.* It is usually symmetrical, and a noteworthy peculiarity is that the displacement is generally upward, or upward and inward. It is more rarely laterally displaced, and never directly downward. An illustration is appended of a case of symmetrical ectopia lentis that was recorded some years ago.[1] (Fig. 5.) The direct cause of the abnormality is doubtless some imperfection in the insertion or inequality of the length of the fibres of the lenticular liga-

Fig. 5.



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[1] Trans. Amer. Ophthalmol. Soc., reported by Dr. W. S. Little, 1883.

ment or zonule of Zinn. Indirectly the influence of heredity is clearly manifest as an etiological factor. In a large majority of the cases the lens remains transparent or semi-transparent. If the ectopia is considerable in extent, the iris may be distinctly seen to be pushed forward by the lens-edge, and that part of the iris that is unsupported by the lens exhibits a tremulousness with jar or motion of the globe. The disturbance of vision will depend entirely upon the extent and the kind of the malposition. Visual acuity will usually be improved by high refractive lenses, concave if the crystalline lens be used as a part of the dioptric system, convex if the functional part of the system be aphakial. All the cases we have seen have been practically aphakial, the lenses being too far removed from the axis of vision to be useful in seeing. Sometimes the lens is not stationary, but, owing to a lax zonule, moves with or follows motions of the eyeball or head. Cases have been reported wherein the lens still in its capsule has passed through the pupil. In such instances the operation of extraction is clearly indicated. An abnormal position of the pupil may sometimes exist, and create much the same result as an abnormal position of the lens. This pupillary anomaly may be congenital or traumatic,—*ectopia pupillaris congenita seu traumatica*,—and the treatment by operation, if any be advisable, will depend upon many varying circumstances.

Acquired abnormalism of position of the lens is usually caused by blows, concussion, or injuries of the eyeball or head, and is called *luxation* or *dislocation.*[1] When the displacement is only slight, it is called *subluxation.* The immediate possibility of luxation depends upon a rupture of the lens-ligament or capsule, so that the lens usually becomes cataractous, either slowly or quickly, from defective nutrition, or from the admission of the aqueous or vitreous humor to its substance, causing molecular and chemical changes that destroy transparency. When the dislocated lens is in the vitreous chamber, its extraction becomes impossible. In such cases, acting, as it does, as a foreign body, the accident may be followed by symptoms of irritation and glaucoma, that may necessitate the enucleation of the globe. Where the luxation is into the anterior chamber, the prevention of such symptoms by means of a prompt extraction is strongly indicated. In this position, or when but partially so misplaced and held in position by the iris, the abnormal position of the lens, if still transparent, may be diagnosticated by the aid of the ophthalmoscopic mirror and from the peculiar position or configuration of the iris. When the lens has become cataractous, its position is easily recognized. In this connection consult also the section on wounds and injuries of the eye, also that on traumatic cataract, and on extraction of cataract.

DEFECTS OF TRANSPARENCY,—CATARACT.—Cataract may be either congenital or acquired; partial (the entire lens not affected) or complete;

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[1] It hardly needs mention that the lens may also be dislocated by intraocular growths pushing the lens out of position or rupturing its ligament, or by such changes in the globe as staphyloma, retro-ocular tumor, buphthalmos, etc., that also rupture the zonula.

hard or soft; capsular (the capsule deficient only in transparency) or lenticular; central or peripheral; anterior or posterior; traumatic or pathological, etc.

The **diagnosis** of cataract is a simple and easy procedure, effected by the aid of reflected and of transmitted light. The pupil should be dilated by a drop or two of cocaine solution (gr. vi ad ℥i) instilled ten minutes prior to the examination, whereby a larger part of the lens is brought into view. By placing the patient so that the source of light is behind and at one side of the head, the light is reflected into the pupil by a twelve-inch-focus ophthalmoscopic mirror, through a central perforation of which the eye is observed. If in place of the usual reddish glow of the fundus-reflection the entire pupillary area is dark or neutral-colored, we conclude—the cornea and remaining ocular media being supposed to be clear—that complete cataract is present. If the crimson fundus-reflex is interrupted by dark spots, striæ, or breaks, a partial cataract is probably to be inferred. Turning the patient so that the light falls a little obliquely into the pupil whilst our own view is more or less direct, and concentrating the illuminating rays by means of a two- or three-inch biconvex lens, we can readily verify the diagnosis. Instead of dark or negative spots as before, we shall now see, by the light reflected from the cataract, light-colored or porcelain-like images of the opacity, whilst the transparent portions of the lens appear dark or invisible. By varying the position and focus of the illumination we can judge of the nature, position, and extent of the opacity. The magnification of the image by a second convex lens may also be found an advantage.

VARIETIES OF CONGENITAL CATARACT.—A primary diagnostic distinction between congenital cataracts consists in determining if the opacity be complete or partial.

COMPLETE CONGENITAL CATARACT is comparatively rare, and not seldom accompanied by microphthalmus or by other bodily or mental defect. The condition itself is an evidence of developmental failure, is binocular, and nystagmus is almost certain to follow sooner or later. As seen by reflected light, the lens appears of a "milky" or opaque-white color. At an early age we cannot always be certain to what extent the retina and nerve may be functional; but if there exists any sign of light-perception, such as that shown in following with the eyes a bright light, or if there are indications of irritation from sudden exposure to such a light, then the plain duty is to proceed with the needle-operation as early as possible. By so doing the development of nystagmus may be prevented or checked, and the possible decay of the deep-seated visual organs, with amblyopia, obviated by bringing them into their normal usage.

Of INCOMPLETE OR PARTIAL CONGENITAL CATARACT the principal varieties are the central, the lamellar, the stellate, the punctate, the axial, and the anterior and posterior polar.

In *congenital central cataract* we find a white opacity occupying the central part of the lens, the rest of the body being normally transparent. The visual acuity is better than would be supposed. The origin of the defect was in some nutritional failure or abnormalism of development in the sixth or seventh week of intra-uterine life, when the nucleus of the lens was in process of formation. Other ocular defects, as microphthalmus, capsular opacities, etc., may also be present. If the opacity be of considerable size and interfere with vision very greatly, or if nystagmus be established or even threatened, either an optical iridectomy or the needle-operation should be advised at once.

Lamellar or *zonular cataract* probably arises in the same way as the central variety, but at a later period of fœtal life. The lens, as is well known, is composed of strata or layers, sometimes likened to the layers of an onion. Some developmental or trophic disorder of intra-uterine life intervening at the period of formation of a layer renders it translucent or opaque either in part or entirely. The defect is, therefore, of both eyes. By attentive observation the transparent nucleus and the transparent external layers may be distinguished from the stratum of cloudy tissue. If for any general reason operative measures may not be carried out at once, continuous artificial mydriasis may be temporarily useful in giving better vision or in preventing nystagmus. But this, for obvious reasons, is objectionable for any great length of time, and, supposing that vision is decidedly subnormal, it is advisable to operate as soon as possible, for reasons already given. If the opacity be limited in extent and an artificial pupil promise to give a clear space for the light to enter, an optical iridectomy is to be preferred to the needle-operation, since in the latter case the power of accommodation is destroyed and the patient henceforth becomes the slave of two pairs of spectacles. But if an iridectomy promise or give no satisfactory result the lens must be broken up and given over to the process of absorption. In older children, when the lens-substance has attained a considerable degree of consistency or hardness, it has been proposed, after breaking up the lens, and after the resultant softening and partial liquefaction have taken place, to extract the lens-matter by a suction-operation, thus obviating the delay and possible danger of the absorption-process.

The varieties of congenital partial cataract denominated *stellate, punctate*, and *axial* indicate peculiarities in the position or form of the opacity. The stellate opacity consists of a star-like figure occupying the anterior portion of the lens-substance. In the punctate the opacities are small, and scattered like dots throughout the lens. In axial or fusiform cataract the axis or region of the antero-posterior diameter of the lens is occupied by a cloudiness or opacity that interferes with vision in proportion to its equatorial extension or the degree of opacification of the affected tissues. As regards the advisability of operative measures, the indications already specified obtain also in these cases.

Anterior capsular cataract is also called *anterior polar* and *pyramidal cataract.* The opacity is at the anterior pole, and is composed of a hyperplasia of the capsular epithelium, which is covered by the capsule, the lens itself remaining normally transparent. The defect may be limited in area, —that is, not extending far towards the equator of the lens; and this is the more usual form. It may also be even with the rest of the capsule, elevated, or extending forward from it. In one case, a child of ten years, that came under our observation, the pyramid extended forward like a glistening white cone through the pupil and anterior chamber, the needle-like point seeming almost to touch Descemet's membrane, and the contracted iris apparently hugging the base of the pyramid.

The **etiology** of this form of cataract is peculiar, and is probably to be explained in this way. It is known that the condition may arise after birth from the contact of the anterior surface of the normal lens with an inflamed cornea, either perforated or not. The same factors are supposed to operate in intra-uterine life. The fact that after birth no defect of the cornea is to be found does not seem to negative the supposition. Whether the abnormal contact of the two surfaces be brought about by the emptying of the anterior chamber succeeding ulcerous perforation of the cornea, or by temporary shallowness of that cavity, or by abnormal pressure either in front or from behind, the essential mechanism remains the same: the corneal inflammatory product passing through the capsule sets up a hyperplasia of the internal epithelium, which results in the capsular opacity, whether the latter be pyramidal or simply leave the normal contour unchanged. The deepening of the anterior chamber by re-formation of the aqueous, or the removal of pressure, etc., again pushes the lens back to its normal position, but carries with it the lasting spot or cone of opaque tissue.

The **treatment** is, of course, operative, and if, as is usual, the opacity is limited to the central pupillary area, an optical iridectomy should be effected at an early date. The position of the iridectomy should be in the superior segment of the iris and slightly to the nasal side, because in this position the upper lid covers the large peripheral part of the opening and leaves the more central aperture for visual purposes. The iridectomy should be as small as possible in all such cases. In this manner the function of accommodation is preserved. But if this position is covered by a segment of opaque corneal tissue, another must be chosen. It may also rarely happen that the capsular opacity extends so far towards the equator of the lens (shown by extreme mydriasis) that no good could come from an iridectomy. In such cases the lens must be extracted by needling and absorption.

CONGENITAL POSTERIOR POLAR CATARACT.—Sometimes a dense white opacity is found at the posterior pole of the lens, limited in area, and in appearance like that just described, but pointing into the vitreous chamber. Moreover, like the anterior variety, it is confined to the capsular tissue, and the lens is not implicated. Its etiology, however, is very different, since it consists, anatomically, of the remains of the vascular structures and connections with the fœtal hyaloid artery, that have not atrophied or become transparent, owing to some disturbance of the normal development. The visual disturbance is in this defect not generally considerable, and will seldom require operative interference.

One form of *congenital membranous cataract* not yet described is seen in poorly-developed eyes: in cases where one eye is microphthalmic we often find a tough membrane like a collapsed lens-capsule without any contained lens-substance occupying the position of the lens, and, although we have what we consider good light-perception, operative interference yields no satisfactory results. The membrane has no elasticity, and tears but does not gape. Mydriatics have but little effect, the irides seeming to be undeveloped, and we are apt to poison our little patients before we secure much, if any, effect. Such cases require the entire removal of a piece of the tough membrane before a useful pupil is secured. Here, then, our prognosis must be guarded, for after having secured an excellent pupil the retina may be defective in development. The teeth of such children are frequently ill developed, few, and shark-like, but in no way like syphilitic teeth. Several operations may follow, and an immense amount of time and patience be sacrificed for little if any result, and prove a bitter disappointment both to operator and to parents.

VARIETIES OF ACQUIRED CATARACT.—As in congenital cataract, so also in the acquired, the most marked distinction lies between the total or complete and the partial or incomplete. In the first class we have to consider two principal varieties,—the soft, including the fluid or "milky," the Morgagnian, and the membranaceous; and the hard, similar in nature to senile cataract. Of acquired partial cataract the chief varieties are the lamellar, the anterior polar, and the posterior polar. Traumatic cataract may be reckoned with total cataracts, but, owing to the complications of the case, it is desirable to classify it alone.

SOFT ACQUIRED TOTAL CATARACT.—In these cases the appearance of the lens, as well as its consistency, may vary within wide limits. The lens may be of a uniform opaque white, or present a delicate striation at the anterior pole; it may look like milk slightly tinged with a blue cast, and be evidently very fluid in consistency,—called *cataracta lactea;* or it may consist of a more solid nucleus floating in a liquefied cortical mass (*Morgagnian*); or, lastly, it may give evidences of a shrunken and folded lens-capsule (*membranacea*). Nutritional disturbance undoubtedly plays the chief *rôle* in the production of soft cataract. Any constitutional disturbance or affection, anæmia, wasting or infectious diseases, cardiac disease, etc., may, by depriving the lens of normal quantity or quality of nourishment, superinduce opacification of its substance. Heredity has also been found to exercise a powerful influence. The direct mechanism of its production consists in the superabundant imbibition or collection of fluid within the capsular cavity or among the lenticular fibres. With a functional retina and good general health, discission of soft cataract should be undertaken as early in life as possible.

HARD ACQUIRED TOTAL CATARACT.—It is often difficult to decide in the case of the young whether cataract be hard or soft. The differential diagnosis is important only in reference to the choice of the method of extraction. The large majority of all cataracts in the young are soft, and usually there is a milk-white and as it were swollen appearance of the lens. Occasionally, and especially, it is believed, in disease of the vessels, particularly of the carotid and its branches, cataract in the young approaches the hardness of the senile variety. In such a case it should be extracted in the same manner as acquired senile cataract.

Traumatic Cataract.—Any injury, whether by direct penetration of a foreign substance or by indirect concussion, that breaks or ruptures the capsule of the lens and permits the ingress of the extra-capsular fluids of the eye, will produce cataract. (Occasional cases have been reported in which a small penetrating body has produced only a limited and as it were encysted area of opacification, leaving the rest of the lens permanently transparent.) With the entrance of the external fluid the lens-fibres swell and break down, until finally the entire body of the lens has been liquefied and carried off by the excretory channels exactly the same as after the discission operation. During this absorption-process the eye should be kept at rest by paralyzing the accommodation with atropine, whereby, also, the iris will more certainly be kept free from entanglements with the capsular or cortical remains. It is possible that a portion may resist the solvent action of the aqueous and require needling. While the lens-matter is swelling, symptoms of increased tension of the eyeball must be watched for and proceeded against as in glaucoma. We must also see that no scraps of lens-substance fall against and adhere to Descemet's membrane, thus causing a large central opacity. Rest for a few days in bed after each needling is advisable in the majority of cases.

Acquired Lamellar Cataract.—This variety is also called *zonular* or *perinuclear* cataract, and is the most frequent of acquired types. It arises in precisely the same way as the congenital. During youth new lens-layers are being formed, and systemic nutritional failure or abnormalism during the formation of a lamina results in its partial or complete opacification. Subsequent strata formed during better health are again normally transparent, so that if the defective layer is not so opaque as to prevent observation of parts beyond it, we may with widened pupil trace the double layer of semi-transparent fibres enclosing a transparent nucleus and surrounded by more external transparent strata. The greater number of these cases may be traced to rachitis. In one hundred and eighty-nine cases Von Arx found that over eighty per cent. showed signs or gave histories of this affection. In the greater number of instances the coexistence of rachitic teeth or cranial asymmetry will point out the general causal nexus. Nystagmus and strabismus are not frequent, but are sometimes complicating results. The advisability of operative measures, and the choice of operation to be carried out, depend upon the extent of visual defect. If not more than one-half the normal, and particularly if the defect be binocular, it will be advisable to operate if we are convinced that the defective vision is entirely due to the cataract. Should mydriasis expose a clear lens-space at the superior or inferior and nasal segment of the lens, an iridectomy should be done at this place. But when no such clear space exists, or when the opacity is extensive and extreme, extraction of the entire lens is seldom done, discission usually being the preferable method.

Posterior Polar Cataract.—The acquired type of posterior polar cataract differs from the congenital in etiology and in the tissue affected. It will be remembered that in the congenital type only the capsule is affected, the lens-substance remaining clear. In the acquired variety the posterior layers of the lenticular fibres are diseased, the capsule preserving its normal clearness. From this fact the latter class is sometimes designated as *true*, and the congenital as *spurious*, or *false*, posterior polar cataract. Acquired or true posterior polar cataract is secondary to chronic choroidal disease, hemorrhage into the vitreous, and other affections of the vitreous. Retinitis pigmentosa is in its later stages very apt to be associated with this type of cataract. The prognosis is grave, and operation, either from its uselessness or from its danger (the eye being usually otherwise seriously diseased), is generally inadvisable.

Acquired Anterior Polar or Capsular Cataract.—The opacity is limited to the capsule, and consists of a proliferation or hyperplasia of the capsular epithelium, caused by the absorbed products of a perforating corneal ulceration. It is not thought that actual contact of the lens-capsule with the posterior surface of the cornea is always necessary, though this is the most common origin. A perforating ulcer makes an outlet for the aqueous fluid; the anterior chamber is thereby evacuated and the lens pushed forward against Descemet's membrane, where it comes in contact with the toxic matter of the ulcer, and this being absorbed by the capsule induces the opacification. With the closure of the ulcer, and re-formation of the aqueous humor, the lenticular system is again pushed back to its normal position. The cataract being usually limited in superficial area requires only a small optical iridectomy at the superior and slightly inner aspect of the iris, by which nearly normal vision will be preserved and the accommodative function retained.

DISEASES OF THE EXTERNAL OCULAR MUSCLES.

The difficulties and perplexities experienced in endeavoring to arrive at an understanding of the abnormalities of the ocular muscles arise from three principal sources. In the first place, the disturbances are exceedingly complex in origin and kind; then, they are in many directions entirely involved in mystery, and extensive and painstaking research is required for their thorough comprehension; and lastly, due in great part to our ignorance of the real nature and origin of the pathologic processes, there is an unfortunate difference of opinion among writers upon a majority of the subjects connected with the study. Add to all this a nomenclature that is often misleading, nearly always illogical and inexpressive, and always unscientific, and we have a consensus of difficulties encountered immediately on entering upon the subject. In a work of the present kind, however, designed not for ophthalmic specialists, we may ignore the more recondite and exceptional phases or aspects, and point out the simpler and more common examples of disease which the general practitioner will encounter. These may conveniently be grouped under four heads,—spasms, paralyses, strabismus, and insufficiency; though the last two names are in many respects highly objectionable terms.

Spasms of the Ocular Muscles.—Like other muscles of the body, the external muscles of the eye may be seized with either clonic or tonic spasmodic activity. The only example of *clonic spasm* that we need to consider is the mysterious and chronic form called *nystagmus*, consisting in, or rather evidenced by, continuous pendulum-like, oscillatory, or tremulous motions of the eyeball. Both eyes are affected, and the movements may be of all degrees of rapidity, and may be from side to side,—horizontal nystagmus,—rotatory, vertical, etc. The affection may arise from visual defect of many kinds, whether caused by corneal disease or damage, by cataract, or by affections of the deeper structures; or it may be produced by cerebral disease of any kind that interferes with the normal function of the centres. It is not infrequently associated with developmental defects and anomalies of the brain and mental faculties. In so far as nystagmus is dependent upon peripheral ocular defects that are remediable,—*e.g.*, in cataract,—prompt action should at once be taken to give the patient the best vision possible before the visual function has been irreparably damaged and before the nystagmus has become too firm a habit. When dependent upon chronic cerebral or spinal disease, as in hydrocephalus, meningitis, neoplasms, hereditary ataxia, etc., the prognosis and treatment are usually hopeless.

There may also be noted a temporary form of clonic spasm due to acute nervous or cerebral disease, as in epileptic attacks, apoplexy, etc.

Tonic spasm of single muscles of an eye has been observed as a reflex from local irritation either of the eye or of adjacent parts, and in acute localized cerebral disorders. Spasm of the corresponding muscles of the two eyes is called *conjugate deviation*, and is an evidence of central disease, —tumors, apoplexy, meningitis, syphilis, traumatism, hysteria, etc. The lateral muscles are those usually affected, the eyes turned either to the right or to the left. Sometimes the superior recti are seized and the eyes are rotated upward. The cerebral lesion may be either in the cortex or in the pons, or in the internal capsule. The direction, right or left, towards which the eyes are turned in spasm may indicate the location of the cerebral lesion. In spasm the eyes, as it has been expressed, look away from the lesion, in paralysis they look towards it. Spasm of the orbicularis is called blepharospasm, and is usually of reflex origin, especially from astigmatism.

Paralysis of the Motor Muscles of the Eye.—It may be safely said that few affections or none offer the physician more recondite problems and require more skill and knowledge in diagnosis than those pertaining to the etiology and location of the lesions causing the paralyses of the various ocular muscles. It is, for example, with our present knowledge, often quite impossible to decide whether the lesion is focal or peripheral, temporary or permanent, direct or indirect, etc., and whether therapeutic or operative treatment is the more advisable.

Paralysis of the external rectus is the most frequent of single-muscle paralyses, due, as Gowers points out, to the long course of the sixth nerve over the pons, which renders it peculiarly subject to accidents of pressure. The paralyses may therefore be upon one or upon both sides. The function of the external rectus is to rotate the eye outward. If the paralysis be complete, and not, as sometimes happens, partial, the diagnosis is easily made: the eye cannot be rotated outward past the median line. If the paralysis be recent, diplopia will be complained of, the distance of the images apart increasing the farther the object fixed is carried towards the side of the paralyzed muscle. The object being held towards the affected muscles and above a horizontal line, the image of the unsound eye is seen as if directed away or inclined from that of the sound eye, whilst below a horizontal line the false image is inclined towards the other. This is caused by the torsional action of the oblique muscles. Coupled with paralysis of the opposite side of the body, paralysis of the external rectus almost certainly points to a hemorrhage or other lesion of the pons.

The superior oblique is the only remaining muscle whose supply is the sole function of a single cranial nerve,—the fourth. Paralysis of this muscle is not of great clinical importance, and may exist without complaint. It is chiefly evidenced by interference with motion downward when the globe is at the same time turned to the nasal side.

Paralysis of the third nerve may be partial or complete. If complete it is called ophthalmoplegia externa, and, unless plainly orbital in origin, points to cerebral basal or crus lesions. This nerve, as is well known, supplies all the remaining muscles of the eye except the external rectus and superior oblique. Therefore, when completely paralyzed, we have a striking symptom-complex, consisting of complete ptosis, immobility of the globe inward, upward, and downward, stabile mydriasis, and paralyzed accommodation. Paralyses of individual branches of the third nerve supplying separate muscles are comparatively easy of diagnosis. Paralysis of the inferior oblique alone is rare and unimportant. In paralysis of the superior rectus, motion upward is impaired; in that of the inferior rectus, the corresponding motion downward; in that of the internal rectus, the internal rotation is defective. In all such cases, if acute, there will be diplopia corresponding to each, with increase of the distance between the images as the object is moved towards the side of the paralyzed muscle, characteristic inclinations of the images, etc. In determining the seat of the lesion we have to consider the history, the coincident affections or paralyses of other muscles, including those of the face and the whole body, the completeness and duration of the paralysis of the affected muscles, the existence of other diseases, as tubercle, syphilis, tumor, etc. The evident existence of orbital disease, the preceding history of exposure to severe cold or of orbital injury, the history of a forceps-delivery of the child, and other considerations of a like nature, may at once show the lesion to be peripheral. Other symptoms of cerebral or basal disease serve to locate the lesion beyond the orbit. The therapeutic measure to be adopted will of course depend upon the diagnosis and location of the lesion. Locally three plans of treatment are offered,—the electrical, the orthopædic, and the operative. Galvanization or faradization of the affected muscles has in some instances seemed to do good. The same may be said of the plan of passive exercise by seizing the conjunctiva at the corneal border with the fixation forceps and rotating it in the direction of the weakened muscle. Exercise with prisms, the stereoscope, or atropine may prove beneficial. Operations are advisable only when other means have failed and when a year or more has elapsed without improvement. Then tenotomy of the antagonistic muscle, or tenotomy with advancement of the paralyzed one, may be undertaken.

STRABISMUS,—SQUINT OR "CROSS-EYE."—This term properly denotes a symptom, and, in fact, strabismus is a symptom of paralysis of the ocular muscles, of tonic spasm, etc. Some authors make the word cover cases of insufficiency. We shall use it only to express the fact that in non-paralytic cases when observing an object with both eyes uncovered, the visual axes do not cross upon the object. Instead of the term latent strabismus, often met with, the word insufficiency may be taken to denote those cases in which the visual axes do meet at the object, but only by a strain or an excess of contraction or innervation upon the part of certain muscles of one or both eyes. If the visual axis is displaced to the nasal side, it is called convergent strabismus; if to the temporal side, divergent. The rare conditions in which the visual line is abnormally directed upward or downward are called respectively sursum vergens and deorsum vergens. If the strabismus is of one eye, it is called monocular or monolateral; if of both eyes, binocular or bilateral. If one eye always deviates, it is called persistent; if either eye successively, alternating. Sometimes the deviation only takes place at more or less regular periods of time, and it is then called periodic. In extreme degrees the fact of deviation is plainly evident, but in lesser degrees it may be shown by directing the patient's gaze at an object and alternately covering and exposing first one and then the other eye. When the deviating eye is forced to fix upon the object by covering the sound eye, the motion of the globe becomes easily recognized. The amount of deviation may be measured by the strabometer or the perimeter. When the deviating eye follows the other in its movements, it is called concomitant strabismus, in contradistinction to paralytic strabismus, in which the motion of one eye is absolutely limited in certain directions. The deviation of the squinting eye is called the primary deviation. If in a given position the squinting eye be forced to fix the object by covering the sound eye, it will be found that the sound covered eye is now squinting. This is called the secondary deviation, and is due to the fact that an excess of innervation is required in the acting muscle of the squinting (though temporarily fixing) eye, and the same excess is also supplied the sound covered eye, because both are dominated by the same centre. Concerning the origin of strabismus and of the amblyopia of the squinting eye, there is at present much difference of opinion. According to the view of Donders, convergent squint arises from the excess of accommodative action in hyperopia, convergence and accommodation being always associated actions. Schweigger, on the other hand, explains the defect by the natural preponderance of the interni over the externi. A third view, but one that, so far as hyperopia is concerned, is in fact but a logical extension of that of Donders, consists in what has been called the innervation theory, advanced by Grut, according to which not the muscles but their innervation is made to account for the strabismus, whether convergent or divergent, and whether associated with hyperopia, emmetropia, or myopia. The question as to whether the amblyopia of the squinting eye is a result or a cause of the strabismus is also a disputed one. The probability is that it is both, the one aiding and exaggerating the other. Concerning this vexed question a parenthetical remark may be interposed that, according to the theory of one of the writers,[1] the long-continued existence of ametropia by supplying the macula with an unphysiological and irritating stimulus itself produces a chronic form of macular choroido-retinitis ("central choroiditis") or pigmentary degeneration and amblyopia, regardless of whether strabismus exists or not, but certainly increased by insufficiency. If found true, this theory will account for many cases of amblyopia heretofore considered as the result of disuse (*amblyopia ex anopsia*).

The following facts therefore stand out clear and undisputed: that most cases of convergent strabismus are associated with hyperopia, or hyperopic astigmatism, and most cases of divergence with myopic defects, and that the most defective eye ametropically is usually the squinting eye.

When a patient with recent paralysis of the external oblique comes for treatment, the first complaint is naturally of diplopia. But in cases of concomitant strabismus there is no such complaint, though the two images are formed upon non-identical points of the two retinæ. How is it that, as is demonstrably the case, the patient sees with the deviating eye and yet does not see double? One explanation is that the mind suppresses the image of the squinting eye, a fact illustrated by the microscopist or ophthalmologist, who keeps both eyes open while using his instrument. Another explanation is also given, that by long habit the mental projection of the image of the deviating eye is such that it corresponds to the true position of the object. Unconsciously the mind makes proper allowance for the malplaced retinal image.

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[1] See Archives of Ophthalmology, vol. xix. No. 1, 1890.

In the treatment of strabismus the first and most important proceeding is to correct the ametropia and thus establish the normal relationship between accommodation and convergence or divergence. It has been customary to say that this is of little avail, but it is our opinion that this latter opinion is not always to be justified, and because of these reasons: 1. That an accurate correction of the ametropia has not been ordered. Reliance has been placed upon the fact that some physician has ordered glasses, probably without the use of a mydriatic and by ophthalmoscopic examination alone,—in which case no reliance whatever is to be placed upon the accuracy of the so-called refraction, regardless of what the skill of the adviser may be. 2. Sufficient time has not been given the muscles to reassume a condition of equipoise or co-ordination. 3. The weaker muscle has not been aided and partially relieved of its strain by a partially-correcting prism combined with the spectacles. By carrying out these measures we can bear witness to the relief of strabismus, in many cases of quite decided long standing and convergence. It hardly needs to be said that in the most pronounced cases, and especially in older children, only operative measures, as a rule, promise success. The cases in which the spectacles do succeed in keeping the visual axis fixed illustrate and prove the innervation theory of the origin of strabismus negatively as well as positively, since, at least for a long time after beginning to wear them, disuse is at once followed by a resumption of the original squinting position. The eye having the greater total lateral motility is the squinting eye, whether the squint be convergent, divergent, or alternating; and this eye is also the more amblyopic of the two.

It is simply a necessary corollary of what has been said that the earlier in life a tendency to squint is arrested by the "atropine-treatment," or by the spectacle-correction of the coincident ametropia, the more promising the result and the more certain that the abnormalisms of amblyopia and squint will not become fixed. The question at once arises, what shall be done with children too young to wear glasses? If the strabismus is convergent, stop all studies and near work. If the convergence still continue, institute the atropine-treatment,—*i.e.*, paralyze the accommodation of the non-squinting eye by a weekly or bi-weekly instillation of a strong solution of atropine, and thus force the squinting eye to assume a permanent normal position until glasses can be worn. It may be added that, if the spectacles be substantial and fitted with judgment and care, children may safely wear them at a much younger age than is commonly supposed. It is not unsafe to prescribe spectacles for a child of five to seven years if the optician and mother (or nurse) are properly instructed and do their respective duties. Prior to this age, if the atropine-treatment has been judiciously carried out, neither the amblyopia nor the deviation has probably become extreme or confirmed. We wish particularly to emphasize the value of what we have called the "atropine-treatment," which may be instituted in babes as soon as squint has made itself manifest. In this way a convergent may be turned into an alternating squint, and both the amblyopia and the extreme loss of muscle-balance and development be prevented until an age is reached when spectacles can be worn. But it must be added that for the success of this plan of treatment watchfulness on the part of the physician must be supplemented by systematic, persistent, and intelligent co-operation on the part of the mother and nurse.

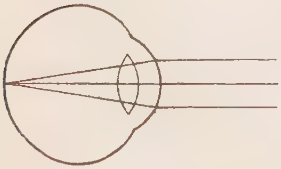
When the strabismus continues despite the spectacles, long worn, tenotomy of the overacting muscle may be carried out; but never, of course, without first having accurate ametropic-correction spectacles ready to be worn from the time of the operation. The extent to which the indirect fibrous attachments of the muscle are to be cut depends upon the degree of the squint. But, since the operation is both painless and without great danger, it is best to err upon the safe side and cut too little rather than too widely. While the tendon is knitting in its new position, all near work should be absolutely forbidden except when under the atropine-treatment explained above. Experience shows that the permanent results of the operation are not settled for several months. There should, therefore, be no haste to repeat or do other operations when at first the results seem unsatisfactory. So long as frequent testing shows the muscle-balance to be in a state of change for the better, no second operation should be undertaken. But if at last it is seen to be necessary, we have to choose between recutting the same tendon and a tenotomy of the corresponding muscle of the other eye, with an advancement of the tendon of the counterbalancing muscle of the squinting eye. It may be necessary to unite advancement to tenotomy of the first operation when the deviation is so extreme—thirty degrees or more—that even a large single tenotomy will not give motion enough to the globe. After the operation atropine should be used in the non-operated eye, in order to hold its fellow in function and by use strengthen it. In divergent squint tenotomy of the externus is rarely useful without advancement of the internus.

INSUFFICIENCY, sometimes called *latent strabismus,* is a term used to express those incoördinations of the external ocular muscles in which the visual lines of binocular vision still meet upon the object, but in which this is effected by an abnormal strain upon or innervation of certain of the muscles. It may be called immature strabismus, or strabismus may be called adult insufficiency. It is evident that it is a more patent form of ocular irritation or "eye-strain" than strabismus, since in it the muscles only keep the visual lines joined at the object by abnormal or over action, whilst in strabismus strain is renounced with the fact of the squint and the giving up of binocular fixation. Insufficiency is almost always present in greater or less extent when there has been long-existing uncorrected ametropia. As in strabismus, so here also, the interni are generally the overacting and the externi the underacting or insufficient muscles in hyperopia and hyperopic astigmatism, the reverse being the condition in myopic defects. There are also in the majority of such cases ophthalmoscopic evidences more decided in one eye of macular injury, with pigmentary stippling and abnormalism and subnormal visual acuity. The defect is roughly estimated in decided cases by fixing the gaze first upon a distant and then upon a near object, and noting the lateral deflection of the eye when it is shut out from beholding the object. The more accurate measurement is made by placing a six-degree or an eight-degree prism, base up or down, before one eye (with ametropia also corrected), and a plane colored lens before the other. The two resultant images of a flame placed twenty feet away appear to normal eyes in vertical alignment. The deviation from verticalness in eyes with incoördinate muscles is measured by horizontally-placed prisms, and gives in degrees the measure of the insufficiency. Where the interni are weaker than the externi, or where a prism with its base to the nose, axis one hundred and eighty degrees, is required to align the two images vertically, it is best to give at once full correction of the insufficiency when ordering the refraction correction, the amount being divided between the lenses of the two eyes. Where the externi are the weaker of the two muscles, and especially if the evidences of eye-strain have not been pronounced, the ametropic may be prescribed without the prismatic correction. In all low degrees (3–5) of external insufficiency it is well to try non-correction for a considerable period first. When the amount is high, partial correction may be at once given. Tenotomy, either complete or "partial," is rarely necessary in insufficiency, and a good, thorough mydriatic refraction will correct both ocular and muscular asthenopias.

REFRACTION.

Emmetropia, or refractional perfection of the visual mechanism, exists when the image of a distant object (theoretically at an "infinite" distance, —practically twenty feet away) is, with paralyzed accommodation, correctly and sharply focussed upon the macula. This condition is diagrammatically illustrated in Fig. 6, in which parallel rays of light (that is, those nearly or supposedly so) from a distant object are united at a point upon the retina. Ametropia, or refractional abnormality, exists when the image is not so focussed. The reason that it is necessary to paralyze the accommodative apparatus to determine these conditions is that the function of the accommodation consists in bringing to a focus rays from an object nearer than the horizon or the so-called "infinite distance." In measurements of the refraction of the eye, this function must therefore be left out of the count, in order that by our correcting lenses we may put the ametropic eye into a condition such that the full amount of the accommodation may be left in reserve for "near work." Such a condition may be called artificial emmetropia. The varieties of ametropia are hyperopia, myopia, and astigmatism.

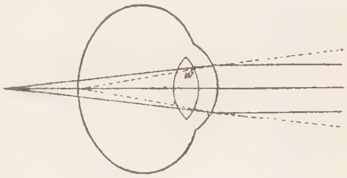
Fig. 6.



HYPEROPIA, or HYPERMETROPIA, commonly called *far-sightedness*, is that condition of the eye or its media in which, with suspended accommodation, the focus of rays of light from a distant object is, or tends to be, behind the retina. It may arise from a subnormal refractive power of the ocular media (cornea, aqueous, lens, and vitreous), or from an abnormally short antero-posterior diameter of the eye. The effects in both cases are the same, so that clinically the distinction is without interest. From the definition above it is readily seen that even for distant objects the accommodative apparatus of the eye must exert itself beyond the normal in order to bring the focus forward upon the retina. When the object is placed within a foot of the eye, the rays from it are of course more divergent and require still greater power on the part of the accommodative apparatus to keep the focus at the retina. From this excess of work and strain arise most of the manifold evils of eye-strain.

An illustration of the hyperopic condition is annexed (Fig. 7). From this it is seen that such an eye is only capable (with suspended accommodation) of focalizing convergent rays. But such rays do not exist in nature. Hence the reason for the excess of focalizing power required of the hyperopic eye. Hyperopia is the normal condition of the animal, savage, and infant eye, and from the fact that the antero-posterior diameter of the eye may increase with growth and age, or that the ocular media may gain additional refractive power, it is clear that it is possible for a hyperopic eye to become emmetropic, or to progress thence into myopia. But these steps can never be retraced: myopia can never develop into emmetropia or hyperopia.

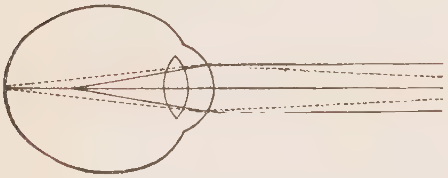
FIG. 7.



The diagnosis of hyperopia, if the child can read letters or figures, is made, and the degree estimated, by means of the test-types and test-lenses, the accommodation having been previously paralyzed. Any refraction, of whatever nature be the defect, is not to be relied upon as accurate that has been estimated with the accommodation functional. The biconvex lens that gives normal vision "$\frac{20}{20}$" or greater, is the measure of the absolute refraction. But we can be sure of this result only when we have proved that no astigmatism coexists, and this, if possible, more unexceptionally demands accommodation paralysis. Each eye must be tested separately. The work as estimated in this way may be "proved" by other methods,—by retinoscopy, the ophthalmoscope, the prisoptometer, etc.; but perfect reliance cannot be placed upon any method except the one first briefly described. In the case of the illiterate and of young children who have not learned their letters, purely objective methods will have to be pursued. Physicians vary in their choice in such cases, and the judgment of a skilled specialist is required to make a diagnosis close enough to accuracy to promise relief.

In MYOPIA, or "near-sightedness," the globe is too long or the refractive media are relatively too powerful. This condition is illustrated in Fig. 8. The natural media focus the rays in front of the retina, the eye being adapted only for very divergent rays, or those from very near objects. Hence to see distant objects a concave lens is used, or one rendering the rays more divergent, so that the natural focus is put back upon the retina. In both hyperopia and myopia it is the diameter of the globe along the visual axis that usually decides the condition, rather than anomalousness or disease of the ocular media. The eye, as is well known, is always under a physiological tension from within outward. If, therefore, the sclerotic be weakened beyond the force required to resist the outward pressure, there follows a distention at this weak point. Such bulging is usually at the posterior pole of the globe ("posterior staphyloma"). The result is myopia. Many theories have been evolved to account for the existence and increase of myopia, but almost all investigations point to the baneful influence of work at short range that is a result of our educational, social, and commercial habits of life. Especially to school-pressure, with poor light, improper desks, and other unhygienic circumstances, is credited a large share in the increase of myopia particularly in European countries. It should not be forgotten that *myopia in the young is itself a pathological condition*, and should be looked upon as a disease whose tendency to increase is to be avoided by all possible safeguards.

FIG. 8.



The subjective symptoms of myopia are simple inability to see distant objects clearly, and in high degrees of myopia a necessity of bringing the book and near work close to the eye. It should be remembered that in hyperopic and astigmatic defects we may have precisely the same symptoms, but there will in such cases be also one or many of the various reflex and irritative symptoms of eye-strain. In myopia uncomplicated by astigmatism and insufficiency there is a noteworthy absence of reflex symptoms. This is because, properly speaking, no eye-strain exists. The endeavor of the eye is to render the lens flatter or less convex, to relax the accommodation beyond its extremest point. Strain may arise from overaction of the interni caused by the great proximity of the work at near range.

Objectively the ophthalmoscope reveals choroidal changes and degeneration in the neighborhood of the optic disk ("conus" or posterior staphyloma) and the macula which in malignant or progressive myopia may proceed to large atrophic patches, hemorrhages, etc., and even to retinal detachment. The diagnosis of myopia is proximately made by the ophthalmoscope, but only with certainty by the test-lenses and distant test-letters. The accommodation must be paralyzed in order to be sure that astigmatism is not present, or, if present, to estimate it correctly. It is commonly said that a mydriatic is not necessary for the correction of myopia. We would not refract child nor adult without it, for we have had many patients who had passed through other hands, and who through reliance upon this erroneous rule had missed correction of the complicating astigmatism that was the source of the reflex troubles. The weakest biconcave lens which, with the astigmatic correction added, gives the best acuity at sixteen inches' distance, will be found the most satisfactory for constant use. Full correction is another and a fatal common error. In degrees above three dioptrics reduction of nearly one-half must be made from full correction, in order to give the eye its least straining action for habitual use. The full measure of the myopia throws upon the ciliary muscle the same excess of work that exists in hyperopia. The amount of the reduction must remain a matter of judgment; the size of the defect, amount of accommodative power, nature of the occupation, etc., are always to be considered.

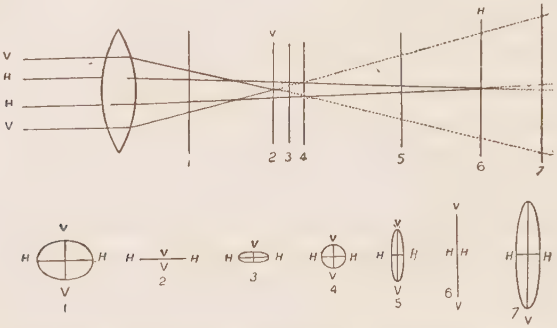
Where myopia exists in children and shows a tendency to increase (*progressive* or *malignant* myopia,—a sad and unfortunate condition), strict instruction should be given as regards the light in near work, which must be clear, steady, and strong, and strike the page *from behind* and one side. Large-type books only should be allowed. The book or work must be held high and well away from the eye, as nearly upon a level with the eye as possible, writing-desks must be sloped and high, the hours of near work reduced to a minimum, frequently interrupted, etc. If the myopia, despite these precautions, still continue increasing, all near work should be forbidden, and the child made to live as much as possible in the open air, being allowed to "run wild." The eyes are certainly more valuable than the so-called education. Perhaps after a few years the myopia may become stationary, and then studies can be cautiously renewed. But such a stationariness does not usually arrive before adult life.

The word Astigmatism is derived from ἀ, "without," and στίγμα, "a point,"—because a cone of light-rays proceeding from a point is, by an astigmatic eye, not brought to a point upon the retina, the focus of the rays in one meridian being either a little in advance of or a little behind the retina. This condition is usually the result of corneal asymmetry, the curvature of the different corneal meridians being unequal, and the retinal image (with suspended accommodation) being, as a consequence, irregular and imperfect.

The cut on the following page may help to a comprehension of this defect. The horizontal lines *H*, *H* enter the cornea at its least curved meridian, and hence their focus is behind that of the more curved meridian *V*, *V*. It is thus seen that the figure or image never makes a true point, but is shaped as shown by the sectional views in 1, 2, 3, 4, 5, etc.

It is evident that astigmatism may complicate either hyperopia or myopia, and in fact it does do so in the great majority of cases. Astigmatism is said to be regular when the two meridians of greatest and of least refraction are at right angles to each other. Irregular astigmatism is rarely met with, and is usually the result of wounds or injuries of the cornea. When one meridian is emmetropic and the other astigmatic, the variety is called *simple;* when the general refraction is hyperopic or myopic with coexisting astigmatism, the terms *compound hyperopic* and *compound myopic* are used; when one meridian is hyperopic and the other myopic, the astigmatism is designated as *mixed.*

FIG. 9.



It can be accurately diagnosticated only by suspension of the accommodative apparatus and the use of the test-lenses, with whatever other devices the individual choice of the oculist may prefer. The existence of uncorrected astigmatism is a prolific source of mischief, and its complete and accurate estimation often demands the greatest skill, patience, and judgment. The ciliary muscle by its unequal and partial contraction seeks to neutralize the effects of the unsymmetrical cornea upon the traversing cone of light, and this unnatural action is often the principal source of the consensus of morbid symptoms called eye-strain. Full correction of the astigmatism is always to be prescribed for constant use.

THE CONSEQUENCES OF UNCORRECTED OR IMPROPERLY-CORRECTED AMETROPIA.—Their name is legion. Few subjects in medicine are more disastrously and inexplicably misunderstood and ignored than this. Numberless lives have been wrecked in consequence, and there are in every city thousands of living examples of the fatal negligence or ignorance of the evil influence of eye-strain upon the growing organism and general health. Physicians may vainly continue for years to treat their patients with every article of the materia medica in the hope of relieving a headache that springs from ametropia, a chorea due to eye-strain, or an anæmia or a dyspepsia that arises from the malassimilation and anorexia whose proper name would be a reflex ocular neurosis. Limitations of space prevent a complete exposition of the subject here, and we can only refer to articles previously published.[1]

Concerning headache, it is at last becoming a matter of common knowledge among the laity that it may be "due to the eyes." That it is generally due to them, and that its true origin is frequently unsuspected by the general practitioner, is a daily experience of every ophthalmologist. But it is a somewhat novel and apparently absurd thought that the gastric and assimilative functions are usually profoundly impaired in the majority of cases of severe irritational ametropia. We have found clinically that headache usually precedes, and is usually continuous with, the gastric trouble. This last commonly consists first in an unaccountable loss or fickleness of appetite. As the irritation has created an abnormal amount of nervous energy, nature seeks an equipoise by lessening the production at the point of origin. The mechanism might be not inaptly likened to the governor of a steam-engine,—the greater the speed the more the steam is shut off below. An analogous but reverse process physiologically is the automatic mechanism whereby deoxygenated blood, by its action upon the centres of respiration and cardiac inhibition, quickens the action of the heart and lungs. With failing nutrition there is general diminution of vitality, a growing languor and malaise, alternating with periods of excessive hyperæsthesia of the nervous system. The irritation continuing, the anorexia proceeds to fits of nausea, and even vomiting, ending finally in one of the many forms of chronic dyspepsia, or "sick headache." The physician has been appealed to, and long courses of dieting, artificial foods, bitters, mineral acids, or tonics, have been tried in vain. Doubtless every physician vividly remembers a number of such puzzling cases. We have had a great many such cases in which, within a week or two after the wearing of proper spectacles, the gastralgias, dyspepsias, and loss of appetite disappeared, and within a few months the body-weight increased from ten to twenty pounds. If the spectacles are accidentally broken, the symptoms quickly recur.

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[1] Especially to Clinical Illustrations of Reflex Ocular Neuroses, by G. M. Gould, M.D., in The American Journal of the Medical Sciences, January, 1890.

In the young the consequences of eye-strain are exceedingly prone to arise or become ingravescent about the time of puberty. There can be little doubt that delayed menstruation may be due directly or indirectly to eye-strain. Another complaint of parents, and one almost always existing in children who are choreic, nervous, and anæmic from ocular irritation, is night-terrors, and restlessness. "Has no more nightmare and crying out in sleep since getting the spectacles," is a frequent report. We have also noted the cessation of nocturnal enuresis, in a few cases under like circumstances. In several cases coming under our observation habitual "car-sickness" has disappeared with the wearing of spectacles. It may be worthy of question if some causal relation may not exist between ocular defect or function and sea-sickness. That chorea may be of ocular origin is now admitted by the best diagnosticians. We have had cases of several years' standing in young girls who had been persistently but unsuccessfully treated with arsenic, the bromides, etc., and in whom all symptoms disappeared almost immediately after the wearing of glasses. In others great excitability, irritability, nervousness, lack of self-control, etc., vanished in the same way. These deleterious effects upon the emotions and disposition may—

and, if long continued, must—have their effect upon the entire character and after-life.

Eye-strain may also influence the life in another way, so that occupation and pursuits in life are unconsciously governed by it. Study and literary labor become so wearisome that the child's mind is influenced against them. The parents are grieved that the taste is slowly but irrevocably turned from intellectual pursuits and the mind directed to physical activities for outlets of its energies. The bearing of such facts cannot be overestimated when we think of our school system; our high-pressure civilization; the suddenness of the strain, since the invention of printing, thrown at once upon the naturally hyperopic eye; the interblending of ocular functions with every act, physical and psychical; and the fact that the enormous load is thrown upon the young organism just at its most plastic and formative period.

A strange fact relating to this whole class of disorders is that the patient may not, and usually does not, feel or exhibit any signs that the eye is the source of all these manifold and seemingly distant results. Sometimes the eye or its appendages may show the results of its own irritational work. In children, styes, blepharitis, and conjunctivitis sometimes exist as the evidences of ametropia, but more frequently the reflexes are to the head, to the digestive system, or to other special or general organs.

We have elsewhere attempted an explanation of this peculiar fact in one class of cases, especially applicable to girls approaching puberty. The essence of the thought is that in the sensitive, emotional, and intellectual of both sexes, but particularly in girls and young women, the irritational eye-strain reflex that normally or physiologically would return eyeward is inhibited, with the result that headaches follow; or it is derouted to other organs that suffer vicariously. The reason of this inhibition, overflow, or deroutation is to be looked for in the enormous importance of the function of vision to the organism in general, and to an exceptional degree in the action of sexual selection, clear and healthy eyes being of the highest importance in influencing sexual choice through beauty. Hence the corollary that the smallest amount of ametropia in girls and women should be corrected, an amount that in men could be safely ignored. According to the fineness or delicacy of nervous organization and the preponderating quality of sexual instincts will eye-strain be disastrous to the general health. All this applies with less force to non-city-bred children or to those who are not pushed by their own ambition, their parents, or the cramming systems falsely called educational of many schools, to an excess of book-work and accommodative effort as harmful to the eyes as it is, both positively and negatively, to the body and brain. However much of the mischief can or will be obviated by relief of the eye-strain,—and certainly much can,—a liberal surplusage will certainly remain.

WOUNDS AND INJURIES.

It should not be forgotten that it is in the treatment of wounds and injuries that the general physician will be called upon for prompt and skilful service, that upon the results of immediate measures will often depend the question of the patient's vision or blindness, and that every case may have possible medico-legal complications. In reference to the latter aspect, the excellent instruction of Arlt[1] is worthy of the study of the careful practitioner.

Foreign Bodies.—On the part of grown people there is usually a clear history and consciousness of the fact when a foreign body is "in the eye," but in children this is not so frequently the case. When there has arisen sudden congestion of the conjunctival capillaries of one eye, we must be on our guard, and at once institute a thorough search for some particle of dust, etc., that may be lodged upon or in the conjunctiva, the cornea, or in the cul-de-sacs. It is necessary to illuminate the cornea from different directions, that a body may be thrown into relief when it is perhaps of the same color as the iris-background, or as the tissue itself. If nothing is found in the exposed parts of the eye or in the inferior cul-de-sac, the upper lid must be everted by grasping the lash, directing the patient to look down, and carefully turning the lid upon itself. It is always a cause of wonder how small a foreign body can produce such discomfort and pain, and the remark illustrates the necessity of scrutinizing every part, of unfolding all wrinkles, and of looking at different angles in order not to miss seeing it. Often a two-inch condensing lens will help to illuminate the object. Before the examination, or at least before attempting removal of a foreign body however superficially located, it is best to use a drop of a two-per-cent. cocaine solution. If the particle be loose upon the conjunctiva or cornea, it may be brushed off with a moist wisp of absorbent cotton; but if embedded, a corneal spud will be necessary for its removal. The instrument should be so held that any spasmodic motion of the eye or head will not wound the part. When irritated, the eye is rolled upward, and hence the instrument should be lightly held. When a cinder or a particle of steel is deeply embedded in the cornea, great care and judgment are required. A common but a reprehensible practice has been to use astringents in eyes from which a foreign body has been removed. It is in no case advisable.

When *a foreign body* has penetrated the globe and can be seen *in the anterior chamber*, it should be removed by incision in such a manner that it can be best reached either by a magnet, if it be steel, or any other instrument, according to the circumstances and the choice of the painstaking surgeon. The patient should be anæsthetized. Removal of *bodies that have entered the vitreous* chamber requires exceptional skill, and is certainly not to be undertaken by any but the most experienced hand. If such a person can see the patient within a day or two, and before the body has become encysted or covered with lymph, it may be removed. But where operation is not at once possible or is for any reason inadvisable, it is best to have the patient lie rigidly still upon the back and with the head high, in the hope that the body will settle in the lower and least-used part of the vitreous chamber and there become encysted and stationary. A few cases have been reported where foreign bodies have entered the lens and remained there without setting up cataract. Of course any attempt to remove such would almost always result in rupture of the capsule, with the consequent cataract.

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[1] Arlt on Injuries of the Eye considered Medico-Legally. Translated by C. S. Turnbull, M.D., Philadelphia, 1878.

BURNS OF THE EYE, either by dry or by moist heat or by chemically-acting substances, are difficult of treatment, and are of such various nature that few hints can be given for guidance in this respect. If the injury be seen at once and if it be caused by acids, then an alkaline lotion—say, a teaspoonful of bicarbonate of sodium to a cupful of water—is indicated, with which the whole eye should be freely irrigated. When the injury has been caused by alkalies,—as, *e.g.*, by lime,—then an acid wash—*e.g.*, vinegar diluted with one-half water—will be very useful. But in either case no time should be lost in waiting for or preparing these things. Water is almost always at hand, and with this the eye should be cleansed and thoroughly washed. The greatest danger in all cases of burns is that the conjunctival surfaces of the opposite lids and globe may have become denuded and in healing they may grow together in an incurable symblepharon. The most promising way of preventing this is to keep the eye bathed and the sulci filled with castor oil, and to break up the adhesions that may begin forming by frequently passing a probe or spatula between the surfaces. If the pain become intense, cocaine must not be used, but anodynes and cold compresses, with paralysis of the accommodation, will be found most efficacious.

INJURIES OF THE EYELIDS require more care and watchfulness than are usually given them, owing to the fact that notches and subsequent cicatrization are very apt to distort and evert the lid, with consequent lachrymal troubles, or to invert it, with all the sequelæ, pannus, etc., that follow upon entropion or inversion or malposition of the cilia. In a general way they are to be treated as an injury elsewhere would be, but with especial reference to the peculiar function of the tissues and the dangers we have adverted to. Gaping wounds should be most carefully and accurately sutured in place, instead of using court-plaster. Owing to the extensive areolar tissue, extravasation of blood is common. If the œdema be great, fracture of the orbital walls or border should be reckoned among the possibilities.

RUPTURE OF THE GLOBE is rare, and is usually the result of contusion with a blunt body. Owing to the fact that the nasal side of the globe is more protected than the temporal, the blow is more commonly upon the latter side, but by reason of the transmitted force, or "contrecoup," the rupture is more frequently in the choroid and upon the nasal side.

Permanent recovery from a rupture through the ciliary region will never take place. The subsequent cicatrization will induce cyclitis and such irritation that enucleation must follow sooner or later. So that the rule is without exception that, where the ciliary body has been certainly broken or cut through, immediate enucleation is the better procedure. Even if the globe be saved, there is apt to be little vision, and the danger of persistent cicatricial irritability and sympathetic inflammation is great. In such cases, also, the lens is frequently dislocated and the capsule ruptured, so that cataract is a common complication. If the lens be in the anterior chamber, it should be extracted at once. If the lips of a purely sclerotic cut or rupture show a tendency to gape, a few delicate stitches may be taken through the external edges of the lips of the wound and the conjunctival tissues. When the cornea is extensively cut or ruptured and a large part of the iris protrudes, this should be drawn out as far as possible and excised. This may even have to be repeated one or more times, in order to avoid extensive cicatricial inclusion of the iris in the lips of the wound. According to the location of the wound, the use of eserine or of atropine may be advisable to aid in the retraction of the iris. The bandage should be firm, but not tight.

PENETRATING WOUNDS AND SYMPATHETIC INFLAMMATION.—Many remarkable cases have been reported of the retention of large foreign bodies that have penetrated the orbit. In such cases, therefore, one must be on his guard not to overlook them, by inquiries as to the exact nature of the object causing the traumatism, by careful examination of the wound, etc. In perforating wounds of the cornea there will probably be hernia of the iris. If this be extensive and cannot be reduced, the protruding portions must be excised as deeply within the lips of the wound as possible, and the eye atropinized and firmly bandaged. If the hernia recur, the same operation must be repeated, since extensive inclusion of the iris in the wound is to be avoided at all hazards, a fact that necessitates a subsequent iridectomy to release it and to keep the eye from constant pain, iritis, and irritation. But if the hernia be of only a small portion of the iris, it is best to try the effect of atropine, rest, etc., rather than mutilate the iris, and also because if only slight adhesion take place a subsequent iridodialysis or iridectomy may remedy the trouble. *When the ciliary body has been cut through*, there is the same danger as that to which we have adverted in the preceding paragraph, and enucleation must follow.

Whether to enucleate at once or not in cases of doubtful implication of the ciliary body is often the most perplexing of questions. If not done, and fatal sympathetic inflammation ensue, it is of course the saddest of results. *Therefore it is better to err on the side of safety and enucleate*, though the condition of the injured eye promise some slight chance of recovery. The existing complications, the extent of the injury, the condition of the other eye, the general health of the patient, etc., must all be considered. At all events, and in all cases, careful search must be made for possibly retained foreign bodies, thorough cleansing and antiseptic treatment instituted, perfect quiet enjoined, etc.

Sympathetic trouble may not only follow traumatism, but may also result from any destructive disease of the eye, especially of the ciliary body. In such cases, that primarily injured is called the excitor, the other the sympathizer. Sometimes the excitor is a shrunken globe that in consequence of extensive choroidal or ciliary disease has become affected with *phthisis bulbi* or progressive atrophy and shrinking. It is customary to divide sympathetic trouble into two groups,—sympathetic irritation and sympathetic inflammation. The method by which the sound eye is affected by the excitor is a matter not yet wholly made manifest. Clinically, the period of irritation—consisting (in the sympathizer) in limitation of the accommodation, sharp pains, photophobia, lachrymation, congestion, etc.—is of extreme importance, as heralding the probable approach of a true inflammation, an irido-cyclitis. In either stage the earliest possible enucleation of the excitor is imperative.

SURGICAL OPERATIONS UPON THE EYE.

In all operations upon the eyes of children, it is better to anæsthetize the patient. The operation can frequently be done during the primary effect of ether or chloroform, when properly given. The stomach should be empty, food having been forbidden for several hours prior to the operation. Subsequent to the operation, we should see that gastric pain does not arise, causing the child to cry, and thus endangering the success of the operation. Paregoric is probably the best anodyne in such cases. A small mustard plaster over the epigastrium, and other measures, may be ordered, according to the circumstances or the preferences of the physician. It is generally useless to attempt bandaging the eyes in the case of an infant, on account of the absence of hair, the smoothness of the scalp, etc. Where a bandage is necessary, it may be retained in place when placed over a tight-fitting cap or hood. A dark room is usually preferable to bandages.

Operations to Relieve Obstructions of the Lachrymal Excretory Apparatus.—Epiphora may arise from an excessive secretion rather than from an impeded outflow, and doubtless operations upon the canaliculi and duct have sometimes been carried out where more careful search would have shown some reflex or other source of local irritation to account for the excess of tears in the eye. Sometimes, also, the canaliculus and duct are normally patent, but the puncta are either slightly everted, or closed by a foreign body, or stenotic from inflammation or cicatricial contraction. In these cases it is unnecessary to slit the canaliculus or to probe the duct, and where there is not positive evidence of nasal disease or dacryocystitis, it is better to begin by opening or extending the opening of the puncta. When there is not perfect apposition of the puncta to the globe, a simple proceeding consists in inserting the sharp point of the scissors into the opening and dividing the conjunctival surface vertically downward for one or two millimetres. Where this is insufficient, or where it is necessary, on account of purulent disease of the sac, to divide the canal lengthwise, a peculiar blade is used—Weber's canaliculus knife—with a tiny bulbous point which guides the advancing knife along the tube. The physician, standing over the anæsthetized patient, uses one hand to withdraw the lid from the globe and make it tense, steady the head, etc. The bulb having entered the puncta, the knife is held horizontally and is advanced along the canal until the bulbous point reaches the lachrymal sac; then, while the lid is held by the thumb of the other hand firmly to the temporal side and kept tense, the knife is raised to a vertical position and the canaliculus is divided up to the mouth of the sac. Constriction of the nasal duct may be incised by now rotating the cutting edge of the blade anteriorly and pushing the knife downward and slightly forward into the cavity of the duct. Sometimes the duct shows a persistent tendency to constriction and closure, though this is seldom seen in children. If so, a probe made for the purpose may be used till the duct remains patent. Care must be used that the probe do not form a false passage, and also that it be inserted properly. Acute abscess of the lachrymal sac should be immediately and freely opened, and after this watchfulness exercised that a lachrymal fistula do not form.

MEIBOMIAN CYST.—Upon everting the lid we sometimes find the cause of conjunctival congestion in a localized purplish discoloration and swelling of the subconjunctival tissues. The duct of a Meibomian gland having become occluded, its retained secretion causes distention and inflammation of the part. It should be freely incised upon the conjunctival surface, and the contents removed by scoop and scraped with a sharp spoon.

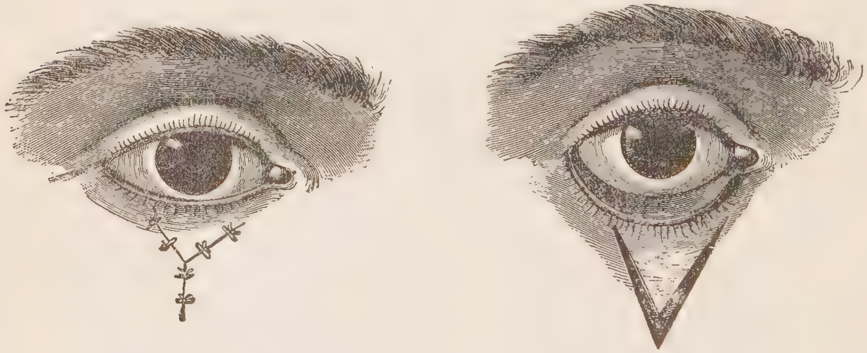
BLEPHAROPLASTY.—Plastic operations upon the lids are for the purpose of correcting entropion, ectropion, cicatrices, or injuries, and for the rare congenital ptosis from lack of development of the muscular fibres. When in entropion only a few hairs are incurved, they are best destroyed by electrolysis, or given a different direction by illaqueation, which consists in drawing each lash through a needle-hole by a lasso or loop of ligature. The tissue containing the hair-bulbs of the lashes may also be excised from the inner margin of the lid after dissecting away the conjunctiva.

If a large extent of the lid is turned in, more radical measures are required. The most common mode of correcting this in the lower lid is by removing a strip of skin parallel with the lid-edge, the width and depth of the portion of excised tissue being proportional to the effect desired. If of the upper lid, the choice lies between several operations.

The Jaesche-Arlt operation consists in splitting the lid along the intermarginal edge three or four millimetres deep. The anterior portion containing the cilia is made free except at the ends by an incision through the skin of the lid about three millimetres from the edge of the lid and parallel with the same. Above this a fold of skin is removed entirely, corresponding in size with the effect desired, and the ribbon of tissue containing the cilia is brought up and fastened in the space of the excised tissue by sutures, thus bringing the edge of the lid with its cilia into a normal position. Von Graefe's operation is similar, except that vertical incisions one-third of an inch long are also made from the two extremities of the ribbon of cilia-tissue, and the ends of the same near the canthi are better elevated by sutures into the outer lips of the vertical incisions. Hotz excises a layer of muscular fibres over the tarsus three or four millimetres in breadth at the upper border of the tarsus of the upper lid or the lower border of the tarsus of the lower lid, and sutures are passed through the upper edge of the tarsus. In Dianoux's operation two parallel bands, one containing the cilia, the one above it simply the skin, each about three millimetres broad, are made by splitting the lid and by an incision parallel to the lid-edge extending to the cartilage. The upper is drawn under and below the lower, and its lower edge sutured to the conjunctival edge of the lid; the ribbon of cilia-tissue is thus raised from the border of the lid, and its upper margin is sutured to the lower of the superior incision of the lid. Streatfeild and Snellen remove a V-shaped wedge of skin, muscle, and tarsus parallel with the lid-edge; and Von Burow's operation consists in a conjunctival incision of the cartilage three millimetres from the border of the lid, causing gaping of the wound and moderate eversion of the free edge.

Wharton Jones's operation for ECTROPION is illustrated in the annexed cuts (Figs. 10, 11). A V-shaped incision is made with the limbs extending toward the canthi. The enclosed triangular flap of skin is then dissected up and reduced by excision to the desired extent, and the lower part of the incision brought together by sutures, so that the final appearance of the wound is that of the letter Y. The contraction thus produced by the cicatrix brings the lid into apposition with the globe. Argyll-Robertson's operation consists in inserting into the lower cul-de-sac a piece of sheet-lead about the size of the lid and conforming to the parts in shape, against which the lid is brought into apposition and the normal position by tension upon ligatures passed from without through the free edge of the lid, thence through the bottom of the cul-de-sac out upon the cheek below. Bits of rubber tubing

FIG. 10. FIG. 11.



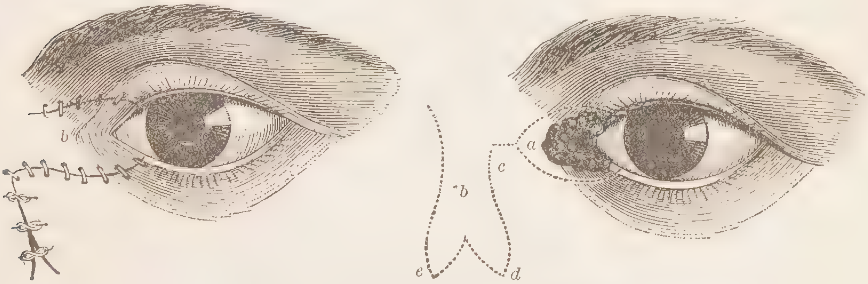
Wharton Jones's operation for ectropion.

are used under the external loops of the ligature, to prevent their cutting through the integument, before traction is made. If the lid be thickened by an overgrowth of subconjunctival tissue, a V-shaped mass may be excised the length of the everted lid, prior to the ligation.

It may be said that operations of this kind are rare in children, though sometimes necessary or possible in those approaching puberty. The cartilage in children is not fully developed, and this fact must be considered when operations are done on the lids.

Traumatic or cicatricial ectropion can only be cured by transplantation of a flap with pedicle from the adjacent forehead, temple, or cheek, or from the arm, or by transplantation without pedicle from the arm. The unsightliness of the facial wound thus produced gradually grows less in time, and the flap from the face usually gives the best result. It must be larger than the freshened or denuded space it is to fill. An ingenious method of repairing a loss of tissue at the canthus is shown in the annexed cuts (Figs. 12, 13). The space *a* being denuded and freshened, the flap *b* is dissected up, twisted upon the pedicle *c*, and the points *d* and *e* are sutured to the lower and upper lid so as to form the normally-shaped angle between.

Fig. 12. Fig. 13.



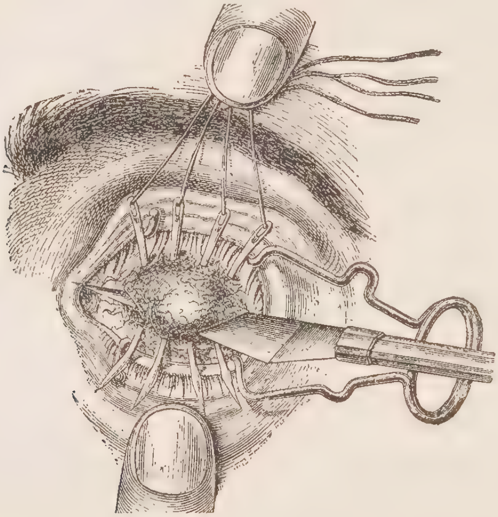
Hasner d'Artha's blepharoplastic operation.

Peritomy, the object of which is to relieve pannus by a dam of cicatricial tissue about the cornea, is performed by dissecting off a ribbon of conjunctiva one or two millimetres wide close to and encircling the cornea. It is rarely required in children.

Dense corneal staphyloma, or bulging of the weakened cornea from the intraocular pressure, may be removed in several ways, one of which is shown in the illustration following (Fig. 14). The main body of the staphyloma having been removed by two incisions through the dotted lines, the sutures are at once tightened and a movable stump thus obtained. Another method is to run a strong continuous ligature through the healthy loosened and undermined conjunctival tissue and sclera surrounding the staphyloma, so that, after excision of the latter, traction upon the ligature acts like a "puckering-string" and at once closes the conjunctiva over the space of the excised tissue. Mules, of England, advises clearing out the entire contents of the globe and filling the cavity with a glass ball.

In cases of dense corneal leucoma the unsightliness may be greatly improved by TATTOOING THE CORNEA, by which a close approach to the appearance of a pupil may be made.

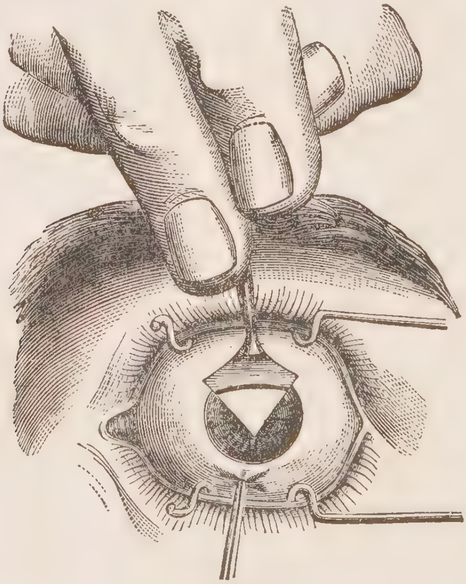
FIG. 14.



Operation for corneal staphyloma.

The incision for IRIDECTOMY, or *to make an artificial pupil,* is shown in Fig. 15. The knife used is a triangular keratome, and is entered at the corneal limbus or border from above and to a depth requisite for the proposed operation. The iris is then seized at the pupillary border by the

FIG. 15.

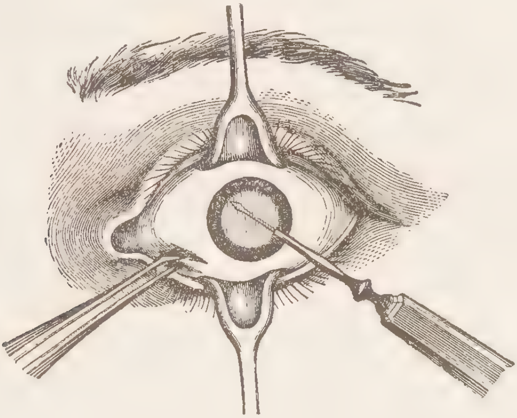


Iridectomy.

iris-forceps, withdrawn, and a piece, of the size and shape desired, excised by one stroke of the scissors. Atropine, to pull the iris ends out of the wound, is periodically instilled, and the eye kept bandaged for two or three days. This operation is useful in anterior or posterior synechia, to make an artificial pupil in case of partial opacity of the cornea, and also as a therapeutic measure in glaucomatous tension caused by adherent leucomata.

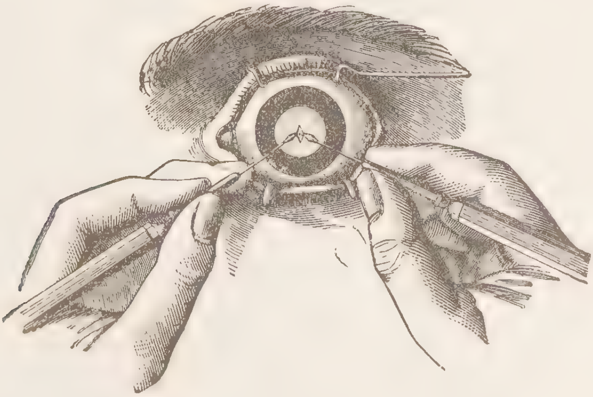
Discission, or Needling, is an operation frequently required in congenital cataract of the young. The adjoining cut (Fig. 16) gives a good idea of the proceeding. The point of the stop discission-needle is passed into the anterior chamber from the side of the cornea and past the dilated pupil until it pierces the anterior capsule and the substance of the lens; by movement of the point the capsule must be freely ruptured and the lens broken up to the desired extent so that the aqueous shall gain admission to it, causing it to swell and become opaque and finally to break down and be absorbed, leaving an unimpeded path for the light. In recurrent or cap-

Fig. 16.



Discission, or needling.

Fig. 17.



Discission, or needling, with two needles.

sular cataract after an extraction of the lens the same operation is made use of to break through and depress out of the way the central portion of the opaque capsule. Two needles are commonly used for this purpose, as shown in Fig. 17, whereby an opening is more certainly and accurately made by rupture and displacement outward of the capsule by the two needles meeting first at the centre of the pupillary space. No reaction usually follows the operation.

TENOTOMY IN CASE OF STRABISMUS.—Graefe's subconjunctival operation is the proper one in these cases. The opening is made at a point tangent to the lower edge of the cornea. The conjunctiva and Tenon's capsule are vertically cut across over the lower edge of insertion of the muscle-tendon, and the opening widened sufficiently for operative purposes. The strabismus-hook is then inserted beneath the tendon, which is drawn forward, and, by inserting the blades of the scissors (blunt points and curved on the flat) between the hook and the globe, the tendon is divided close to the globe. If the most extreme effect possible is desired, the lateral and posterior attachments of the capsule of Tenon may be divided. A suture may be used to close the conjunctival opening and drawn more or less tight to reduce the effect of the operation. Put atropine into the fellow-eye, or use correcting spectacles at once.

ADVANCEMENT.—When the effect of a tenotomy is insufficient to give the desired change in the visual axis, it must be repeated when the opposing muscle is advanced. To do this, a ligature passed about the tendon at its insertion firmly secures the muscle by the loose ends of the ligature. The tendon is then divided close to the sclerotic and its lateral attachments are freely divided. Another ligature is now passed through the muscle behind the one grasping the cut end, which last is excised with as much of the tendon itself as is desirable to give the effect of increased power over the globe. The free ends of the ligature passed through the muscle are now passed through the pericorneal conjunctival and subconjunctival tissue above and below, and traction and knotting secure the divided tendon in its new position till union has taken place. It is generally considered best to make about one or two millimetres of over-effect at first. If the strength of the sutures is doubtful, a stay or anchor suture may be made from the skin of the inner canthus.

Prince's "pulley" operation is perhaps preferable. A ligature is passed vertically in and out several times through the conjunctiva one millimetre from the cornea in the line of the muscle. An opening is then made over the tendon-insertion, and, without dividing it, the muscle is loosened from its attachments behind its insertion, so that a second ligature may be passed through the conjunctiva and muscle at this place. The tendon is then cut at a point removed from the insertion proportionally to the effect desired. The first or anchor suture near the cornea is now made into a loop by tying, one end of the suture through the muscle having first been passed through or under the loop to be made. It is obvious that traction upon the ends of the ligature through the muscle acts now like a pulley to draw the cut end of the muscle forward. The amount of advance may at the time or subsequently be regulated at pleasure.

If vomiting is threatened during an operation for tenotomy or advancement, the speculum should be quickly removed and firm compression made by the hand protected by cotton or a bandage. This is done in order to avoid subconjunctival hemorrhage and the rare but possible and disastrous result of a hemorrhage into Tenon's space, whereby mobility of the eye is destroyed and other complications may ensue from the presence of an extensive clot.

ENUCLEATION OF THE EYEBALL.—The patient is anæsthetized, as in every case of manipulation of the eyes of children, the eyelids are held apart by a speculum, and the globe is held by the fixation-forceps. The conjunctiva is separated from the corneal attachment as close to the cornea as possible, with a pair of curved blunt scissors. Tenon's capsule is then opened over the insertion of the external rectus, which is now caught with the strabismus-hook and divided close to the globe. Each of the muscles is in turn then severed in the same way until the globe is held only by the optic nerve. Passing the scissors on the nasal side between the capsule of Tenon and the globe, and pressing the globe upward, the nerve is divided close to the globe in ordinary cases, but as far back as possible in case the enucleation is made during or following malignant disease. Antiseptic sponges or a pledget of sublimated cotton may be used to arrest the slight hemorrhage. After thoroughly cleansing the socket, the lashes should be softened by vaseline, to keep them from becoming matted together. A firm bandage should be applied for twelve hours, and the socket cleansed and dressed for several days until healing is well advanced.

ARTIFICIAL EYES can be worn in two or three weeks after enucleation. In all cases the eye should be too small rather than too tight or prominent. It should be removed every night, kept in an antiseptic liquid overnight, and anointed with vaseline prior to insertion. When it becomes rough from long wear, it should be repolished or another substituted for it. The socket should be cleansed with an antiseptic lotion once a day. It may be added that the art of choosing and fitting artificial eyes requires exceptional skill, judgment, and experience. The stock to choose from must be very large, and the conformation of the socket studied in order to give comfort to the patient and an approximation to the normal mobility and appearance, which is sure to follow a properly-removed globe.

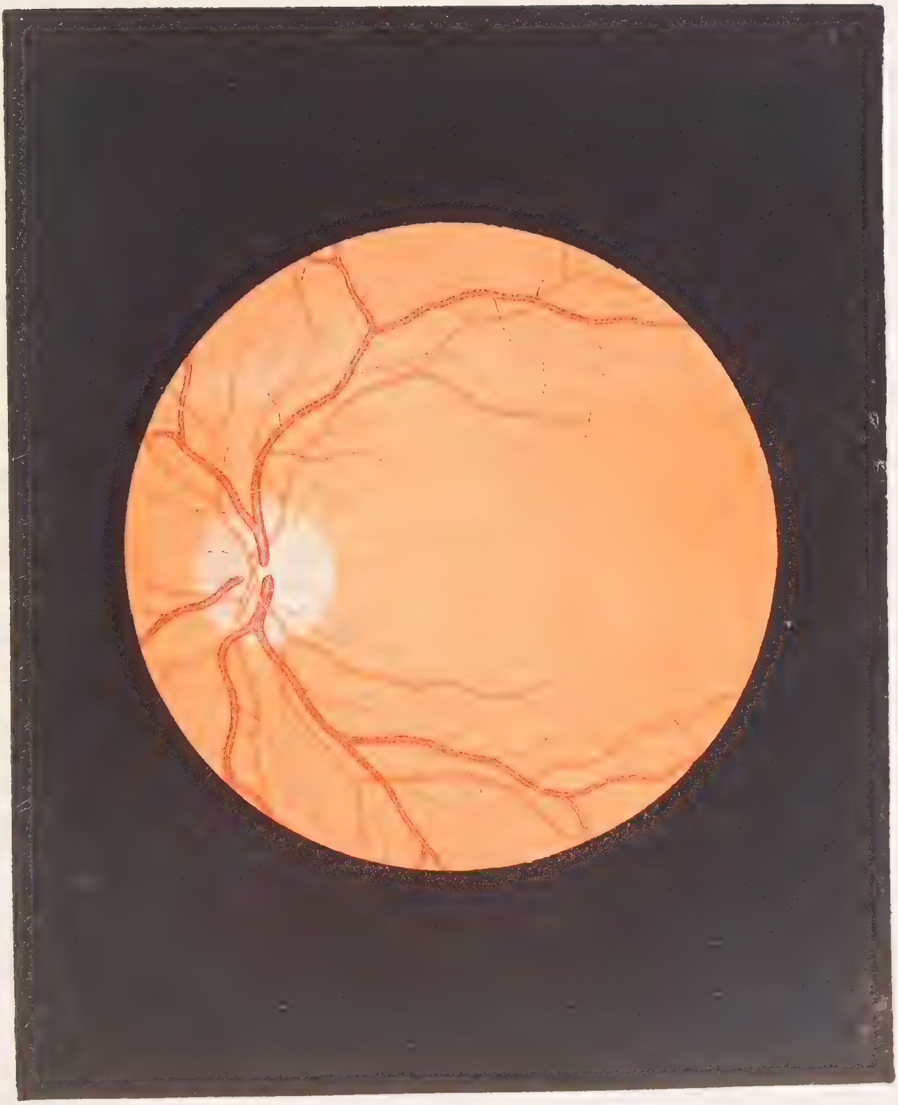
OPHTHALMOSCOPY: LOCAL DISEASES AND FUNCTIONAL DISORDERS OF THE CHOROID, OPTIC NERVE, AND RETINA.

By CHARLES A. OLIVER, M.D.

Hughlings Jackson once said, "It is, I submit, imperative in all cases of severe cerebral disease, at all events in cases of an acute kind, to examine the eye with the ophthalmoscope, whether the patient complains of defective sight or not:" this assertion he has since made much stronger by adding the words "even if he affirms that he can see well, and if he reads small type readily." To this we may add that proper and careful ophthalmoscopic examination should be made in all cases where organic disease of any related kind is suspected. With the present inadequacy of knowledge of the exact relationship between certain visible intraocular changes, and the many varieties of supposed causative systemic disturbance, it is, of course, impossible to give any typical description of the changes that may occur in the two most important ocular registers (the retina and the optic nerve), although in any definite disorder this difficulty will undoubtedly be greatly removed by strict, frequent, and painstaking observance of the method.

Here, idiosyncrasies, like those in any other part of the organism, are so numerous, and congenital abnormalities, though slight and often unnoticed by the incompetent observer, are so frequent, that mistakes must for a long time in the future be expected, until thorough systematic exercise with instruments of greater power has rendered the method a certainty.

With the present plan of study by means of the ophthalmoscope, a background comprising nerve-tissue, connective material, and a portion of a peripheral vascular circle is stretched before us. Its various parts are all arranged in a definite way. The intraocular bulbar ending of the second nerve—the so-called optic disk—is most frequently recognized as a small oval expanse of lighter color than the rest of the ground situated to the inner side of the ophthalmoscopic field. From some part of its surface can be seen an entering vessel dividing into numerous stems, which by the color and reflex of its contained blood, its comparatively smaller size, and



Normal Eye-Ground of an Individual with Light-Brown Hair. (Jaeger, Beiträge zur Pathologie des Auges, Taf. I.)

its lesser tortuosity, can be readily differentiated as an artery from the branches and main trunk of the outgoing veins: one is the central retinal artery, and the other is its corresponding vein.

Systematic and careful study will soon render an observer able to note the many differences in the appearance of these structures, and to draw proper conclusions as to whether there are departures from what is understood to be normal. First studying any change indicative of abnormality and of local disease alone, we will finally consider those alterations which ofttimes indicate the existence of systemic disease.

The instrument should be of the best pattern, and the observer should be sufficiently taught to note understandingly what he sees. Careful record should be made of the details of the fundus, no matter whether they are pathologically interesting or not. Every detail should be written, so that if there be any future change the comparison will become at once apparent.[1]

The classification which follows has been made not only because there is at least some anatomical correlation between the special form of local disease and the gross systemic lesion, but also for convenience of handling the material whilst in search of related changes. Of course, in the present chaotic state of etiology, it would be impossible to assert dogmatically the position of any symptomatic form of disease of the choroid, optic nerve, and retina, as having origin in any certain structure; but wherever the position is fairly definite, or the symptomatology points more especially to the situation of the general disease, the changed condition of these portions of the ocular apparatus will be noted in the text.

I. LOCAL DISEASES.

MALFORMATIONS AND ABNORMALITIES.

Malformation in the shape of the disk, irregularities of size independent of refractive error, changes in the apparent surface not the result of pathological condition, and alterations in the character of the physiological excavation, have all been mentioned by various authors.

Both Fuchs[2] and Loring[3] have seen the disk covered by a thin opaque membrane, which concealed the entrance and exit of the retinal vessels: this anomalous condition of the head of the nerve does not seem to interfere with vision in any way, on account of its situation in the blind spot of Mariotte in the visual field. The writer has recollection of a very interesting example in the right eye of a young girl treated in the out-door patient department of Dr. William F. Norris's service at Wills Eye Hospital. Vision and accommodation were both normal after correction of an existent ametropia, and the visual fields were apparently not reduced or disturbed in any way.

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[1] In all ophthalmoscopic examinations, the writer has endeavored to make it an inflexible rule to obtain in association as many of the ophthalmic conditions as possible; and in numerous instances he has been rewarded by the discovery of an ocular symptom that has proved of immense collateral advantage.

[2] Archiv für Ophthalmologie, xxviii. 1, 139.

[3] Text-Book of Ophthalmoscopy, p. 98.

One of the most frequent variations in the *choroidal* structure is that of the *pigment.* Instead of being ideally perfect in distribution, as is so frequently seen in diagrammatic sections of the human eye, it often forms areas of aggregation, etc., as, for instance, around the edges of the optic disk. Here, as we so frequently note, the masses assume definite forms, such as rings, loops, and crescents; these, as is well known, are by far the most common upon the lateral borders of the nerve-head, especially to the outer side. If these groupings are not more than ordinarily pronounced, the case cannot be considered atypical. Again, in many instances, where there are no special appearances of choroidal inflammation, the heavy black interspaces between the choroidal vessels are plainly visible in the peripheral portions of the fundus. Moreover, isolated aggregations of pigment may from time to time appear in cases that do not present any assignable cause or reason for such occurrences, except the possibility of low grades of choroiditis during the child's intra-uterine existence. Loring[1] cites a remarkable instance, and Jaeger[2] gives an almost similar case, except that in his case the massings of pigment were situated in the superior temporal portions of the fundus.

Just as in the lower forms of animal life, where we have individual instances of great deficiency of general pigmentation, so here in the uveal tract, the choroid is made to suffer. Although grading from the most pronounced types of African negro to the lightest Caucasian the amount of pigment is constantly decreased, yet in both of these extreme types of man we not unfrequently have exceptional examples of great decrease of the amount of pigment; these cases being classed under the generalizing term *albinism.* Here the fundus is almost brilliant in appearance, the underlying vessels of the choroid being plainly visible. Jaeger (*loc. cit.*) gives us a beautiful chromo-lithograph of this condition. Most frequently the region of the macula suffers the least, there generally being a fair amount of pigment situated in this portion of the fundus.

Congenital deposits of pigment upon the disk-surface, from the very minute quantities so ordinarily seen, up to the dense massings depicted as such by Jaeger,[3] should not be confounded with pathological change. Similar pigmentations as seen by Liebreich[4] are sometimes met with in varying amounts, from the narrow concentric and line-like aggregations bordering the edge of the nerve, down to almost entire deficiency, as in albinism, where the underlying choroidal structures are plainly visible.

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[1] Text-Book of Ophthalmoscopy, Part I., 1886, Plate II. Fig. 5.

[2] Beiträge zur Pathologie des Auges, 1856. [3] Ibid.

[4] Atlas of Ophthalmoscopy, Plate XII. Fig. 3.

Abnormal tint of the disk-substance has been mentioned, as, for instance, in Case VI. of Jaeger's collection, which if studied by artificial light (the condition under which it was sketched) gives a distinct bluish cast.

Jaeger[1] and Mauthner[2] have described what they term an *abnormal transparency of the nerve-fibres*, giving the disk an appearance of greater depth than usual: this, which might be supposed to be due to transparency of the transverse bands and vessels of the lamina, is explained by Loring[3] on the supposition that the lamina is really situated more posteriorly. Loring says that he has never seen this condition, but has recognized what appeared to be an abnormal transparency of the nerve, "in which minute vessels appeared to be embedded as if in some gelatinous substance." This he attributed "to a lack, or almost entire want, of connective-tissue elements, and to a paucity of the smaller vessels." If carefully searched for, this condition will be found to be more frequent in children than may be supposed. In not a few instances, small irregular areas can be distinctly discerned, especially to the temporal side of the nerve-head, in which the nerve-substance appears normally more transparent than in the adjacent parts; these in most cases being bilateral, and seemingly unassociated with any apparent pathological change.

Cases of *retained nerve-sheaths* are sometimes seen, where instead of the opacities of the medullary sheaths terminating at the cribriform plate of the optic nerve, they either gradually lessen and cease at different distances out in the retina, giving somewhat the same appearance that is normally seen in the fundus of the eye of the rabbit, or the fibres seem to become transparent and opaque in turn, leaving islets of whitish striated massings, as described by Beckmann[4] and Von Recklinghausen,[5] in which special cases the condition was substantiated by post-mortem examination made by Virchow. Schmidt[6] reports a similar case confirmed by autopsy. Jaeger[7] depicts an instance in an otherwise healthy eye. Liebreich[8] gives two instances, the first of which is remarkable not only for its immense area and thickness, but also as exhibiting a small isolated spot far removed from the general mass. Juler[9] gives two unusually well marked examples. In both of his cases the medullation seemingly begins at the edge of the disk, and is curiously divided into four comet-like processes extending along the lines of the larger retinal vessels some distance into the periphery of the fundus.

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[1] Einstellungen des dioptrischen Apparates, S. 31.

[2] Lehrbuch der Ophthalmoscopie, S. 258.

[3] Text-Book of Ophthalmoscopy, p. 100.

[4] Archiv f. Path. Anat., xiii. 97.

[5] Ibid., xiii. 357.

[6] Klinische Monatsblätter für Augenheilkunde, 1874, S. 186

[7] Hand-Atlas, Taf. VI. Fig. 36.

[8] Atlas of Ophthalmoscopy, 1870, Plate XII. Figs. 1 and 2.

[9] A Hand-Book of Ophthalmic Science and Practice, 1884 (Amer. edit.), p. 201.

Noyes[1] also exhibits a drawing of the same condition. The accompanying phototype from a sketch which was kindly made for the writer by Dr. B. Alexander Randall shows one of the most marked examples seen in the writer's collection. The boy, aged nine years, applied for the correction of an error of refraction (H + Ah), never having had any subjective symptoms of the condition.

The diagnosis, which is quite easy after having once encountered a case, is based upon the peculiar glistening character of the whitish-yellow striation (slightly greenish at times) and the fringe-like border of the distal extremities. As a rule, the opacity seems to prefer association with the course of the retinal vessels, which it more or less conceals. In the annular variety (as in the sketch) the centre of the nerve, as a rule, is visible, and presents, most probably by surrounding contrast, a very peculiar green tint. The calibre of retinal vessels seems in no way impaired, and the color of the contained blood appears normal. The uninvolved portions of the fundus do not show any indications of pathological process, and, curiously, the macular region is seldom involved. Rarely and only in the extreme cases is defective vision complained of; this, when present, in the opinion of Mauthner,[2] appearing to be partially the result of high hypermetropia with so-called "amblyopia ex anopsia." Be this as it may, it is positive that if careful examination of the vessel-fields be made in all such cases, corresponding defects will be found, whilst the unaffected areas of the ground seem to project normal color-differentiation. In most of the cases seen by the writer the anomaly has been unilateral, and in none of the few instances searched for has he been able to find a similar condition in the parents. Of course, treatment is out of the question.

As it is well known that the greatest amount of *optic nerve massings* is found at the inner upper and lower portions of the disk, thus practically giving greater elevation to the nerve-substance in these situations, it may be interesting to note an instance in a boy described by Mauthner,[3] in which the fibres appeared to be caught into two bundles, one above and the other below, holding the entire retinal circulation within their grasp; this appearing in an eye where astigmatism of sufficient amount to produce a similar picture did not exist.

Coloboma of the choroid, which is generally situated inferiorly, is usually associated with colobomata of the iris and lens. The defect appears as a large ectasia or series of depressions, separated from the non-colobomatous portion of the eye-ground by an irregular black line of pigmentation. If carefully looked for, the retina appears as a thin filmy haze stretched over the entire surface. If the coloboma be typical, it gives the effect of a large, glittering, whitish, and irregularly concave piece of enamel, containing in places a fine radiating net-work; the whole being covered by a delicate

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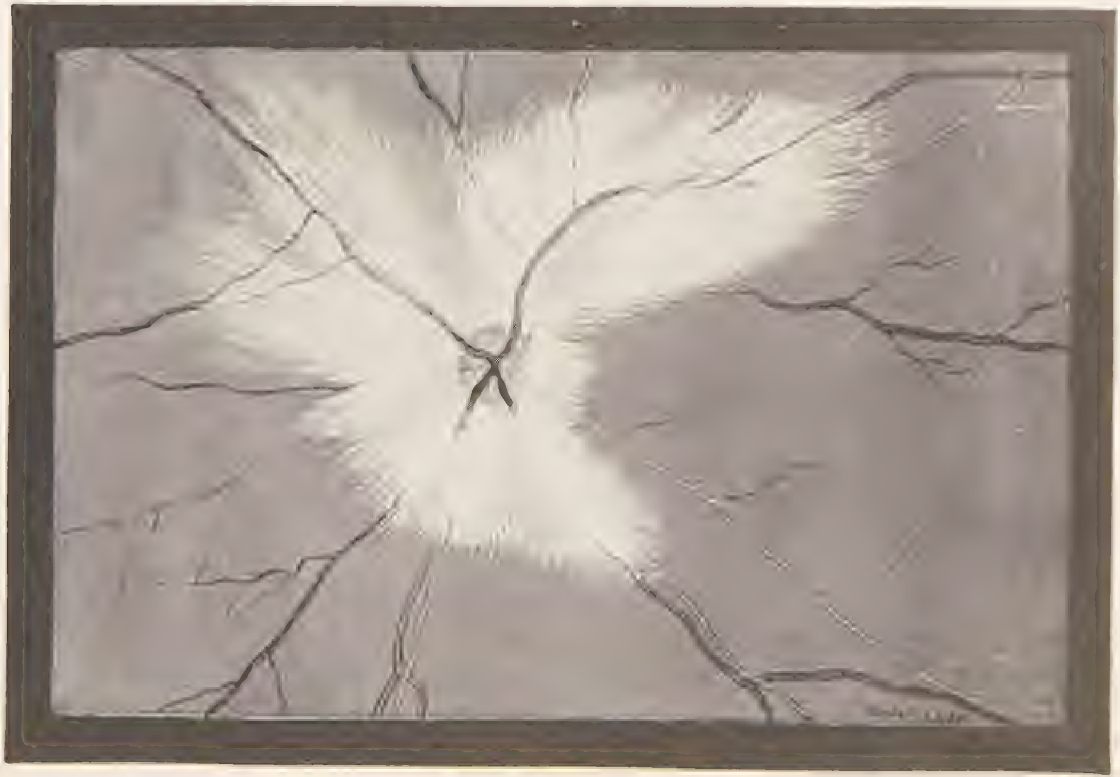
[1] A Text-Book on Diseases of the Eye, 1890, Plate III. Fig. 2.

[2] Lehrbuch der Ophthalmoscopie, 1868, S. 266.

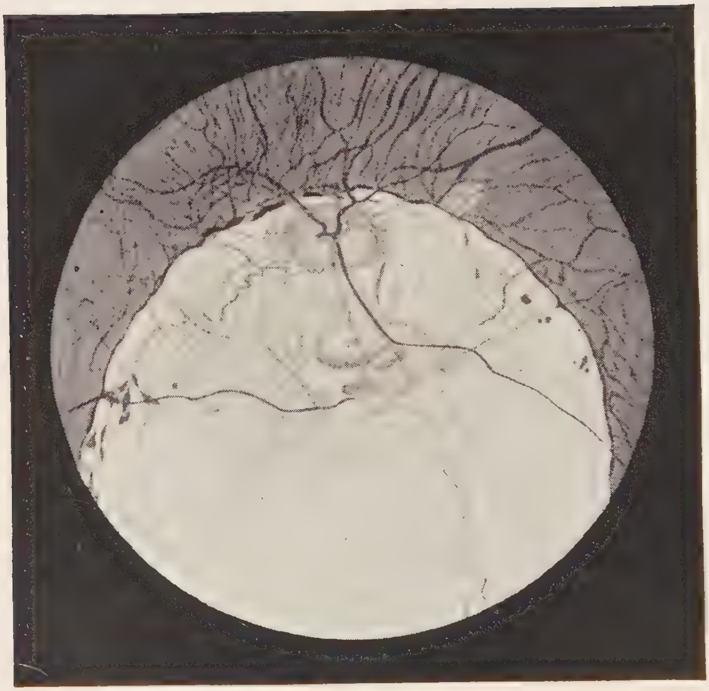
[3] Virchow's Archiv, x. 267.

PHOTO. I.



Retinal Nerve-Striations (original)

PHOTO. II.



COLOBOMA OF THE CHOROID. (Jaeger, Beiträge zur Pathologie des Auges, Plate XLVI.)

grayish veil carrying a series of interlacing, larger, bright- and dark-red lines. Most frequently the ectatic portion is divided into a series of irregular depressions into which the retinal vessels may dip. At times the nerve-head itself, especially if it be partially or wholly included in the colobomatous area, appears of a peculiar greenish-gray tint (gas-light), with very little capillarity in its substance. In such cases the retinal circulation is impaired, and the vessels themselves are small and irregular. Again, many small scleral shoots from the posterior ciliaries may appear at points, showing themselves, as Loring says,[1] as a fine delicate net-work, of which isolated branches here and there can be traced directly into the sclera or followed over the white surface of the coloboma into the normal district of the choroid. Should the choroid show signs of disturbance, or should the depressed areas contain much pigment-massing, it is most probable that the case is not one of true defect, but is the result of inflammation during fœtal life. Vision is always affected in a position corresponding with the area of deficiency. Benson[2] adds a case of uniocular coloboma of the choroid with colobomata of the iris and lens in a twelve-year-old boy. The fundus in the colobomatous area was four diopters lower than the sound portion, which itself was highly myopic, whilst the normal background of the fellow-eye could be seen with a convex lens. The accompanying reproduction of the ordinary condition seen is from Jaeger (Plate XLVI.).

Colobomata of the sheaths of the optic nerve, described by Liebreich,[3] Makrokoki,[4] Nieden,[5] Randall,[6] and others; *colobomata situated in the macular region,* as seen by Burnett and Reich,[7] and the questionable one of Loring;[8] the curious coloboma to the nasal side of the eye-ground, associated with acquired changes, seen by De Schweinitz and Randall,[9] and one very interesting, still unpublished case by the writer, where there is a small ectasia utterly devoid of any inflammatory products, situated just up and in from the left disk of a young girl, are all extremely rare. The pictures they present are so typical of malformation that when carefully studied it is impossible to confound them with apparently similar conditions dependent upon traumatism or disease. Two additional cases involving the macular region are described by Silex.[10] Here, however, as the author says, the changes are most probably dependent upon intra-uterine choroiditis, and are not caused by faulty development.

Idiosyncrasies in vascular distribution to the optic nerve and retina are so pronounced and frequent that it is sometimes questionable whether to legitimately class them as abnormal or not. Curious twistings, redoublings, and intertwinings of the retinal vessels, especially near or on the disk, are of frequent notice. An excellent example of this occurring in the inferior artery of the retina is described by Little[1] under the title of "A Case of Persistent Hyaloid Artery." Czermak[2] reports an almost similar instance. Under the title of "A Case of Tortuosity of Retinal Vessels, in Connection with Hypermetropia," Mackenzie[3] gives a sketch of curiously twisted and redoubled retinal vessels which he saw in the left eye of a twenty-year-old girl.

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[1] A Text-Book of Ophthalmoscopy, Part I., 1886, p. 93.

[2] Transactions of the Ophthalmological Society of the United Kingdom, 1884, p. 357.

[3] Atlas d'Ophtalmoscopie, Plate VII. Fig. 4.

[4] Archiv für Augenheilkunde, xxi. 29.

[5] Archives of Ophthalmology, viii. 501.

[6] Transactions of the American Ophthalmological Society, iv. 558.

[7] Archives of Ophthalmology, xi. 461.

[8] Text-Book of Ophthalmoscopy, p. 95.

[9] Archiv für Augenheilkunde, xix., Taf. IV.

[10] Ibid., March, 1888.

Post-natal persistence of the hyaloid artery of fœtal life, usually present, according to Müller,[4] in oxen and some other animals, such as swine, the moose, and the sheep, has been seen and described in man by Hannover,[4] De Wecker,[5] Little,[6] Kipp,[7] and others. It appears generally as a wavy slender cord running forward from the excavation of the disk out into the vitreous, sometimes extending sufficiently far, as in one case observed by the writer, to spread out over the posterior pole of the lens into a series of minute capsular branches. It is very seldom patulous, as in a case of Zehender's.[8] Seeley[9] gives the sketch of one in which the detachment occurred at the nerve. Manz[10] has described an almost similar condition post mortem. Despagnet[11] cites a case of monocular persistence of the canal of Cloquet without other abnormality of the eyes: this is very instructive in view of the fact that Everbusch thinks that many so-termed instances of persistent hyaloid artery are in reality nothing but the ordinary canal of Cloquet as found in man, rendered visible to the ophthalmoscope by intraocular irritation and inflammation. Liebreich (*vide* article by Little, *loc. cit.*) instances a supposed case of persistent hyaloid vein, but, as Little justly asserts, there is no anatomical proof of the existence of such a venous trunk. The condition is usually unilateral, although Kipp's case (*loc. cit.*) was binocular. Care should be taken to avoid confusion with new blood-vessel formations, which from time to time have been noted by most competent observers,—these later conditions being nothing but part and parcel of coexistent inflammatory results. Of course, if the embryonic remains are large and extensive, and the case one of long standing, consecutive irritation changes might arise, and thus easily mask the original

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[1] Transactions of the American Ophthalmological Society, 1881.

[2] Archives d'Ophtalmologie, iii. 502.

[3] Transactions of the Ophthalmological Society of the United Kingdom, 1884, p. 152.

[4] Gesammten Schriften.

[5] Klinische Monatsblätter, 1863, S. 260.

[6] Proceedings of the Philadelphia County Medical Society, iv. 54.

[7] Archives of Ophthalmology and Otology, iii. 70.

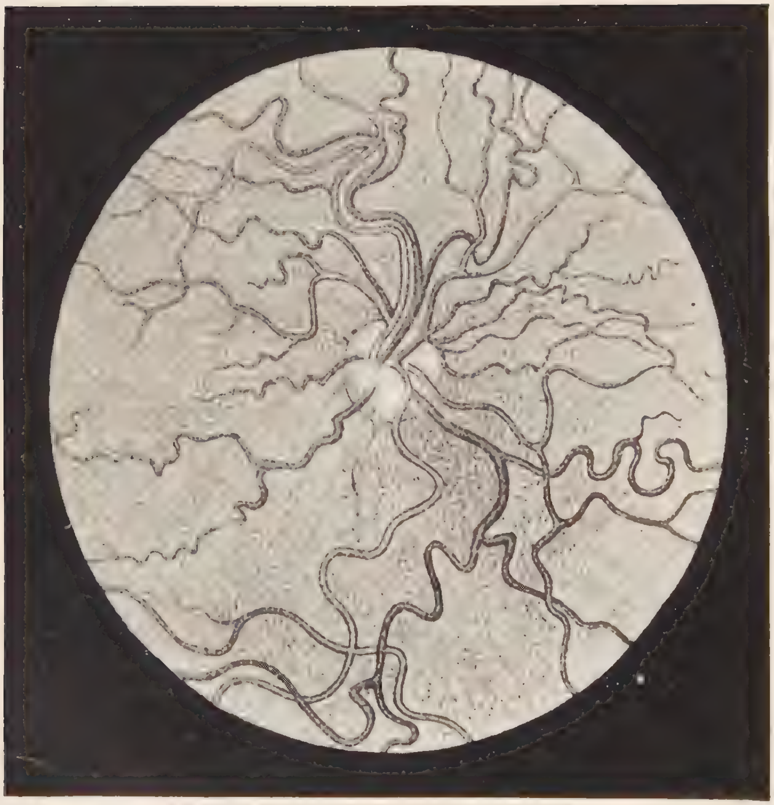
[8] Klinische Monatsblätter, 1863, S. 259.

[9] Transactions of the American Ophthalmological Society, 1882.

[10] Graefe und Saemisch's Handbuch, 1880, ii.

[11] Recueil d'Ophtalmologie, September, 1888.

PHOTO. III.



Undue Tortuosity of the Retinal Vessels. (Benson, Transactions of the Ophthalmological Society of the United Kingdom.)

condition and give rise to confusion in diagnosis. Careful study of the case, with special attention to the state of the related tissues, will frequently give clue to the proper answer.

Actual decrease or increase of the normal number of retinal vessels, though probably more frequent than has been noticed in ophthalmic writings, should always be looked for and considered. Mooren's case[1] of complete want of retinal vessels in an infant, and Von Graefe's instance[2] of faulty development of the retinal vessels in a young lad, are typical extremes of the former type, whilst Benson's[3] and Nettleship's[4] descriptions and pictures of three cases in which both the retinal arteries and veins were so extremely tortuous, large, and numerous as to give the eye-ground the appearance of commencing inflammation, well illustrate the latter. The accompanying reproduction shows this condition very well in Mr. Benson's case. Two of the reported cases were subjected to traumatism, though whether this constituted any causal relationship, or whether the conditions noted were mere intraocular expressions of some obscure congenital or neural disturbance, it is impossible to determine.

Anomalous vascular anastomoses, though comparatively infrequent to any marked degree, should be carefully studied and noted, so as to prevent improper deductions as to result in cases where the usual symptoms of disease are changed by collateral circulation through anomalous channels. Here vessel-distribution and blood-supply, as elsewhere, are subject to so much variation that constant guard must be kept upon the possibility of intercommunication. Benson[5] gives a drawing showing an anomalous distribution of the retinal arteries. There were evidences, however, of some past pathological process in the retina. He believes his case unique "where three-fourths of the retina received its blood-supply from the inferior artery, and only one-fourth from the superior artery of the disk." Photo. IV., facing page 174, gives an excellent idea of this remarkable peculiarity in the venous distribution on the disk as seen in a case by Randall,[6] who gives an interesting review of venous anomalies upon the optic disk.

Lang and Barrett[7] found in forty-eight unselected cases that eight (sixteen and seven-tenths per cent.) gave distinct evidences of *cilio-retinal vessels.* They define the anomalous condition as one in which the vessel "dips into the nerve near the margin of the optic disk, and which can be seen to arch outward, that is, away from the disk, before it finally disappears from view." Randall[8] reports some very interesting cases, and thinks that "about one eye in every five examined shows some form of the condition in question."

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[1] Ophthal. Beobachtungen, 1867, S. 260.

[2] Archiv für Ophthalmologie, i. 403.

[3] Transactions of the Ophthalmological Society of the United Kingdom, ii. 55.

[4] Ibid., ii. 57.

[5] Ibid., 1883, p. 101.

[6] Transactions of the American Ophthalmological Society, 1888, p. 117.

[7] Royal London Ophthalmic Hospital Reports, January, 1888.

[8] Transactions of the American Ophthalmological Society, iv. 511.

Gunn details an instance of *direct arterio-venous communication of the retinal vessels* in one eye, the case being complicated with cilio-retinal anastomosis in the fellow-eye. Under the title of "Persistent Hyaloid Artery," Wells and Liebreich instance a case[1] in a sixteen-year-old boy, "where, arising from one of the arteries of the disk, was seen a small arterial twig running with a slight bend for a short distance into the vitreous humor, ending in a loop and passing over at once into a vein, which, twisting itself like a corkscrew three times around the artery, terminated in one of the large central veins."

Spontaneous pulsation of retinal arteries is quite rare in healthy children, and, when present, is generally dependent upon anatomical peculiarities on the disk-surface or slight unaccountable changes in the character of intra-ocular tension. Venous pulsation, which is readily provoked by increased cardiac action through excitement or sudden movements, must not be mistaken for a symptom of pathological change. A few moments' rest will ofttimes dissipate it, so that when the fellow-eye is examined all trace of it will be lost.

Though comparatively rare in children, yet irregular isolated *thickenings* of all grades of opacity *in the vessel-walls* are met with, and sometimes of sufficient moment to render the contained current invisible. These changes are most pronounced upon the main stems and at the vessel-entrance. In marked cases, the intervening extents of vessel-wall have their density sufficiently increased to change the color of the underlying blood-column, and to permit the side walls of the vessel to be distinctly seen as translucent bands.

Areas of glittering reflexes known as "*shot-silk opacities*," which move with every turn of the ophthalmoscope, especially along the course of the retinal vessels and in the macular region, are frequently seen. They manifest themselves only in the young, and gradually disappear after adolescence. They are dependent upon a normal increase of connective-tissue elements with increased power of reflection.

TRAUMATISM.

Rupture of the choroid the result of *contrecoup* has often been described. Immediately following a blow upon the eye, which has not been of sufficient moment to disfigure the organ externally in the least, there is more or less complete loss of sight. Curiously, however, the apparent gravity of the accident does not seem to bear any relation to this small, though, of course, most important, traumatism. Thus, Mauthner[2] relates a case of double rupture produced by a blow with the fist, whilst Von Ammon's classical case[1] of breakage of the choroid in the yellow-region spot, with bulging of the retina at this point, and no other extravasation within, except a few isolated masses between the choroid and sclerotic, as the result of a discharge of a musket filled with water, gives us an example of a very unusual method. White Cooper's interesting judicial case,[2] where an eye was forcibly struck by a wooden missile, affords another peculiar mode of accident.

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[1] Transactions of the Pathological Society, 1871, p. 222, and A Treatise on the Diseases of the Eye, 1883 (Amer. ed.), p. 503.

[2] Lehrbuch der Ophthalmoscopie, 1868, S. 446.

If attempts be made to examine the interior of the organ by the ophthalmoscope, the path of view may be so obstructed with blood-extravasations as to render this procedure impossible. In such instances either the diagnosis is a faulty one, as necessarily showing that the retina must be involved in the break so as to allow the passage of blood into the vitreous, or else, although the diagnosis is correct, the true condition of break of the choroid is complicated by implication of some of the other structures in the disturbance, such as involvement of the iris or of the ciliary body.

Sometimes, however, the amount of blood-extravasation is so trifling and limited that the fundus details can be seen directly after the traumatism. If so, the break may generally be found somewhere in the posterior pole of the organ, appearing as a C- or S-like white stripe which is concentric with the temporal border of the optic disk. This break is generally bounded by irregular depositions of pigment, whilst small fresh hemorrhages may be seen scattered near and over it. Ordinarily the fundus otherwise appears normal, though in some instances the neighboring area looks disturbed. Sometimes narrow tears may connect with the principal one, or even separated multiple breaks may be noticed. In a few days the hemorrhages disappear, leaving their usual characteristic signs.

Vision, especially central, which at first may be even annihilated upon account of the blood-extravasations, gradually partially recovers, to decrease again with the ordinary signs of metamorphopsia, etc., as secondary changes of cicatrization set in.

Treatment, which is of but little use, and which is directed towards blood-absorption alone, consists in local rest of the organ and leeching, combined with the internal administration of alteratives and absorbents.

Breakage of the retina in association with rupture of the choroid and extravasation of blood into the vitreous has often been observed. Shaffner reports an interesting case in a boy. Noyes[3] details a most curious one in a thirteen-year-old girl, where, in addition to the retinal detachment, there was a laceration of the membrane at the macula lutea. In nearly all of these cases there is no evident external lesion. The prognosis is dependent upon the position and the amount of the disturbance, though, unfortunately, by reason of the *contrecoup*, as just shown, the break is generally situated in or near the macular region. No treatment, except rest and quiet, with the possible use of alteratives, can be advised.

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[1] Archiv für Ophthalmologie, i. 2, 154.

[2] On Wounds and Injuries of the Eye, 1859, p. 233. In reference to the legal question in this case, it may be of interest to note, in passing, an instance which came under the writer's care some time ago, where a fortunate (or possibly unfortunate) attorney in a suit for damages was struck in the left eye by the fist of the defendant. Careful examination revealed a large choroidal break in the macular region, with all the characteristic subjective symptoms. Medical testimony, expressed very briefly and to the point, had the result of bringing in the verdict of "mayhem," with the accompanying penalty.

[3] Transactions of the American Ophthalmological Society, 1871, p. 128.

In almost every case of *injury to the retina from a foreign body which has gained access to the interior of the globe*, the associated results are so severe that it is impossible to decide the position of the offending material. Here, if the substance be iron or steel, the magnetized needle of Pooley will be of service in its detection, and if the case be seen early, the electro-magnet may be of use in extraction. If the particle be of any other nature, such as stone, glass, etc., operative attempts should also be made for its removal, provided there be any possibility of success. As an example of the possible freedom of the organ at times from the series of dangerous after-symptoms usually seen in such instances, the writer has in mind a case seen in consultation with Dr. James Tyson, in May, 1888, of a lad in whose left retina a small sliver of steel became embedded, the passage of the stroke being through the cornea, the lens, and the vitreous. Fortunately, the foreign body escaped striking the iris or the ciliary region, and in consequence, with the exception of a slowly-forming cataract, the eye has remained perfectly quiet and painless from the date of the accident to the present writing, nearly two years. Although many cases could be quoted where foreign bodies have become encapsulated in the membrane and remained quiescent for long periods of time, yet active inflammation, resulting in destructive panophthalmitis, is so prone to occur, that it is best to give a guarded prognosis in every case. If possible, and most certainly if under the immediate care and control of a competent observer, enucleation or evisceration should be postponed until the child's skull has sufficiently developed to prevent facial asymmetry. If the slightest well-grounded suspicion of involvement of the opposite organ be entertained, it is best to sacrifice all question of cosmetics and get rid of dangerous tissues.[1]

Detachment of the retina may occur from blows, as in Brailey's case[2] of a twelve-year-old boy who received a blow upon the eye with a stone; from penetrating wounds of the eyeball, posterior to the ora serrata; or as a sequel of subretinal hemorrhage, as was most probably the cause in Snell's case[3] in an eleven-year-old boy. It has also been seen as one of the sequelæ of progressive inflammation and stretching of the ocular tunics, as, for instance, in malignant myopia, or it may even appear as a symptom of circumscribed malignant disease of the choroid, as shown by Poncet.[4] The theories of its pathogenesis, which are manifold, can be best studied in the noble work of Nordenson,[1] in which he sustains Leber's belief of shrinkage of the vitreous with traction,—a theory supported by De Wecker[2] and controverted by Boucheron and Abadie.[3] The diagnosis, which is very easy, is made in most instances by direct evidence with the ophthalmoscope, in association with corresponding loss of the visual field, and diminished tension. The prognosis is very unfavorable. Enjoined rest, the best of nourishment, with remedial agents such as pilocarpine to promote absorption, can be resorted to, and if, as is almost universally the case, no good arises, operative procedure to dislodge the fluid and produce localized plastic formation may be tried. Treatment, however, is usually of no avail.

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[1] See Noyes, A Text-Book on Diseases of the Eye, 1890, p. 577, on the same subject.

[2] Transactions of the Ophthalmological Society of the United Kingdom, v. 11.

[3] Ibid., vi. 38.

[4] Quoted by Noyes, A Text-Book on Diseases of the Eye, 1890, p. 544.

Choroidal detachment is extremely rare. Noyes,[4] after a long and large experience, has never had a case to come under his notice. In over twenty thousand new cases of eye-disease personally seen by the present writer, he has failed to find a single instance. Cases, however, have been spoken of: thus, Von Graefe[5] describes such a case, and Iwanoff[6] has been so fortunate as to be able to study an eye affected with this condition. Ophthalmoscopically, an isolated area of choroid can be seen protruding markedly into the vitreous, the bulging portion being fixed and immobile. Its surface is uninterruptedly traversed by the retinal vessels. Should the underlying material be serous in character, the bulge may be somewhat pallid; whilst should it be blood, the area will be dark-colored. As the condition advances, intraocular tension falls, inflammatory symptoms set in, and panophthalmitis with phthisis bulbi ensues,—these sequelæ serving to distinguish it from neoplastic formation. Treatment is of no practical value, although aspiration or drainage in the early stages may be tried.

In a peculiar variety of cases, where the traumatism has avoided the eye itself and acted upon the bony walls of the orbit, there is often a subsequent involvement of the optic nerve and retina without early ophthalmoscopic evidence of intraocular disturbance. The after-changes which come on are optic neuritis with consecutive atrophy, retinitis, venous distention, hemorrhages, etc., which suggest either direct pressure upon the optic nerve, as reported by Knapp, interference with the passage of blood in the central retinal artery, as suggested by Noyes,[7] or fracture of the bony optic canal with extravasation of blood into the sheaths of the optic nerve, as shown by Von Hölden.[8] Pigment-deposits upon the disk, from degenerative changes in the blood which has been extravasated from the intravaginal spaces in the optic nerve, have been seen. In a few cases no ophthalmoscopic signs have been observed. In this last grouping, Schweigger's saying,[1] that "the anatomical condition of the nerve-trunk in neuritis has been examined often enough to prove that changes not visible by the ophthalmoscope may play an important part in causing the disturbances of vision," must be borne in mind. If the nerve be injured sufficiently forward to include the central artery of the retina, the ophthalmoscopic signs simulate those of embolism. Pagenstecher cites the history of a very interesting case in a young girl. Treatment is either purely surgical or to be directed against the special dyscrasia or condition, combined with ocular rest.

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[1] Die Netzhautablösung, Wiesbaden, 1887.

[2] Annales d'Oculistique, March, 1888.

[3] Ibid.

[4] A Text-Book on Diseases of the Eye, 1890, p. 586.

[5] Archiv für Ophthalmologie, iv. 2, 226.

[6] Ibid., xi. 1, 191.

[7] A Treatise on Diseases of the Eye, 1881, p. 350.

[8] Berlin, Graefe und Saemisch, vi. 588.

A case of metamorphopsia following a blow on the eye has been described by Aub,[2] which can probably be attributed to a low grade of inflammatory change in the retina in the region of the macula lutea, causing a dislocation of the position of the rods and cones. The writer has seen two cases of traumatism without apparent external lesion, except an irregular thickening of the corneal epithelium, where the ophthalmoscope showed a circumscribed area of disturbance in the choroid and retina between the macula and the disk; this condition of the fundus rapidly disappearing in a few days' time upon protecting the eye from light and undue exposure. Jackson[3] gives two chromo-lithographs of a similar though more marked condition seen in the eye of an adult.

Damaging effect upon the retina from exposure to direct solar rays has been observed. Central scotoma is the initial symptom. Examination of the fundus shows a bright whitish spot at the macula lutea, surrounded by a red rim. The permanency of damage to the tissues is in direct relation to the amount of primary injury. In Deutschmann's experiments upon the retina there were changes in the vascular walls, with coagulation of retinal albumen. Exalted sensibility of the retina from the same cause has been noted. In three patients who were unduly exposed to direct solar rays, Magawly[4] has noticed central scotomata for red with reduction of vision to one-fourth of normal. The symptoms soon ceased upon placing the patients in the dark and protecting their intraocular tissues by dark glasses. Lubinsky[5] has seen thirty cases of suffering from undue *exposure to the electric light.* Besides daily exacerbation of photophobia and lachrymation, the ophthalmoscope revealed a slight congestion of the optic nerve tip. Maklakoff[6] has studied the personal effects of the "voltaic light" upon the eyes. He concludes that such a light nearly always acts by its chemical properties.

Ametropia as found in abused and stretching eyes may give all the evidences of low choroiditis with retinal disturbance, and even optic nerve swelling, without any subjective acknowledgment of the intraocular condition.

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[1] Hand-Book of Ophthalmology, 1878, p. 484.

[2] Archives of Ophthalmology and Otology, ii. 173.

[3] Transactions of the American Ophthalmological Society, 1888, p. 68.

[4] Zeitschrift für Schulgesundheitspflege, No. 4, 1889.

[5] Wjestnik Ophtalmologie, May, 1889.

[6] Moniteur d'Ophtalmologie, January, 1889.

Direct implication of the optic nerve with extension to the retina from other inflammatory processes in the orbit, such as cellulitis, periosteal disease, tumor, etc., may occur, where either pressure from the morbific material causes rapid loss of sight followed by atrophic changes in the disk, as described by Allbutt, or invasion of the products of the disease into the tissues of the nerve itself may give rise to a similar ophthalmoscopic sign. Here, at first, rapid monocular failure of vision, with but little congestion of the disk and a doubtful haze of the retina around and near the macular region, is the symptom usually noted. With this failure of sight there is generally associated periorbital neuralgia, with pain upon motion of the globe or when decided palpation of the organ is made. In view of Hock's[1] belief that the position of pressure or the localized retro-ocular neuritis may be determined by the direction of motion giving the greatest pain, it would be well to apply this diagnostic procedure to a careful test in all suspected cases. If the case progresses, paralyses of contiguous nerve-terminals may occur, atrophic changes in the disk, with increasing "fog" before the eyes, central scotomata, and rapidly-lessening field of vision and diminution of central color perception take place, until at last atrophy with blindness ensues. Nettleship[2] details some instructive cases in adults. Again, an intraorbital point of infection may produce a descending neuritis with all the ordinary subjective and objective signs of inflammation. The ophthalmoscopic picture in these cases is discriminated by the concomitant extraocular symptoms from similar fundus changes that may appear in cases of cerebral disease, or which are seen in the course of some general dyscrasia. In fact, in all cases of monocular neuritis suspicion should most certainly point towards local disturbance. In every case of this character that the writer has seen there has been some local trouble either in the orbit itself or in the cranial cavity just at the optic foramen to account for the neural disturbance. Of course it will be readily understood that a localized inflammatory condition of the tissues of the orbit can easily appear as symptomatic of some general dyscrasia. Here the intraocular conditions are identical. It must be remembered that mere stretching of the intraorbital portion of the optic nerve may also give rise to deterioration of vision. Ayres's cases,[3] in which there was extravasation of blood in the orbit, causing both pressure and stretching, furnish good illustration. True orbital aneurism may be productive of the same condition. Hermann Pagenstecher[4] relates an almost unique instance of direct injury of the optic nerve, with rupture of the central retinal artery and corresponding vein, by an iron rod. The patient, a girl of twelve years, was completely blind. Ophthalmoscopically, the optic disk could not be seen, on account of a large, dense, glistening effusion, apparently several times greater in area than the nerve-head itself. A single retinal vessel was alone visible. In several days' time absorption took place, which revealed the disk-surface, whilst collateral retinal circulation ensued. Hocken[1] quotes from Mackenzie an historical case related by Hórstius in the seventeenth century, where a fourteen-year-old boy was rendered blind, without any perceptible ocular lesion, by an arrow-tip which had been driven into the orbit. The foreign body remained *in situ* for more than thirty years. The present writer remembers an instructive case of retro-bulbar breakage of the optic nerve anterior to the entrance of the central retinal artery, which he saw in his service at St. Mary's Hospital. The wound, which was self-inflicted, was caused by the upward entrance of a No. 22 pistol-cartridge, which passed through the lower outer part of the right orbit into the cranial cavity. Eight hours after the injury, the optic nerve entrance seemed whitish, swollen, and puffed, whilst the portion of the retina that could be seen peripherally appeared utterly devoid of capillarity. In the few days that remained for study, no changes, except a slight subsidence of the disk-swelling, with a gradually-increasing haze of the outlying retina, took place. Retinal circulation never reappeared. The cornea became hazy and dry, the conjunctiva seemingly thickened and became slightly covered with excreta. Death took place in four days after the injury.

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[1] Centralblatt für prakt. Augenheilkunde, iv., 1884.

[2] The Lancet, 1880, i. 766.

[3] Archives of Ophthalmology, 1881, p. 42.

[4] Archiv für Ophthalmologie, xv. 1, 223.

TUMORS OF THE CHOROID, OPTIC NERVE, AND RETINA.

Tumors of the Choroid.—Although it is well known that almost all choroidal tumors are sarcomatous in character, which form of neoplasm is eminently one of adult life,[2] yet it is deemed of sufficient importance not only to speak of the occurrence of choroidal tumor during childhood, as a disease which should be carefully differentiated from subretinal effusion, with which it has been unfortunately confounded, but also to give a short exposition of its characteristics, by which recognition of its presence may be obtained in time to effect a ready and speedy removal of a nidus of general infection. The disease itself is comparatively rare in Germany,—one in fifteen hundred cases,—as Fuchs[3] tells us, with a still lower proportion—one to two thousand two hundred and eighteen cases—in the English hospitals, as shown by Berry.[4]

Of slow growth, with a broad foundation, and generally first appearing between the disk and the macula, it gradually pushes the retina forward into the vitreous. Frequently this elevation is surrounded by a serous effusion, though this, if carefully looked through, is generally insufficient to hide the brownish mass beneath.[1] Often this protuberance can be seen covered by a faint and irregular congeries of vessels, situated upon a deeper level than the overlying retinal stems. This stage of the disease is known by Knapp[2] as the first—the "quiescent or non-irritative"—stage. Should the neoplastic formation be situated nearer the equator of the eye, towards the ciliary region, the second stage, or the "inflammatory" (Knapp), much sooner appears. Here glaucomatous symptoms arise, tension increases, ocular and ciliary neuralgia ensue, the lens opacifies, the anterior veins enlarge, the cornea becomes anæsthetic, and the anterior chamber is annihilated: in fact, all the symptoms of increased pressure are present. Ophthalmoscopic examination now becomes impossible. In various grades of objective change, some in one case more pronounced than in another, the second stage gradually ceases, and the "extraocular stage" (Knapp) is reached. The mass now perforates the globe and rapidly increases and ulcerates. The external appendages and the surrounding tissues soon become involved, whilst the pain recommences, until at last the "metastatic stage" (Knapp) is reached. Finally the case leads to fatal termination.

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[1] A Treatise on Amaurosis, 1842, p. 96.

[2] Fuchs's (loc. cit.) statistics give but eleven under ten years, and twenty-seven under twenty years of age, out of two hundred and fifty cases.

[3] Das Sarcom des Uveal-Tractus, 1882.

[4] Diseases of the Eye, 1889, p. 348.

As our study in this section is limited to the ophthalmoscopic diagnosis of this insidious and secret disease, it will be only necessary, from what has been said before, to give a few differential points which denote its presence. Except in the very rare form of leuco-sarcoma, the most incompetent observer can hardly fail to distinguish it from glioma of the retina: moreover, as the treatment is the same in these two diseases, no great therapeutic error can arise. With choroidal tuberculosis the problem is more difficult. In fact, this form of choroidal disease has been mistaken for sarcoma; yet here the general type of the oatient, the ordinary relative positions of the two varieties of growths, the usual greater multiplicity and smaller size of the tubers, the color and the long persistence of the sarcomatous growth, with its usual increasing pressure-signs, all serve to separate the diagnosis of one disease from that of the other. Complicated as choroidal tumor is almost from the first with retinal detachment, the observer must be well trained, or the subretinal effusion be very transparent, to enable him to differentiate it from simple retinal detachment. Later, however, as the choroidal mass grows and forces the liquid and solid contents of the globe against the various canals which serve to maintain normal equilibrium between fluid and solid, the pathognomonic pressure-symptoms of new growth appear, and intraocular tension increases, with all its dire consequences; whereas in retinal detachment intraocular tension, as is well known, gradually lessens. Moreover, at first some value may be set upon the almost absolute immobility of the overlying retina, as well as upon its comparative smoothness.

In children the subjective symptoms at first are seldom, if ever, brought to attention, and it is only as the second and later stages are reached that the disease is recognized. Prognosis, as a rule, is bad, whilst treatment resolves itself into an immediate enucleation, even though vision be comparatively good.

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[1] The fact that Hirschberg (Archiv für Ophthalmologie, xvi. 304) reports an instance of leuco-sarcoma in a girl of twelve years, should render us careful in this differential point.

[2] Die Intraoculären Geschwülste, 1868.

Granuloma, angiomata, and enchondroma of the choroid have all been seen. The cases have almost universally been found among adults.

Tumors of the optic nerve, comparatively rare themselves, are more frequent in childhood than in adult life, Sym having found sixty per cent. of sixty-nine cases in patients under twenty years of age. They appear both in the intraocular ending and in the nerve during its passage through the orbit. The most frequent forms are sarcomata and gliomata, with their related types, and neuromata, as in Perls's case. Sutphen[1] notes an extraordinarily large-sized and peculiarly-shaped sarcomatous growth of the small-cell variety. Syphilitic gummata are said also to have been seen. The left nerve seems to be the one the more frequently attacked. Von Graefe gives a case[2] in which, ophthalmoscopically, there could be seen a swelling confined to the nasal half of the disk, with dilatation and tortuosity of the retinal veins and contraction of the arteries from a myxoma of the nerve posterior to the globe, and Lawson[3] gives the clinical history of a case with the pathological report of a post-ocular sarcoma of the optic nerve undergoing myxomatous degeneration in a twelve-year-old boy, who for months had gradually lost vision, associated with steadily-increasing proptosis. These two instances may be cited as very interesting examples of such growths. The accompanying phototypes give excellent ideas of the appearance of the tumor in the latter case, both before and after section. Frothingham[4] gives the histories and the results of examination by the microscope of his cases of round-celled sarcomata in children. Ayres[5] records a most careful study of a case of sarcoma of the small round-celled variety in a twelve-year-old boy. In view of Michel's observation[6] of hyperplasia of the intracranial portion of the optic nerve, and the chiasma itself, in a case of elephantiasis in a man, it would be of interest and value to have a series of careful ophthalmoscopic examinations made by those who are favorably situated among a large number of infected children (or adults), in order to determine the exact nature of the resultant physical changes and the character of the functional disturbance produced.

Diagnosis is, in the main, dependent upon the following symptoms: progressive outward, upward, and forward, and sometimes downward, protrusion of the eyeball; rapid loss of vision, with frequent ophthalmoscopic signs of infiltration; pressure and atrophy; long continuance of seemingly

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[1] Transactions of the American Ophthalmological Society, 1889, p. 451.

[2] Archiv für Ophthalmologie, x. 1, 193.

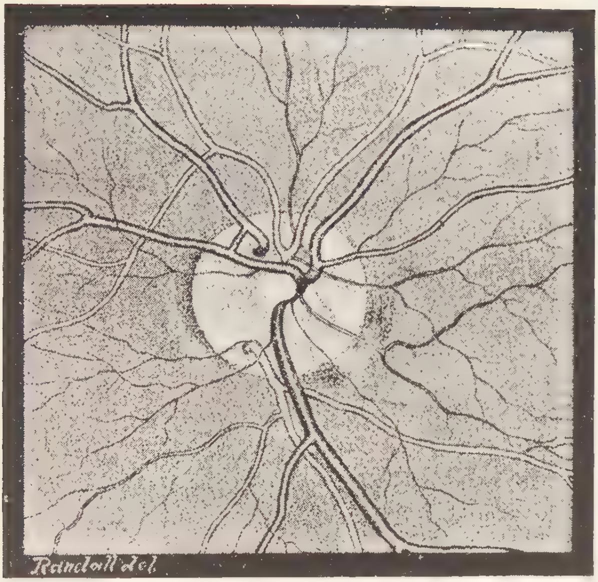
[3] Royal London Ophthalmic Hospital Reports, 1888, p. 1.

[4] Ayres, Journal of the American Medical Association, December 10, 1887.

[5] Loc. cit., March 8, 1890.

[6] Lehrbuch der Augenheilkunde, S. 642.

PHOTO. IV.



Anomalous Venous Distribution on the Disk. (Randall, Transactions of the American Ophthalmological Society, 1888.)

PHOTO. V.

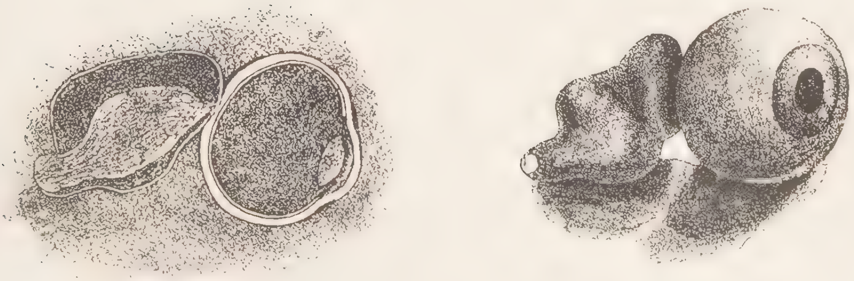


Fig. 1.

Fig. 2.

Post-Ocular Sarcoma of the Optic Nerve. (Lawson, Royal London Ophthalmic Hospital Reports, 1888.)

free ocular motion; and, generally, complete absence of pain. Treatment in every instance should consist in early extirpation of the entire tumor-mass. If the eyeball is not affected, the nerve itself, as Knapp suggests, may be excised. This rule is especially applicable to children, though care should be taken to watch carefully the slightest tendency to recurrence from some infiltrated nerve-tissue which may be inadvertently left. It should be remembered, however, that fatal septic meningitis has several times taken place in cases where the eyeball has been allowed to remain, and that even where such brilliant success has followed as in Schiess-Gemusseus's case,[1] instances have been reported where suppurative panophthalmitis has followed the traumatism.

Glioma of the retina, as Virchow[2] terms it, is almost essentially a disease of infancy and childhood, rarely, if ever, appearing in the adult. Without entering into the various disputes as to the initial point of infiltration, and avoiding any discussion as to its exact pathological nature, it will be sufficient to state that the bulk of opinion shows that it is practically identical with small round-celled sarcoma, and that, although it may begin in any of the retinal layers, it most frequently first manifests itself in the "external granular." In regard to its etiology, Brailey[3] is of the opinion that retinal glioma runs a much slower course than is commonly supposed, and fancies "that it always takes its rise within the period of intra-uterine life."

During the early stages there are no symptoms visible to the naked eye, but at a later period a peculiar whitish-yellow reflex, often metallic in tint, just back of a partially-dilated pupil, manifests itself, giving the appearance of the "amaurotic cat's eye" of Beer. Should the case have been examined earlier, as has been done by Von Graefe,[4] numerous small white swellings would have been seen in one of the granular layers (or in the fibre layer) of the retina. These would soon have increased in size and their surfaces coalesced. Should the disease have commenced in the granular layer, the retina itself would have become detached in areas at its outer portion, giving rise to circumscribed masses of protuberant vascular vegetations containing more or less broad, smooth surfaces. If not, the massings would generally have projected into the vitreous, and have carried the implicated portions of the retina with them before the membrane had become detached.

It is at this time the case is generally seen. The tension of the globe is perceptibly increased. The intraocular changes continue, secondary foci appear, and more intense pressure-symptoms ensue. The lens and the iris are pushed against the cornea, the lens rapidly degenerates, the pupil becomes dilated and immobile, and the cornea is "steamed" and anæsthetic:

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[1] Archiv für Ophthalmologie, xxxiv.

[2] Die Krankhaften Geschwülste, ii. 1, 159.

[3] Transactions of the Ophthalmological Society of the United Kingdom, 1885, p. 62.

[4] Archiv für Ophthalmologie, xvi. 129.

in fact, all the symptoms which are found in acute attacks of glaucoma ensue. Often, at this point, suppurative inflammation takes place in the interior of the globe, and the eye soon becomes atrophic and degenerated, allowing the neoplasm to escape and invade the adjacent structures. If not, the tumor gradually seeks its way out by either creeping along the nerve-tissues or actually breaking the ocular walls. Once free, it rapidly becomes a projecting mass of deep red tint, constantly oozing blood and secreting pus. New foci form and coalesce into large, irregular masses. The infiltration passes up into the brain-cavity, the orbital walls become involved, and metastases occur, until, at last, from exhaustion, or from some intercurrent complication, the patient is relieved by death.

In spite of reported cases of long remission, recurrences[1] and metastases are so frequent and so increasingly formidable that prognosis is truly grave.

Treatment.—If the neoplasm be even extremely minute and confined to the interior of the globe, immediate enucleation, taking care to excise the optic nerve as far back as possible, should be practised. After the removal of the eyeball, its exterior should be carefully studied, as Bull[2] has shown that small secondary tumors on the outer side of the sclera may exist without any visible microscopic connection with the intraocular growth. If the orbit be invaded in the least, or if the globe show marked signs of internal inflammatory reaction without breakage, total evacuation of the entire orbital contents should be done. If double gliomata exist, double enucleation must be resorted to, or exenteration if necessary.[3] In all such cases, however, we can do no more than repeat Frost's question, "Are there any cases in which life has been actually preserved by the excision of both eyes?"[4]

Sarcoma of the retina in the young is practically unique, although there can be but little doubt of its possible existence. It generally results by extension from the choroid, as in Williams and Ayres's case of a twelve-year-old girl, where Knapp[5] after careful examination found that the choroidal neoplasm had extended to the retina by direct propagation. Enucleation of the infected organ should be advised and immediately done, and the child's parents warned of probable distant metastases.

Tubercles in the retina will be considered under Tuberculosis.

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[1] Noyes (A Text-Book on the Diseases of the Eye, 1890, p. 583) says, "a single case is given in which the patient survived after the removal of a secondary tumor."

[2] Wells, A Treatise on Diseases of the Eye, 1883, p. 550.

[3] Dujardin's report of a case of monocular glioma in which four weeks after the enucleation of the affected eye the fellow-eye, which appeared perfectly normal, both externally and ophthalmoscopically, became totally blind, is interesting as showing that the neoplasm had most probably travelled back to the optic nerve of the affected organ, and, upon reaching the chiasm, had destroyed the related tissues of the sound eye.

[4] Transactions of the Ophthalmological Society of the United Kingdom, v. 64.

[5] Archives of Ophthalmology and Otology, 1874, p. 241.

II. SYMPTOMATIC DISORDERS.

LESIONS DEPENDENT UPON DISORDERS OF THE CIRCULATORY SYSTEM.

Vascular disease in the young is not so prone to give ophthalmoscopic pictures of changed and altered retinal circulation as it is in older eyes, which are less elastic and in which the tissues at large have not the same powers of ready compensatory action. Even in extreme cases intraocular tension so carefully protects the retinal vessel-walls as to allow very little or no departure from the normal.

In *congenital cyanosis* the retinal veins have been found greatly dilated, giving, according to Gowers,[1] proof that the distention of the venous radicles contributes to the general cyanotic tint of the child. Stangloncier[2] reports the occurrence of retinal hemorrhage immediately preceding death, and Leber[3] has seen cases where both the retinal arteries and the retinal veins were distended.

Acquired valvular disease may show itself in the retina by marked changes in the retinal circulation, and, in fact, these changes have contributed to the diagnosis of the condition.

In some cases of *aortic insufficiency* with regurgitation, Quincke,[4] confirmed by Becker,[5] has shown that there is an alternate systolic flush and diastolic paling of the disk, comparable with the capillary pulsation seen through the finger-nails of such subjects. In other cases the intraocular pulsation may be seen in the larger veins and arteries of the retina.

Any condition, such as *mitral disease,* causing pulmonary obstruction and producing general venous engorgement must naturally give rise to distention with apparent increase, and tortuosity, of the retinal veins. Extreme dilatation of the right heart may cause similar appearances. Choroidal hemorrhage has been noted by Westphal[6] in what is termed malignant endocarditis. Generally, however, such cases are found in the female adult and seem to be associated with the post-puerperal period. Valvular disease of the heart, especially where there is a tendency to the formation of blood-clots and vegetations, is sometimes productive of *embolism of the central artery of the retina,* or of one of its branches within the eye. It can, as Gowers says, occur at any age. Warren Tay[7] has had the good fortune to see an infant of twelve months that, without definite history, presented an appearance in each macular region which closely simulated embolism of the central artery of the retina. Four months later he notes that although the macular changes remain the same, yet "the disks are now *undoubtedly becoming atrophic.*" Later,[1] he notes a third instance in the same family.

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[1] Medical Ophthalmoscopy, 1882, p. 200.

[2] Inaug. Diss., Würzburg, 1878.

[3] Graefe und Saemisch, v. 524.

[4] Berliner Klinische Wochenschrift, 1868.

[5] Archiv für Ophthalmologie, xviii. 207.

[6] Archiv für Psychiatrie, ix. 3, 389.

[7] Transactions of the Ophthalmological Society of the United Kingdom, 1881, p. 55.

If the embolus should become lodged in the main trunk, the child will complain of a sudden loss of vision,—generally upon the left side. If one of the minor stems be involved, the loss of vision may not be observed. Should the former case be seen immediately after the accident, the nerve will appear pallid, the arteries greatly contracted, and either empty or containing very thin stationary columns of light blood; the veins will appear reduced in size, tapering as they enter the affected region, and holding currents of immobile dark blood, whilst no visible pulsation in the retinal vessels can be produced by artificial increase of intraocular tension from pressure of the finger upon the globe. In a few hours the disk-edges disappear beneath the retinal elements, which have become transformed into a large, irregular area of œdematous and semi-opaque swelling extending beyond the macular region. By reason of the thinness of the membrane in the region of the yellow spot, the choroid is seen more plainly at this place, and gives the characteristic cherry-red spot. This spot, as a rule, is surrounded by a corona of small vessels. Beaded columns of blood now begin to pass through the most patulous veins, whilst the arterial currents become re-established in the same way. The calibre of the veins increases. Small hemorrhages occur, and remains of old hemorrhages appear. The œdema, which may have become greater, gradually lessens, until at last the disk-edges again appear, the cherry-colored macule fades entirely, and the original retinal level is reached. If, however, the process continues, atrophy of the nerve ensues, and degenerative changes in the affected region take place, until the eye, if not entirely blind, is rendered useless for all practical purposes. The accompanying reproduction from a doubtful case of Jaeger[2] gives an excellent idea of the condition during the height of the attack.

If the embolus be confined to one of the smaller twigs, the localized results may be of such little moment as not to interfere with useful vision.

Prognosis is bad in direct proportion to the size and situation of the embolic mass. It may be interesting to note in this connection that Benson reports one case[3] in which the presence of a cilio-retinal artery offered sufficient collateral circulation to preserve good central vision, whilst the peripheral field entirely disappeared. Massage to promote re-establishment of circulation has been tried, and, although as yet with no practical value except in a few instances, is worthy of repetition. Absorbents, or drugs which have effect upon the size of vessel-calibre, might be tried. The attack should act as a warning, and the parents should have certain hygienic measures as to the management of the child and a system of dietetics as to its care given to them towards the prevention of the danger of the lodge-

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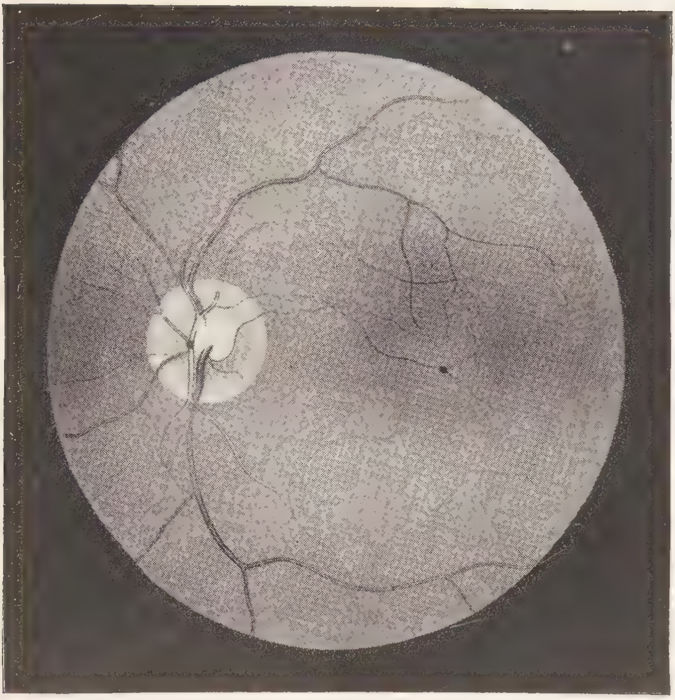
[1] Loc. cit., iv. 158.

[2] Beiträge zur Pathologie des Auges, Plate XXX.

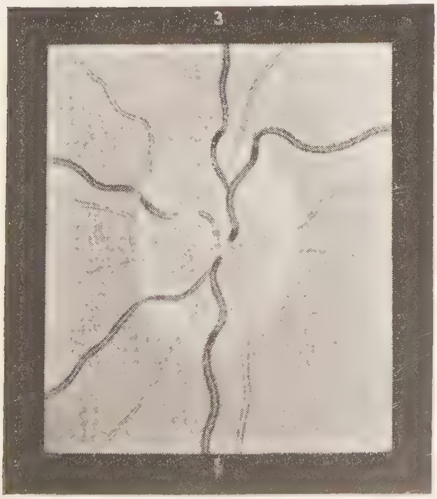
[3] Royal London Ophthalmic Hospital Reports, x. 3, 336.

PHOTO. VI.



Embolism of the Central Artery of the Retina. (Jaeger, Beiträge zur Pathologie des Auges, Plate XXX.)

PHOTO. VII.



Optic Neuritis of General Anæmia. (Gowers, Medical Ophthalmoscopy, 1882.)

ment of other emboli in more serious places, such as the brain; whilst the physician should exercise due care that the rules made are faithfully carried out.

In two cases of cardiac disease, where sudden blindness, resolving itself into a temporary central scotoma, followed by a return to normal vision, was associated with a peculiar retinal haze and congestion extending from the macular region to the disk, Knapp[1] ascribed the intraocular appearances to *embolism of the choroidal vessels.* Both subjective and objective symptoms ultimately disappeared.

Diseased condition of the coats of the vessels may either directly or indirectly give rise to *thrombosis of the central retinal artery.* No authentic description of its early ophthalmoscopic appearance in children has, so far as the writer is aware, been made. The probable primary change would be decreased size of the retinal artery and its branches. Unfortunately, from the usual character of the vessel-disease, canalization of the thrombus can hardly be expected, and atrophic changes in the globe would soon occur and destroy the organ, either partially, as in Virchow's[2] case, or totally, as in Gowers's.[3]

Phlebitis from mitral and aortic disease may produce *thrombosis of the central retinal vein* in the young. Local disturbance in some part of the membrane has produced thrombi in the walls of the retinal veins. The ophthalmoscopic appearances are somewhat like those of embolism, but there are said to be certain distinctive differences between the two affections. In thrombosis the arteries are never empty; the veins, although, as a general rule, enormously dilated, may be normal in size; hemorrhages are much more numerous; venous pulsation can sometimes be provoked, and the vitreous may become opaque. Vision, as a rule, is never completely lost, and improvement may take place.

It must be conceded by those who have had much opportunity for observation that differential diagnosis is often exceedingly difficult, and may, indeed, prove impossible, on account of association of the two conditions.

The treatment is practically the same as that for embolism.

Choroiditis of a purulent type has sometimes been seen as one of the sequelæ of emboli in the choroidal arteries, or even of thrombi in the ophthalmic veins. Although of infrequent occurrence in children, yet cases have been observed. The entire grouping of panophthalmitis signs, so common after traumatism, are repeated in all their severity, whilst the termination of the case, as is well known, is almost sure to be absolute blindness, with destructive and atrophic degeneration of the globe.

So-called *ischæmia of the retina* is quite rare. Von Graefe's[4] early description of a case in a girl of five and a half years, who suddenly became blind in both eyes, is of clinical interest. The optic disks were found slightly hazy, the retinal arteries greatly contracted, and the retinal veins enlarged and tortuous. Intraocular tension in each eye seemed normal. The child's pulse being quick, weak, and thread-like,—indicating feeble heart-action,—he was led to believe that the blood-current force was unable to overcome the ordinary tension of the globe. This view induced him to lower intraocular tension. This was done by puncture of the cornea, with evacuation of the aqueous humor, in one eye, and an iridectomy upon the fellow-eye, just as is done in glaucoma. The effects were marvellous. Sight speedily returned to normal, and all the ophthalmoscopic changes disappeared. The condition of the veins in these cases would lead the writer to think that the true cause is most probably compression of the central artery by hemorrhage in the nerve-substance, or is one of the results of a small thrombus which has become lodged in the corresponding vein during the passage of the vessel through the nerve posterior to the globe. The whole subject, however, must remain *sub judice* until more accurate data are obtained.

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[1] Archiv für Ophthalmologie, xiv.

[2] Archiv für Path. Anatomie, x. 159.

[3] Medical Ophthalmoscopy, 1882, p. 29.

[4] Archiv für Ophthalmologie, viii. 1, 143.

Endocarditis,[1] degenerative changes in the vascular walls from various dyscrasiæ, hereditary conditions, as seen in cases of hæmophilia, etc., may all be productive of *hemorrhage into the retina*. The ophthalmoscopic appearances are typical. There is very little or no inflammatory change visible. Areas of extravasated blood, especially near the bifurcation of the larger vessels, show themselves, indicating, by their peculiar shapes, the structure of the retinal layer in which they have occurred. In a few weeks' time they gradually fade, leaving, in many instances, either faint pigment-massings or spots of disturbed choroid and degenerate retina.

The prognosis is directly proportionate to the position, the number, and the size of the hemorrhages.

Treatment.—Local measures, independent of rest of the eye, are of but little use. The cause must be sought, and measures directed towards it, taking care to keep the patient as quiet as possible, the emunctories well open, and the digestion in a good condition.

Eales[2] has described some cases of *recurrent retinal hemorrhage* associated with epistaxis and constipation. They appear at or just after adolescence. All the cases seen were in males. The extravasation generally comes on during a paroxysm of coughing or laughing, but may ensue without any assignable cause. No evil results seem to follow, the blood gradually clearing away and allowing a clear view of the fundus. One peculiarity noticed was a tendency to tortuosity of the peripheral portions of the retinal veins. The writer has been so fortunate as to see one of the cases mentioned in Mr. Eales's paper, and found direct evidence of hemorrhagic extravasation into the sheaths of one of the retinal arteries, whilst the patient presented unmistakable signs of hepatic disease.

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[1] Chronic endocarditis will be spoken of more fully under "Chronic Bright's Disease," to be found in a succeeding section: this has been done not only to keep the subject-matter together for associated reference, but also because it will most probably be sought for in this position.

[2] Birmingham Medical Review, July, 1880, p. 262.

Small aneurisms in various viscera are apt to have *localized dilatations of the retinal arteries* associated with them. They are almost unknown in the young, but are found connected with vascular changes in the special dyscrasiæ of the old, as renal disease, syphilis, etc. They are recognized either as large, oval pulsating tumors at or near the disk, or as minute pin's-head dilatations, generally situated in the bifurcations of the smaller arterial stems. Hocken[1] instances an aneurism of the central artery of each retina.

Intracranial aneurisms, which as a rule are basilar, very seldom, if ever, cause any visible dilatation of the retinal arteries. Leber says that in some cases of teleangiectasis of the conjunctiva and lids, the intraocular vessels are found in a similar condition.

Sudden hemorrhage, especially if spontaneous and in subjects with greatly-impaired strength, may give rise to *loss of sight, with or without decided ophthalmoscopic change.* It may appear at any time of life, though most of the reported cases have been seen in adults. The blinding is double in about ninety per cent. Upon account of the many classes of subjects and the special dyscrasia and condition under observation, the ophthalmoscopic appearances described have been so varied that it would be impossible to formulate any strict typical picture. Neuritis, retinal complication with hemorrhage, disk pallid, with diminished arteries and large veins, and total absence of symptoms, have all been noted by competent observers.[2] If the disturbance has been profound, atrophic changes in the disks manifest themselves, and the eyes become permanently blind.

The treatment consists in attention to the special disorder.

In *general anæmia* retinal and optic nerve changes are almost protean. Gowers[3] has seen neuritis in two chlorotic sisters suffering from amenorrhœa. Photo. VII., facing page 178, represents the appearance of the nerve-head in the first case, when the red corpuscles were sixty per cent. of normal, and the hæmoglobin thirty per cent. After one recurrence "the disks were perfectly normal," whilst the hæmoglobin had risen to eighty per cent. and the corpuscles to ninety-six per cent. Bitsch[4] reports a similar condition in a girl of sixteen. Becker[5] has observed spontaneous arterial pulsation in similar cases. As a rule, the blood-currents appear impoverished, allowing in some instances the current of an underlying vessel to be plainly recognized through the contained material and walls of the upper one. Both sides of the vessel are flat and broadened. Hemorrhages are rare.

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[1] A Treatise on Amaurosis, 1842, p. 36.

[2] See Norris's "Medical Ophthalmoscopy" in volume iv. of Pepper's System of Medicine.

[3] Medical Ophthalmoscopy, 1882, p. 328.

[4] Klinische Monatsblätter, 1879, iv. 144.

[5] Ibid., 1880, i. 1.

The prognosis is favorable.

The treatment consists in hygiene, nourishing and easily-digested foods, iron, etc.

The condition in *pernicious anæmia* is totally different. The changes are more pronounced. The disk is pallid and its edges are ofttimes obscured. The arteries are pale, reduced in size, and frequently wavy. The veins are broad, flat, and tortuous, and contain pale blood. Striated hemorrhages appear along the course of the vessels. This is excellently shown by the accompanying reproductions of Jennings's diagrammatic sketches showing the progressive development of hemorrhages in the left retina of a case of an eighteen-year-old patient seen under the care of Dr. Mackenzie.[1] The post-mortem examination left no doubt as to the genuineness of the diagnosis, although "the question might be raised whether syphilis should be blamed for originating the blood-disease."

The color of the entire fundus seems somewhat lighter than normal. Frequently, and more especially in the region of the nerve-entrance, there appear small whitish, irregular spots (lymphoid-cell agglomeration). Curious "irregularly round or ovoid hemorrhages with yellowish-white centres" have been seen by Norris.[2] These centres, according to Manz,[3] may either be composed of round, colorless cells enclosed in sacculations of the capillaries, or be the empty dilatation of the vessel-terminals themselves. Quincke[4] has seen both retinal œdema and stellate whitish opacities in the macular region. Marked optic neuritis has been found by Mackenzie.[5]

There is no special treatment for the ocular changes, except rest of the eyes. Attention should be given to the general condition.

In *leucocythæmia* the conditions noticed in the various forms of anæmia appear to be much aggravated, and in many instances may pass into actual inflammation of the retina. Not only has the choroid become lighter in tint and the retinal circulation more pallid in hue, giving the eye-ground an "orange-yellow" reflex,[6] but hemorrhages are extremely apt to take place. Associated with these changes, isolated white and yellowish thickenings, probably due to aggregations of escaped leucocytes, with localized degeneration of tissue, may be frequently seen in the periphery of the fundus and around the yellow spot. According to Gowers, in some instances their areas are "edged by a halo of extravasation." Œdema of the retina, with white patches in the adventitia of the vessels, has also been noticed. This is beautifully shown in the accompanying reproductions from Becker's most instructive case[7] in an adult female. The sketches were made nine weeks

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[1] Transactions of the Ophthalmological Society of the United Kingdom, 1881, p. 51.

[2] Pepper's System of Medicine, vol. iv.

[3] Gowers, Centralbl. für d. Med. Wiss., 1875, p. 675.

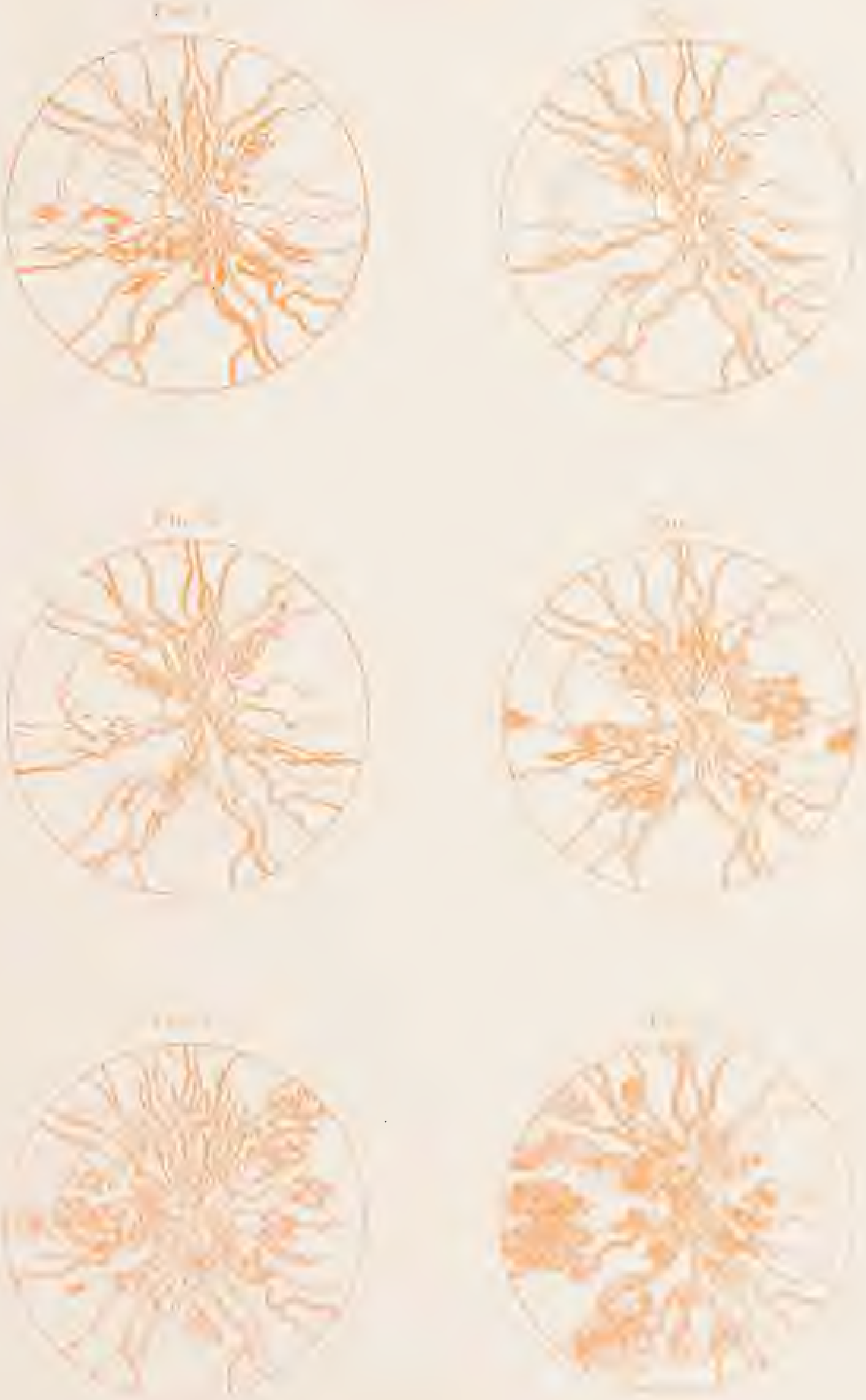
[4] Deutsches Archiv für Klin. Med., 1877, S. 1.

[5] The Lancet, December 7, 1887.

[6] See Liebreich, Deutsche Klinik, 1861, 50, and Becker, Archiv für Augenheilkunde und Ohrenheilkunde, 1869, 1, S. 951.

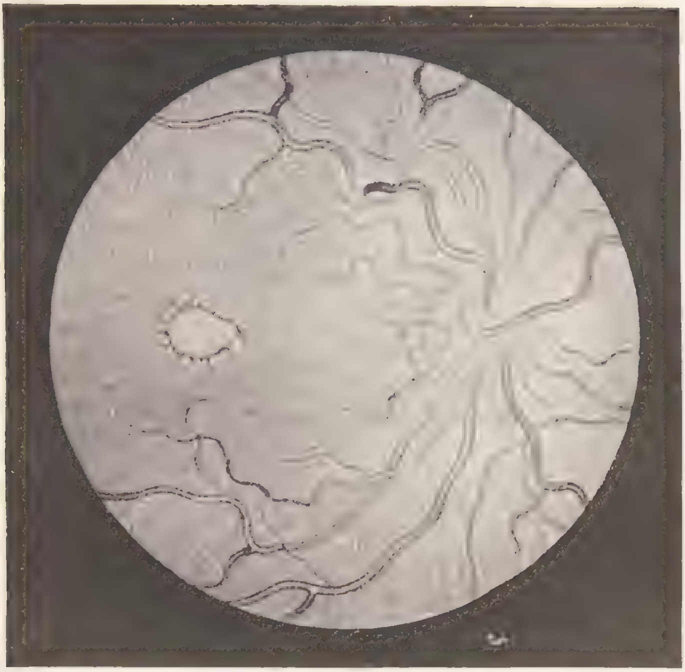
[7] Archives of Ophthalmology and Otology, vol. i. No. 1, p. 341.

DIAGRAMS.



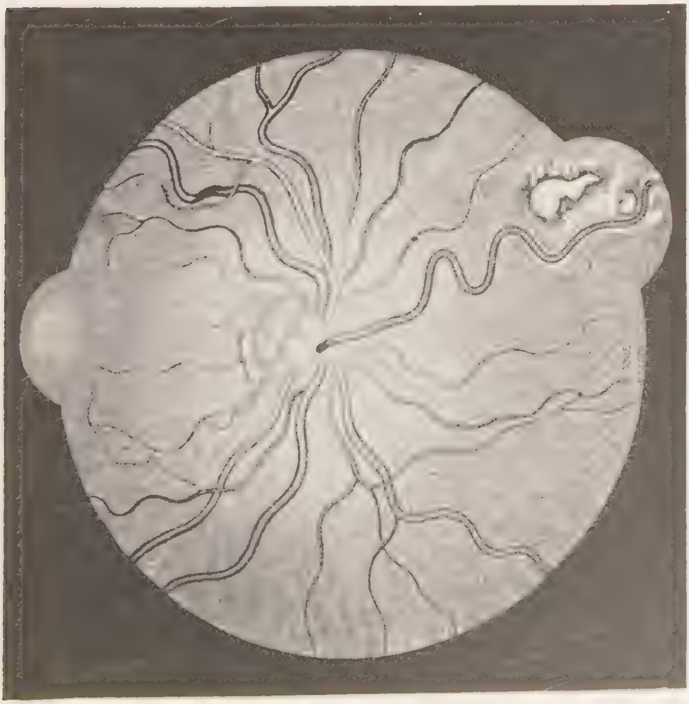
Progressive Development of Hemorrhages in Pernicious Anæmia. (Mackenzie, Transactions of the Ophthalmological Society of the United Kingdom, 1881.)

PHOTO. VIII.



Retinal Œdema and Vascular Opacities in Leucocythæmia. (Becker, Archives of Ophthalmology and Otology, vol. I., No. 1.)

PHOTO. IX.



Same Eye-Ground Nine Weeks later.

apart. Examination with the microscope in some of the cases has revealed a sclerosed and degenerate condition through the entire retinal structures. Through the kindness of Dr. John B. Shober and Mr. John Sailer (medical student) the writer has had opportunity to study the eye-grounds of a seven-year-old boy suffering from this form of disease. At the time of examination the child had been under treatment for several months, but, in spite of this, both the arteries and the veins of the retina were somewhat tortuous and the venous currents appeared slightly pallid.

Heinzel[1] has given a beautiful clinical picture of a case in a four-year-old child, in which, in addition to the other symptoms, there was intense swelling of the optic disks.

The functional ocular trouble, of course, does not demand any special medication, rest of the organs being all that is necessary. The treatment must be directed towards the general condition.

Purpura hæmorrhagica and *scurvy* may both give rise to hemorrhages into the retina. Lawford[2] gives an interesting case of double optic neuritis following purpura in a girl of twelve years. In the discussion, Nettleship thinks that the symptoms pointed towards a localized papillitis rather than towards a descending neuritis, though it is remarkable that the disk-changes were not by any means so marked, even at the climax. Quite a number of cases have been reported. The prognosis for vision is dependent upon the amount and the situation of the extravasation. No special treatment is required, the intraocular condition being best combated by attention to the general condition.

Lawford[3] gives a most instructive section of a portion of *choroid* included in an area *involved by a nævus;* this occurred in an eight-year-old girl whose left eye was enucleated for glaucomatous symptoms. The left side of the child's face was occupied by a large dull-red capillary nævus involving the eyelids; the bulbar conjunctiva was not implicated. In front of the opposite ear a small patch existed. Under the microscope the blood-vessels of the choroid could be seen throughout the whole thickness of the tunic.

Miller[4] gives a case of nævus of the right temporal and orbital region with nævus of the choroid associated with detachment of the retina in the right eye. Microscopically it appeared as a cavernous angioma. In this case, however, as Brailey remarks, it is curious that sequelæ of inflammation existed. A case of most probable venous angioma involving the skin of the right side of the face, the right sclerotic ("anterior ciliary"), and the retinal veins of the same side, occurring in a nine-year-old epileptic girl, is reported by Horrocks.[5] In connection, it is of interest that left hemiplegia, more marked in the upper extremity, existed, and that the convulsive seizures caused clonic spasm of the left side of the trunk and its limbs.

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[1] Archiv für Ophthalmologie, xxiv. 3, 241.

[2] Transactions of the Ophthalmological Society of the United Kingdom, 1882, p. 86.

[3] Ibid , v. 136.

[4] Loc. cit., iv. 168.

[5] Transactions of the Ophthalmological Society of the United Kingdom, iii. 106.

Local pulmonary disease cannot give any more than the ophthalmoscopic pictures indicative of disturbed vascularity.

LESIONS DEPENDENT UPON DISORDERS OF THE NERVOUS SYSTEM.

Brain and Envelopes.—In *cerebral anæmia,* according to Gowers,[1] permanent amaurosis, probably due to an affection of the retina, may occur in cases which have had loss of sight during the attack. During the tonic portion of an attack of epilepsy, which presumes a probable anæmic condition of cerebral substance, the fundus has been described as comparatively bloodless, the disk pallid, and the retinal vessels in a state of tonic spasm. Although this *a priori* is most certainly true, yet the chances for observation have been so few and the attendant difficulties so many that it will be necessary to observe a great number of cases before full credence can be placed in the few isolated and individual instances given.

In opposition to the findings in a number of well-observed cases, it is most probable that there is an increase of retinal circulation and a suffusion of the disk during the active stage of *cerebral hyperæmia.* Doubtful cases have been alluded to, but, as no distinctive changes of such a character have been accurately noted by competent observers, the subject must remain *sub judice* until a great number of similarly placed cases are associated in combined study. Extended and repeated ophthalmoscopic examinations of the fundus during ordinary syncopal attacks would go far towards a solution of this much-discussed problem.

When *cerebral hemorrhage* takes place, which, of course, is very rare in children, occurring, for instance, as in Marshall's case, during an attack of pertussis, not only may there arise defects in the visual field, due to cortical pressure in the occipital region or pressure upon some portion of the intracranial extension of the optic nerve and tract with subsequent degenerative changes, but true retinal hemorrhages may exist. These conditions, however, are relatively so infrequent that some authors give them but little place among the possible ocular symptoms in this disease.

Based upon well-grounded clinical studies, we can confidently assert that no time of life, except the first few months of infancy and the very oldest age, is exempt from the formation of *intracranial growths.* The great prevalence of recognizable cases in childhood and adolescence (with those of early and full maturity) can be well understood when we consider that at these times mental activity, with its necessary accompaniment of marked and oftentimes intense cerebral action, is at its greatest,—a condition in which the slightest pathological formation in the cerebrum would make itself known by the many objective and subjective changes so common in these disorders. Although it is safe to assume that the intraocular disturbance is usually more pronounced and of longer duration in childhood and infancy than it is in similar disease during the later periods of life, yet it is so much more frequently overlooked, by reason of the youth of the subject and the greater difficulty of ordinary examination in such cases, that statistics fail to give any higher percentage of occurrence. If careful ocular examination should be made in every suspected instance of cerebral disorder, much more light would be thrown upon the true character and significance of the case,—additional symptoms which might prove of inestimable value in the accurate determination of such diseases.

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[1] Medical Ophthalmoscopy, 1882, p. 123.

In childhood the tubercular, the gliomatous, and the sarcomatous types of intracranial growth are probably the most common; the first especially permitting the choroid and the retina to share in the deposition. That this is true, and that some growths are more prone to express their presence peripherally in the eye-ground than others, is distinctly shown by Starr.[1] He thinks, for instance, that as the gliomatous variety of tumor is very vascular and, as it were, erectile, variations in its disposition to erection mean corresponding increases in intracranial pressure, which in the majority of instances cause "sudden changes of intensity in the symptoms, accompanied by visible changes of circulation in the retina;" this intraocular condition being shown by such radical measures as "hot baths, cold douches to the spine, mustard baths to the feet, or free watery purgation." It is also quite probable that the cerebellar tumor is much more prevalent in childhood than in later life.

Gowers's opinion,[2] which is concurred in by Bramwell,[3] that optic neuritis is present in at least eighty per cent. of all cases, is most probably nearly correct, in spite of the higher ratios of Annuske[4] and Reich.[5] Whilst the condition seems to be more prevalent when the neoplasm either involves the cerebral base or is embedded in the substance of the brain-mass itself, yet cerebellar growths seem to be remarkably prone to give the most intense forms of optic neuritis. Almost all observers agree that the presence and the degree of optic-nerve swelling and inflammation are in direct ratio with the rapidity of development and the quickness of growth of the tumor, and are not dependent upon the size of the mass.

In the great majority of cases the optic neuritis is double. Sometimes, however, during the course of the disease, the acute conditions in one eye may subside sufficiently for a careless or an incompetent observer to assert that there has never been any previous inflammation in the nerve, and that the intraocular expression of the disorder is limited to but one organ; thus unfortunately invalidating much of the usefulness of the condition as a localizing symptom. As markedly illustrative of this, the writer has had the opportunity to watch the progress of a cerebral case for a long time, where a uniocular optic neuritis had been diagnosed, and yet where there were at times unequivocal symptoms of a low grade of neuro-retinitis in the fellow-eye, not only as shown by the ophthalmoscope, but also as evidenced by slight though pronounced contraction of the visual fields, with decided diminution of central vision for both form and color.

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[1] Medical News, January 12, 1889.

[2] Medical Ophthalmoscopy, 1882, p. 141.

[3] Intracranial Tumors, 1888, p. 64.

[4] Archiv für Ophthalmologie, xix. 3.

[5] Klinische Monatsblätter, 1874.

In those rare instances of true unilateral type, clinical investigation seems to point towards the opinions of Jackson,[1] Broadbent,[2] Pagenstecher,[3] and others, who have shown that the inflammation of the nerve is on the side opposite to the brain-lesion. Bramwell[4] doubts this, and is of opinion that the present number of such cases is "too small to allow of any very definite generalization being made." In partial confirmation of the statement of the former observers, the writer has recently seen two cases of double optic neuritis—both in children—in which the lesser degree of neuro-retinitis was upon the same side as the intracranial growth. A third case, also in a child, just studied at present writing with Dr. Morris J. Lewis, in which the choking of the left optic nerve was one diopter higher than its fellow, showed a tumor of the pons which was more marked on the right side. In further support, the writer has just had another curious ante-mortem coincidence in an adult, where in a yet unpublished case, seen in association with Dr. Francis X. Dercum, all the general and special localizing symptoms pointed towards a left-sided gross lesion. Here the right eye contained a large splotch-like hemorrhagic extravasation upon and around the optic disk, with an undue tortuosity of the retinal vessels, whilst the disks themselves appeared to be free from any coarse change. At the autopsy, a sarcomatous growth was found deeply embedded in the left cerebrum, involving the pulvinar and contiguous portions of the internal capsule and the striated body.

This can probably be best explained upon the supposition advanced by Broadbent in partial explanation of the monocular type of the optic neuritis, that the mechanical impediment to the passage of fluid into the intravaginal sheaths of the optic nerve by the growth is greater upon the affected side of the brain than it is upon the opposite.

It must be distinctly understood, however, that this rule can only be true when the optic-nerve extravasation has been secondary, as it were, to the great increase of cerebral bulk. An illustration of the opposite condition is to be found in Dr. James J. Putnam's[5] most interesting exception, where a sarcomatous tumor, involving the posterior half of the right middle frontal convolution, gave a much greater optic neuritis upon the same side as the tumor-mass. Here it is most probable that the extravasation of the increased cerebral fluid into the optic-nerve sheaths was quite early in the later history of the case, thus allowing the greatly augmented after-pressure from the sudden and excessive exacerbation of the tumor-growth upon the right side, at the time when first seen at the hospital, to block the previous extravasation, and push it more forward into the interior of the right eye than into the left eye, and thus give the greater intraocular swelling upon the right side. This supposition is strengthened by the post-mortem examination, at which "the surface of the right hemisphere was found pushed across the median line," "the base of the brain appeared normal, except that the optic tracts were excessively flattened by pressure," and "the right optic nerve was somewhat larger than the left, and reddish in color."

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[1] Royal London Ophthalmic Hospital Reports, vii. 573.

[2] Transactions of the Ophthalmological Society of the United Kingdom, p. 110.

[3] Royal London Ophthalmic Hospital Reports, vii. 130.

[4] Intracranial Tumors, 1888, p. 36.

[5] Boston Medical and Surgical Journal, April 10, 1890.

From the time of Von Graefe's suggestion[1] of increased intracranial pressure exerted upon the cavernous sinus, causing stasis in the ophthalmic vein,—a theory which was soon cast aside on account of anatomical reasons,—to the latest modifications by Bramwell[2] of the combined notions of Leber[3] and Deutschmann,[4] of the presence of irritant micro-organisms in the cerebro-spinal fluid produced by the neoplasm, which, passing down the intravaginal space of the optic nerve, produce peripheral inflammation, many theories have been evolved for the causation of the condition. Though Bramwell's assertion (*loc. cit.*) that "the pressure-irritation theory of Leber and Deutschmann is . . . the most likely explanation in the majority of cases," is most probably true in explanation of many instances, yet we must agree with the same author when he states, "I may at once say that, for my own part, I think it probable that the double optic-nerve neuritis of intracranial tumors is not always produced in the same manner." Besides, it must be distinctly understood that there are many similar instances of intracranial growths where, without any assignable cause of differentiation, optic neuritis is present in some and absent in others. Again, it must be remembered that there are examples on record of actual descending neuritis either in the contiguity of the nerve-tissue itself or in its trabeculæ. In fact, the subject is still *sub judice*, and not until we shall be in possession of a long series of carefully-made clinical histories in association with post-mortem examination of involved tissues by expert microscopists, combined with laboratory experimentation, will any legitimate generalization as to the causation of peripheral optic neuritis be possible.

No sharp divisions can be given as to the age of the neuritis by the apparent grossness of objective alteration. Neither can any decided difference between the ultimate result of two apparently diverse conditions be vouchsafed by the visible results alone. So much must be taken into consideration besides the ophthalmoscopic appearance, so many of the finer and almost imperceptible details of physical change seen must be carefully differentiated and understood by the ophthalmoscopist, and conditions vary so constantly in the same case, that numerous examples must be presented to the same trained eyes before adequate data of sufficient diagnostic importance and prognostic moment can be given.

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[1] Archiv für Ophthalmologie, vii. 2, 88.

[2] Intracranial Tumors, 1888, p. 67.

[3] Klinische Monatsblätter, 1868, S. 302.

[4] Leber, Neuritis Optica, 1887.

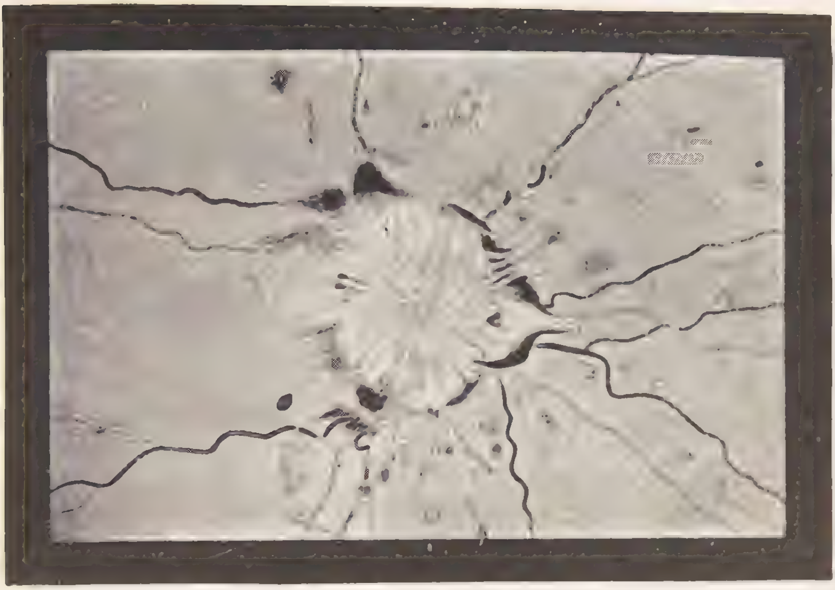
Roughly, a type may be made from which all manner of departure must be expected. Thus, for example, in either a slight attack or in the incipiency of a more pronounced one, the nerve-head will appear somewhat hazy; the scleral ring is fainter and at times covered with a coarse thickening of the retinal fibres,—this latter condition being more pronounced to the inner, upper, and lower borders of the nerve. Ofttimes the retinal arteries will be a trifle too wavy, and possibly almost imperceptibly contracted near the disk, whilst the corresponding veins will be more than ordinarily tortuous, and will carry rather darker blood than usual.

If the case be more pronounced, the disk will be actually swollen and pushed forward into the vitreous, the greatest amount of swelling being noticed in the upper, inner, and lower portions of the papilla; the adjacent retinal substance will be prominent, and situated upon a higher level than that of the periphery of the membrane; the scleral ring, with the adjoining pigment-massings so commonly seen to its inner and outer borders, will be absolutely lost to view beneath the swollen substance; the retinal veins will be markedly tortuous, enlarged, and lost to view at many points, in and just beyond their passage into the nerve; the corresponding arteries will appear greatly contracted whilst dipping in and out of the swollen tissues; dark venous blood, and at times pallid arterial currents, are seen in the twisted and partially-blocked blood-channels; fine striated hemorrhages, fan-like and flame-shaped,—especially at the bifurcation of the retinal vessels,—come and go, whilst small vessels which remain unapparent in health become plainly visible. This can be well seen in the accompanying phototypes, reproduced from Gowers's "Ophthalmoscopy" (2d edition, p. 359).

When the active conditions have ceased, the involved tissues slowly pass into a state of quiescence. The nerve-swelling gradually decreases, the outer or temporal borders being those that are first seen. The retinal prominence lowers, carrying the retinal vessels to their proper levels. The vessels themselves become more nearly normal in appearance, and the blood extravasations slowly fade away. This can be plainly seen in the second picture (*loc. cit.*). Should, however, more contraction of tissue take place, the nerve-material will still further squeeze the retinal circulation in its grasp; the retinal vessels, especially the arteries, will diminish more markedly, the borders of the disk will become more or less pronounced, and the surface of the nerve will sink into irregular mouldings. The third sketch (*loc. cit.*) well illustrates this. Should the case still progress, the changes of degeneration will become more and more manifest to the ophthalmoscope, until at last, in not a few cases, so-called "total atrophy" will take place.

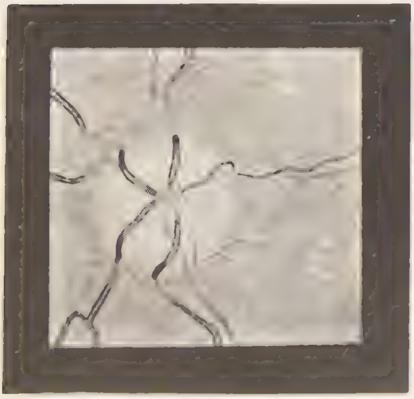
As has been abundantly proved by most competent and painstaking

PHOTO. X.



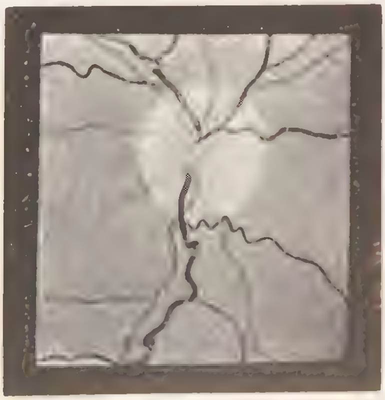
Neuro-Retinitis of Intracranial Tumor. (Gowers, Medical Ophthalmoscopy, 1882.)

PHOTO. XI.



Partial Post-Neuritic Atrophy. (Gowers, Medical Ophthalmoscopy, 1882.)

PHOTO. XII.



Regressive Neuro-Retinitis of Intracranial Tumor. (Gowers, Medical Ophthalmoscopy, 1882.)

observers, no dependence can be placed upon the state of vision as an exact index of the amount of disturbance. Berry,[1] Bramwell,[2] and others have all reported "good," "perfect," and "normal" vision in cases where the ophthalmoscope has shown intense neuro-retinitis. Hughlings Jackson[3] has seen instances where momentary attacks of blindness have been asserted,—these most probably the result of momentary increases of pressure. During the course of a neuritis a rapid permanent failure of sight in a few hours' time has even been noticed. Mackenzie[4] broadly asserts that he "would go so far as to say that in the practice of physicians who examine *all* their cases with the ophthalmoscope, whether the case was a cerebral one or otherwise,—whether there were or were not ocular symptoms,—in at least one-half, if not more, of the cases in which optic neuritis was discovered, it would be found unassociated with any marked, and often without appreciable, defect of sight."

As an example of one of the more constant objective symptoms of intracranial neoplasm, optic neuritis becomes one of the most valuable and important to the clinician. Usually unequally bilateral, it is almost certain to appear in some stage of the disease. Taken alone, not much dependence can be placed upon it as a localizing symptom. In conjunction with other ocular groupings and general motor and sensory disturbances, it becomes invaluable.[5] Again, we must remember that, as Hughlings Jackson states,[6] "optic neuritis points to the general nature of the local disease, not to its particular nature." It indicates, as he most pertinently says, the presence of a "foreign body" alone. Moreover, we can understand that the exact position of the mass cannot be determined from this symptom alone, because, as we well know, a foreign body, rapidly growing, will cause both direct and indirect pressure, with all their results, no matter in what intracranial point it may be situated. Certain it is that the nearer the mass is to the large fluid cavities and their intercommunications, the more certainly are we to have peripheral expressions of mechanical interference, whilst the less removed the neoplasm is from the associated intracranial tissues of the second nerve and its internal prolongation, the more certainly must we expect to have results of pressure and even destructive change: thus, roughly, cerebellar, deeply-seated cerebral and basilar growths seem the most prone to produce optic neuritis.

The fact that increased intraocular pressure has time and again been shown not to be the sole cause of optic neuritis does not in any way militate against these views. It cannot be denied that in some cases of neoplastic formation with pronounced destructive changes there can be an actual descent of the inflammatory material along the tissues of the nerve itself. Travelling by means of the meninges along the arterioles, and at last reaching and inflaming the optic-nerve connective-tissue material itself, the intraocular expression of optic neuritis is obtained. Both Brailey and Edmunds have distinctly proved this.[1] Still further, Edmunds[2] believes that it is a coexistent basilar meningitis from the brain-tumor which plays the *rôle* of causation of the double optic neuritis in such cases.

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[1] Diseases of the Eye, 1889, p. 301.

[2] Intracranial Tumors, 1888, p. 38.

[3] Transactions of the Ophthalmological Society of the United Kingdom, i. 70.

[4] Ibid., i. 95.

[5] True as this may be in the main, it is equally certain that cases of double optic neuritis in association with other seemingly focal symptoms have been recorded where post-mortem examination has failed to reveal any gross intracranial lesion. Fagge furnishes us with a most instructive example. (See Bramwell, Intraocular Tumors, 1888, p 42.)

[6] Transactions of the Ophthalmological Society of the United Kingdom, 1881, p. 82.

Secondary optic atrophy is most probably produced by either direct or indirect pressure of the neoplastic formation upon some intracranial portion of the optic nerves themselves, or their prolongations backward.

Prognosis.—If vision be momentarily impaired, as we sometimes find, especially in the so-called "choked disk" variety of optic neuritis, the ultimate visual result is always in direct relation with the amount of inflammatory tissue-change or degeneration, which lessens physiological action to a degree in proportion with the amount of nerve-tissue left after the primary gain from the original amount of œdema has been effected; that is, there is a marked primary loss of physical action, the result of the initial changes, immediately followed by a gain of perception dependent upon the amount of restoration of physiological ability of the diseased nerve-tissue. Following this gain, there is a slowly-decreasing vision, the result of post-neuritic change, the amount of the final sight being dependent upon the degree of consecutive inflammation or degenerative change. Where the neuritis has taken place without previous choking, the vision gradually fades from the beginning without any intermediate gain, only to stop at a point which indicates cessation of the optic-nerve change. If the case be one of simple atrophy (either primary or secondary)[3] from the first, the gradual failure of vision is much more steady, and, as a rule, more pronounced.

As *treatment* of the neoplasm by drugs is eminently unsatisfactory, and as the recent advances in cerebral localization with the brilliant results of antiseptic surgery render operative procedure for the removal of intracranial tumors so justifiable in many of the cases of accessible growths, careful study for such treatment should always be instituted. If syphilis be the suspected source of the growth, a proper course of alteratives with sarsaparilla, as Wood[4] suggests, should be tried. Should the mass be inaccessible, morphine injections, local applications of cold, as recommended by Bramwell,[5] or free watery purgation, as spoken favorably of by the same author,[1] may be used. Of course, local treatment of the optic neuritis itself—one of the effects—is out of the question.

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[1] Transactions of the Ophthalmological Society of the United Kingdom, 1881, p. 112 *et seq.*

[2] Ibid.

[3] For definition of these terms see page 128 *et seq.* of vol. ii. of Gowers's Manual of Diseases of the Nervous System, 1888.

[4] Pepper's System of Medicine, v. 117.

[5] Edinburgh Medical Journal, 1879, p. 1073.

As *cerebral abscess* is generally the sequel of suppurative processes about the bones of the ear, it is usually situated, as shown by Barr,[2] in the temporo-sphenoidal lobe of the same side. Cases of traumatic abscess, acute localized meningitis with pus-formation, and metastatic abscesses have been reported by Harrison,[3] Barker,[4] and Fraenkel.[5] Extension from nasal and orbital disease has also been seen. It is not uninteresting, therefore, in this connection, to note Keen's words:[6] "The presence or absence of choked disk does not seem to be pathognomonic, as it is sometimes present and sometimes absent. When present, even if bilateral, it is almost always more marked on the side of the lesion, though this is sometimes reversed." Thus associated with ear-disease it at times affords an important clue to the form of intracranial lesion.

Though generally a disease of adolescence and early adult life, yet the proportion of occurrence in childhood is so high that its presence should be carefully considered in every case of neuro-retinitis where there is a probability of intracranial mischief. It is certain that if the pus-formation be rapid and extensive, marked pressure will soon ensue, and give rise to optic neuritis. As distinguished from that of tumor, the optic neuritis of abscess is usually not so marked, is more disposed to be unilateral, and is, as a rule, more decided in its onset. When the abscess is situated posteriorly, the intraocular symptoms are generally wanting. As Keen says (*supra*), "Optic neuritis is not frequent in cerebellar abscess." Pflüger,[7] however, has seen one case of abscess of the left hemisphere of the cerebellum in which double optic neuritis with both optic nerve and retinal hemorrhages existed.

As vascular disease productive of thinning of vessel-walls is almost essentially a disease of adult life, *intracranial aneurism* as one of the results is almost unique in children. For this reason intraocular change expressive of the condition is comparatively unknown and seldom recorded. "Probably an ophthalmoscopic examination," as Gowers says,[8] "would have revealed it in a larger proportion of cases." Michel[9] reports a double optic neuritis from pressure of a varix-like aneurism of the two internal carotids. Mitchell gives[10] a most interesting case in an adult of an aneurism of an anomalous artery, causing antero-posterior division of the chiasm of the optic nerves and producing bitemporal hemianopsia. The eye-grounds, which were studied by Dr. William Thomson, showed "no changes except at each papilla, where the vessels of the retina appeared perhaps somewhat attenuated, whilst the neuroglia, especially of the left eye, was pale, the porus opticus enlarged, and the appearances those of partial atrophy. There was no swelling of either papilla, nor any change in the retina that would indicate a previously 'choked disk.'"

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[1] Edinburgh Medical Journal, 1877, p. 688.

[2] British Medical Journal, 1887, i. 723.

[3] Ibid., April 21, 1888.

[4] Ibid., April 14, 1888.

[5] Deutsche Medicinische Wochenschrift, No. 18, 1887.

[6] Reference Hand-Book of the Medical Sciences, vol. iii.

[7] Archiv für Ophthalmologie, 1878, ii. 171.

[8] A Manual of Diseases of the Nervous System, 1888, p. 497.

[9] Archiv für Ophthalmologie, xxxiii. 2, S. 225.

[10] Journal of Nervous and Mental Diseases, January, 1889.

In either instance, whether the case be one of actual nerve-inflammation, as in Michel's case, or of secondary nerve-changes from pressure, as in Mitchell's case, increased or continued growth of the aneurismal dilatation must produce degenerative changes in the optic nerve.

The prognosis as to sight is truly grave, and check of loss of visual power can only be expected from some radical surgical or manipulative procedure addressed to the main vessel or trunk itself.

As the increase of the secretion of the choroid plexus in the lateral ventricles, etc., taking place in *simple internal hydrocephalus*, becomes very pronounced, there may be some dilatation in the calibre of the retinal veins; this, however, upon account of the comparative elasticity of the bony and cartilaginous structures and the yielding of the cranial sutures to internal pressure, is quite infrequent. In a few instances atrophic nerve-changes have taken place with and without signs of previous neuritis; the latter being most probably dependent upon pressure upon the internal prolongations of the optic nerves themselves. In an acquired case in a three-and-a-half-year-old boy seen with Dr. W. W. Keen, the writer found a slight diminution of the retinal arteries with a corresponding enlargement of the retinal veins; the nerve-substance being entirely too gray for the age of the patient. In this case there were no gross evidences of past optic neuritis.

Pachymeningitis, either external or internal, is so excessively rare in children that no unquestionable case of consequent optic-nerve inflammation, as far as the writer's observation goes, is on record. This can be well understood when it is considered that small localized inflammations of the dura mater may be so trifling in their indirect results upon contiguous tissue, and exert such a minor degree of increased pressure, that neither marked inflammatory extension nor occlusion of outgoing lymph-channels by pathogenic material may ever take place during the course of the disease. If present, it is probable that in most instances it has been caused by direct implication of the optic nerve anterior to the chiasm, thus giving rise to the uniocular form of intraocular inflammation or degeneration. In association with Dr. Charles H. Burnett the writer has studied one such probable case in an adult.[1]

In *hæmatoma of the dura mater*, its occurrence is rather more often noted, not only upon account of the slightly greater frequency of the disease in childhood, but also because in these cases there is more changeability in the foreign and offending material. Optic neuritis may thus occur early, with the appearance of other acute symptoms, especially if the formation of the clot be rapid and extensive.

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[1] American Journal of the Medical Sciences, January, 1884.

Acute lepto-meningitis of all forms, which is so common in children, is more prone to give ophthalmoscopic signs of its presence when the membrane is inflamed at the cerebral base. Its form of optic-nerve disturbance may be either some variety of descending neuritis, as regarded by Von Graefe, or even perineuritis, as has been described by both Alt and Von Ziemssen, though so-called "papillitis" has been seen and noted by competent observers.

As distinguished from the optic neuritis of brain-tumor, Bramwell[1] believes that "the papillitis associated with tumor is, as a rule, more intense than that due to meningitis;" whilst Hughlings Jackson thinks that the swelling of the disk in tubercular meningitis is slight and not extensive, the disk appearing succulent. Gowers[2] says that the swollen nerve-head is paler than that which is usually found in similar conditions. In the few cases observed by the writer the optic disk appeared somewhat swollen, its edges were hazy, and the venous engorgement was slight; the retinal hemorrhages ordinarily seen in optic neuritis being but once present. Occasionally, whitish areas with white points can be noticed in the retina.

In the *tubercular variety* (see section on Tuberculosis) aggregations of tubers may be observed in the choroid. With the exception of the purulent type of meningeal inflammation, where intraocular evidences of thrombi and emboli may appear in addition to the neuritis, this form of the disease is most apt to give the most pronounced picture of nerve-head change; this, no doubt, being in a measure due to intracranial aggregations of tubercles, which thus practically form tumors themselves. By careful and periodical search, Garlick[3] ascertained its presence in twenty-one out of twenty-six cases.

It is probable that were all cases of meningitis carefully examined ophthalmoscopically during the attack, many of the cases coming to the ophthalmologist later in life, with a history of past symptoms of cerebral disorder and consequent defect of sight, would show ophthalmoscopic signs of optic neuritis. In the chronic form of the disease the disks may become more and more atrophic after each subsequent slight exacerbation. In this case the value of the symptom is very great, as the disease may be so insidious, and its general symptoms so masked, that it is difficult to obtain any certainty as to the correct diagnosis. If the child be old enough, periodical examination of vision and the fundus oculi, with careful perimetric study, should be made, especially if any vague general symptoms indicative of the disorder appear from time to time.

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[1] Intracranial Tumors, 1888, p. 45.

[2] Medical Ophthalmoscopy, 1882, p. 150.

[3] Medico-Chirurgical Transactions, 1879, p. 411.

In the *syphilitic form*, more especially in infants, where the region of the posterior fossa is invaded and internal hydrocephalus from intraventricular closure takes place, ophthalmoscopic signs of optic neuritis should be searched for in every instance. It is possible that Case II. of Hutchinson's clinical studies, "Amaurosis with White Atrophy of the Optic Nerves in Connection with Inherited Syphilis,"[1] belongs to this category.

In the *epidemic cerebro-spinal variety of meningitis*, optic neuritis appears late in the disease (about the fourth day), possibly partly owing to the comparative invulnerability of the larger nerve-bundles to either peripheral neuritis[2] or descending neuritis itself. After the primary attack of inflammation, the nerve rapidly passes into a state of consecutive atrophy; this is most probably due to the passage of a specific form of blood-poison acting so as to produce localized inflammatory reaction, which is often distinctly proved by the post-mortem appearances of purulent depositions in the uveal tract and the presence of thrombi and embolic infarcts in the veins of the retina. Both retinal and ciliary-body inflammation have been seen by Oeller.[3]

The grave form of the disease, where ophthalmoscopic changes are to be expected, is so rapid in its termination, and the patient is usually so restless, that it is often difficult, even when the pupils become dilated, to obtain any satisfactory view of the fundus oculi. In all such cases some mydriatic, such as homatropine or cocaine, should be employed. If the physician be at all expert, he will be able readily to examine the eye-ground whilst the patient is in a supine position.[4]

If the media be sufficiently clear, the eye-ground generally gives all the conditions of the lodgement of emboli or thrombi to a greater or less degree.

In spite of these conditions, cases have been reported where useful vision has remained, and where consecutive atrophic changes have stopped sufficiently early to prevent total annihilation of sight.

In *insolation*, or *thermic fever*, while meningitis (even in the young) has been brought on by the direct action of heat caused by paralysis of either the heat-centres or the vaso-motor nerves through increased stimulus from exposure of the body to increased temperature,[5] both optic neuritis and consecutive atrophy may be seen. Hotz,[6] who reports several such cases, goes further than this, in asserting that choroiditis of exudative type has been seen, its presence being due to extension of inflammatory material along the optic-nerve sheath. As Gowers,[7] however, says, "the absence of choroiditis in other cases of such extension renders the explanation difficult to accept." Fortunately, the disease is very rarely, if ever, seen in children.

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[1] A Clinical Memoir on Certain Diseases of the Eye and Ear consequent on Inherited Syphilis, 1863, p. 164.

[2] See article by Mills, Medical News, March 3, 1888.

[3] Archiv für Augenkrankheiten, 1878, S. 357.

[4] See Seguin upon this subject, Annual of the Universal Medical Sciences, 1889, ii. 59.

[5] See Thermic Fever, or Sunstroke, by H. C. Wood, Jr., M.D., 1872, p. 102.

[6] American Journal of the Medical Sciences, July, 1879, p. 105.

[7] Medical Ophthalmoscopy, 1882, p. 161.

Insular sclerosis, an affection of every age of life, though more particularly found among young adults, is a disease which is very apt to attack the second or optic nerve. Here we should expect to find degrees of visual loss of power in exact relationship with the number and the extent of the sclerotic patches in the sensory portions of the visual apparatus. Curiously, however, as Gowers[1] says, "the nerve-fibres passing through are not destroyed, their axis cylinders persist, and retain impaired functional power, although their medullary sheath may disappear." If the patch be situated far back, impairment of sight, followed by secondary changes which may become visible ophthalmoscopically, can take place. Should the islet of sclerosis be near the ocular globe, inflammatory signs in the region of the disk may manifest themselves. Usually both nerves are affected, though, as a rule, they are disturbed unevenly.

As nearly fifty per cent. of the cases of *migraine*, or hemicrania, are found during the period of adolescence, and as its subjective ocular symptoms, which are so numerous, varied, and frequent, simulate those of serious organic disease, it has been thought well to give them in some detail. Temporary hemianopsia of a thin fog-like area which slowly sweeps across the lateral fields of vision, dense central spots which extend peripherally and lose their central density, and "large dim specks which either move laterally or diagonally,"[2] generally either usher in the visual manifestations or mark the termination of a series of spectral appearances. At times there are vague impressions of moving water, or the appearance as if "the air itself were somehow visible, being composed of fine luminous grains which do not obstruct the vision" (*loc. cit.*). If the hyperæsthesiæ occur first, white or light zigzags, revolving concentric wheels, changing and blending colors,—in fact, as one patient says, "all manner of kaleidoscopic forms and colors that will not hold long enough to be examined,"—are visible.[3]

In children these conditions may be associated with considerable rise of temperature, which, upon account of the general subjectiveness of the symptoms, may ofttimes mislead one in the proper diagnosis of the case, and tend apparently to increase the gravity of the conditions.

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[1] Medical Ophthalmoscopy, 1882, p. 168.

[2] Extract from the notes of a private patient.

[3] This patient writes, "One curious thing, a sort of compensation for the discomfort of over-sensitiveness, is the vivid beauty of the impression made by colors which are rather tame to a healthy eye. Sometimes, curling up in a certain big chair after dinner, and going off in a five minutes' nap, my eyes on their first opening see in certain paintings on the wall some things which the artist probably aimed to say, but which do not always show; something in the first glance out of the window: harmonies of tints, depths and perspectives in the lights and shadows, as if we had caught Nature unawares and off guard. Even the first glimpse under such conditions of the gilt lettering on some books on the shelves, some of them pretty shabby, too, affects one with a positive thrill of pleasure at their beauty."

These, which are but a part of sensory disturbances, may at times be associated with motor derangements about the eye.

One peculiarity seen in a case of the writer's was that the intensity of the coloration of the spectra decreased and their character changed as the attacks became less frequent and less intense: the colors became less vivid, and changed to those which are ordinarily seen with less peripheral or external stimuli;[1] the forms became less pronounced, and the blind spots and blind areas less troublesome.

Be the pathology what it may, though most probably some change directly or indirectly connected with cortex irritation and disturbance, it is possible that even here visible retinal changes may exist which are so slight, and so masked by ametropic and other local conditions, that no characteristic eye-ground expressive of the disturbance can be noted. As the case advances, however, and may pass through the stage of chorea (?) into epilepsy (?), the fundus-oculi changes may become more and more marked, until at last the peculiarities of retinal fibre thickening with slight perivasculitis, vessel tortuosity, and incipient red-gray degeneration are finally established as the visible results of a process which has been taking place within the intracranial substance, and which may be readily seen post mortem by careful examination with the microscope.[2]

Stanford Morton[3] makes the curious observation of apparent obstruction of retinal circulation in the right eye of a young woman of twenty years who was suffering from migraine. No evidence of cardiac lesion was obtainable, though the patient had had chorea for several years. No mention is made of the existence of rheumatism.

Chorea.—In ordinary cases of chorea during childhood, fundus lesions have been generally regarded as negative: thus, Gowers[4] emphatically says, after describing three complicated cases where slight optic neuritis existed, "During the last twelve years I have examined with the ophthalmoscope a large number of cases of chorea, but have met with morbid changes in no other instance,"[5] and De Schweinitz in a later communication[1] presents the following as one of his conclusions: "Embolism, atrophy of the disk, and optic neuritis may occur during or after attacks of chorea, but appearances in the fundus oculi characteristic of the disease have not been found."

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[1] In this connection, though not exactly akin, it is of extreme interest to note the changes of color seen by Dr. S. Weir Mitchell's patient (quoted in Dr. Edward H. Clarke's unfinished essay, "Visions: A Study of False Sight (Pseudopia)," 1878, p. 246), who during a severe illness had a series of "visions." "Bright green" changed to "brown," and "vivid red" became "black," as her general symptoms subsided.

[2] Philadelphia Medical Times, February 5, 1887.

[3] Ophthalmic Review, March, 1890. [4] Medical Ophthalmoscopy, 1882, p. 172.

[5] On page 558 of his "Manual of Diseases of the Nervous System," 1888, he uses these words: "In most cases of chorea the ophthalmoscopic appearances are those of health. In a few there is optic neuritis, usually slight in degree, just enough to be unequivocal. It passes away when the chorea is over. In only one case have I seen considerable neuritis, comparable to that seen in a case of tumor: the inflammation passed entirely away with the chorea. It is probable that the neuritis is related to the cause of the chorea rather than directly to the morbid process in the brain. Many of the patients had considerable hypermetropia, and it is known that this condition disposes to slight neuritic changes in the disks, and may aid other influences in leading to the change."

That these conclusions are true there can be no doubt, but curiously, in addition, in several cases where, through the kindness of Dr. Wharton Sinkler and others, the writer had opportunity afforded him to study the eye-grounds of such cases, the fundus in nearly every instance examined presented an appearance simulating, though to a minor degree, that seen in the eye-ground of children of the same age who were suffering from epileptic seizures, these fundus-changes being associated with a doubtful blood-pallor, and seemingly so pronounced and fixed, even after correction of refraction-error, as not to be wholly explained by any existent ametropia. This coincidence, for at present it must be so considered (since insufficient material has as yet been seen from which to draw any positive conclusion), confirms the writer in the belief in the close relationship—if he may so term it—between migraine, chorea, epilepsy, and general paralysis of the insane (temporary and permanent cortex irritation, disintegration, and death).

Another curious fact in these cases, not exactly appropriate here, but which the writer intends to explain more fully in a more suitable place, is a decided clonicism of the ciliary muscle in a few cases, giving rise to apparent momentary increases of refraction.[2]

The two cases of embolus of the central artery of the retina seen by Swanzy[3] and Foerster (quoted by Gowers[4]) can be possibly explained upon the supposition of cardiac disease, which has been found to be so prevalent in such cases. Sym's case[5] probably belongs in this category.

For the relief of any eye-strain we should see that every ametropic child has its refraction-error thoroughly corrected by the use of a mydriatic, taking care to know that both the lenses and their mountings are properly and carefully readjusted every few months. The Weir Mitchell plan of rest-cure may be made of great use in these cases, together with short séances of judicious movements "slowly done *with force and completely finished*," as suggested by Seguin.[6]

In spite of Gowers's belief[7] that "the appearance of the fundus oculi between the paroxysms is, as a rule, normal"[8] in *epilepsy*, yet it is absolutely certain that if a series of careful observations be made in any variety of this disease, a type of eye-ground will soon manifest itself to the observer, this being the more pronounced in those cases where the seizures have taken place over a long period. Not only has the peculiar condition been found among adults, but it is plainly manifest in younger subjects.[1]

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[1] New York Medical Journal, June 23, 1888.

[2] Momentary lessening of hypermetropia, reversal into myopia, and increases of myopia, through all grades and amounts of astigmatism.

[3] Royal London Ophthalmic Hospital Reports, viii. 181.

[4] Medical Ophthalmoscopy, 1882, p. 171.

[5] Edinburgh Medical Journal, March, 1888.

[6] New York Medical Journal, April 5, 1890.

[7] Medical Ophthalmoscopy, 1882, p. 172.

[8] Further on in the text, the author modifies this statement by the following words: "The only deviation from the normal state of the fundus which has seemed to me frequent

The nerve-head appears dull red-gray, whilst its edges are hazy and hidden above and below by a fine (sometimes coarse) retinal striation which extends far out into the periphery of the fundus. Both the arteries and the veins of the retina are tortuous and a trifle large in size, whilst the vessel-walls appear thickened. The choroid itself seems somewhat disturbed. This picture is so constant in varying degrees, and is seemingly so uniformly in direct relation with the gravity and the number of the paroxysms, that it can be only considered as the visible ophthalmoscopic result of a similar condition in the related cerebral cortex, which may be seen post mortem in these subjects by careful study with the microscope.[2] Knies[3] has since found identical changes in the retina and optic nerve. As most of these cases were idiopathic in type where post-mortem examination failed to reveal any gross macroscopic lesion of either the intracranial or the ocular contents, but where the microscope showed signs of cortex irritation and inflammation, with similar changes in the retina, it must be conceded that the slight and easily-overlooked intraocular expressions noted bore no relation whatever to the sequelæ of old syphilis, such as choroiditis, chorioretinitis, etc., or to the results of coarse organic lesion of the brain, such as neuro-retinitis, retinal hemorrhage, etc.

Moreover, Norris[4] says, "In several of the chronic cases which the writer has had an opportunity of examining, there has been a low grade of atrophy of the disks, with concentric limitation of the field of vision." In confirmation of this latter statement, the present writer, in an analysis of the ocular symptoms obtainable in epilepsy in the male adult,[5] has found the visual fields for form and color reduced to from one-third to one-twentieth of normal areas. Both Allbutt and Bouchut hold that the disks appear congested during the "interparoxysmal state." Kostl and Memetschek's assertion[6] of the comparative frequency of spontaneous venous pulsation

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is an unusual equality in size of the retinal arteries and veins. The latter are not, as a rule, larger than normal, and the arteries appear as if large from a lax state of wall."

[1] Through the kindness of Drs. Isaac N. Kerlin and A. W. Wilmarth, the present writer has been enabled for the past two years to engage in studies upon this and kindred subjects at the Pennsylvania Institution for Feeble-Minded Children at Elwyn, Pennsylvania. As soon as the records are sufficiently complete for proper generalization they will be published.

[2] See Second Annual Report of the Pathological Department of the State Hospital for the Insane for the South-eastern District of Pennsylvania, by Drs. Francis X. Dercum and Ida V. Reel. Published in Seventh Annual Report of the Hospital, 1886.

[3] La Semaine Médicale, June 13, 1888.

[4] Pepper's System of Medicine, vol. iv.

[5] Philadelphia Medical Times, February 5, 1887.

[6] Prager Vierteljahrschrift, SS. 106 u. 107.

has not been substantiated by so careful an observer as Gowers;[1] it is likewise certain that in a large number of notings of cases which have been zealously and painstakingly studied by the present writer there cannot be found a single record of such a symptom.

During the convulsive seizure, ophthalmoscopic examination is so difficult that diverse opinions have been sedulously contended for by various observers. Pallor has been the most frequent, as noted by Hughlings Jackson,[2] Schreiber,[3] and Arlidge,[4] whilst both congestion and pallor have been recognized by Allbutt.[5] In the tonic stage of the paroxysm Gowers[6] has noticed increase in both size and darkness of the retinal veins. In one case seen by the present writer, where a convulsive seizure ensued during the time that the eye-ground of an epileptic patient was being studied by the direct method, an imperfect view of the fundus oculi was obtained during the cyanotic stage, which not only revealed a questionable increase in size of the retinal vessels, but also gave a doubtful enlargement of the size of the entire details of the fundus; this, if correct, may be attributed to a possible tonic contraction of the ciliary muscle during the general tonic spasm, which produced a temporary increase in the convexity of the crystalline lens, with apparent enlargement of the ophthalmoscopic image,—a solution of a problem akin in a measure, though probably more plausible, to that suggested by Knies[7] to explain the apparent change in the size of the retinal vessels.[8] Finkelstein,[9] who has studied the fields of vision immediately following epileptic seizures, has found some very curious and interesting temporary changes in order, size, and perversion, and believes that these peculiarities, in association with some similar conditions of ordinary color-perception, may prove of value in the differential diagnosis of true and simulated epilepsy.

The eye-grounds in *idiocy*, which are now being studied by the writer, have in a considerable number of instances given characteristic congenital anomalies with pictures of products of inflammatory change.

The mental grade of *imbecility—i.e.*, the ability of the subject to make continued use of the eyes for prolonged near work—seems to bear greatly upon the condition or appearance of the fundus oculi. In the lower grades, where mentality is of such a character that the eyes are seldom, if ever, used, the fundus in most instances fails to present those changes, such as "dirty red-gray appearance of the optic disk; irregularity of physiological excavation; non-visibility of the superior and inferior portions of the scleral ring; absorbing conuses in all of their varieties; increase in density and thickening of the retinal fibres; opacities of the vascular lymph-sheaths; disturbed states of the choroid; and gross errors in astigmatism, with changes in indices of refraction,"[1] which are so ordinarily found in the abused eye of the mentally healthy at the same age.[2]

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[1] Medical Ophthalmoscopy, 1882, p. 173.

[2] Lancet, February 17, 1874.

[3] Ueber Veränderungen des Augenhintergrundes, 1878.

[4] West Riding Asylum Reports, i.

[5] On the Use of the Ophthalmoscope, 1871.

[6] Medical Ophthalmoscopy, 1882, p. 173.

[7] Sitzungsbericht der Heidelberg. Ophth. Gesellschaft, 1877.

[8] See Gowers's Medical Ophthalmoscopy, p. 174, ed. of 1882.

[9] Inaugural Dissertation, 1887.

The case is far different when the little patient has been placed in the highest grade of school training. Here not only do we see the abused tissues of the overused eye of childhood, but we find that the changes in refraction-error, with all the consequent fundus peculiarities, are much more prevalent and pronounced than among the mentally healthy placed under the same condition of school hygiene.

Spinal Cord and Coverings.—Contrary to common belief, acute inflammation of the spinal cord proper (*myelitis*) has most certainly at times eye-ground symptoms associated with it. Noyes[3] gives a remarkable instance in a young man. Steffen,[4] Erb,[5] Chisolm, and Seguin have all seen cases. The intraocular symptoms are described as low-grade optic neuritis, associated with all degrees of lowering of central vision and decided though changeable diminution in the visual fields.

Sharkey and Lawford[6] add a case of acute optic neuritis with acute inflammation of the spinal cord. Their case (in a seventeen-year-old girl), which happily embraced both an extended clinical history and a most careful study with the microscope of the involved tissues, showed intense inflammation of the optic disks, nerves, and chiasma, with a less involvement of the tracts, whilst the adjacent meninges gave slight evidences of inflammatory change.

According to some observers, *spinal concussion* has been productive of failure of vision. Allbutt,[7] who believes in an association between the two conditions, attributes the fundus-lesions to the effects of meningeal disturbance. Noyes[8] relates a case in an adult, where, after the receipt of a sudden and severe blow upon the lower end of the spine causing intense pain at the base of the skull and along the spine, there were defective vision and contracted fields. Ophthalmoscopically, "there was extreme hyperæmia of the optic disk both in the large and small vessels." He believes that a paralysis of the fibres of the sympathetic might well be assumed as the cause of the vascular dilatation. Erichsen[9] asserts that in the vast majority of cases unattended by fracture or dislocation there was distinct evidence of visual impairment. Examination of his cases shows that vision was impaired in about one-seventh of the total number. Necessarily this proportion must be accepted *cum grano salis*, upon account of the omission of careful ophthalmic examination in the greater number of his recorded and quoted instances.

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[1] See Transactions of the American Ophthalmological Society, 1887.

[2] This is true even in the adult imbecile of the same grade.

[3] Archives of Ophthalmology, 1880, ii. 199.

[4] Sitzungsbericht der Heidelberg. Ophthalmologischer Gesellschaft, 1879.

[5] Archiv für Psychiatrie, x. 146.

[6] Transactions of the Ophthalmological Society of the United Kingdom, i. 240.

[7] The Ophthalmoscope in Diseases of the Nervous System and of the Kidneys, 1871.

[8] A Text-Book on Diseases of the Eye, 1890, p. 631.

[9] On Railway and Other Injuries of the Nervous System, 1875.

The symptoms generally complained of are a species of hemeralopia, muscular asthenopia and insufficiency, and double sight, followed by muscæ volitantes and colored vision. Conjunctival congestion has also been noted. As all these conditions are subjective, care must be taken to ascertain that there is no malingering, especially in cases of young hysterical persons. Careful tests for all manner of deception, with close examination and consideration of every related ophthalmic symptom, should be made before any opinion is given as to the relation of doubtful cause and apparent effect. (See section on malingering.) Thorburn,[1] after a comparison of some fresh investigations with the past conclusions of others, says, "From the above summary we are led to the conclusion that the occurrence of optic neuritis is extremely rare in the cases formerly described as concussion of the spine, and that even when present there is no indication whatever that it bears any relationship to a lesion of the spinal cord." He has no faith[2] in the association of the intraocular disease with a doubtful spinal traumatism in Thorowgood's observation,[3] where "choked disks" appeared in a young healthy girl twelve years of age, one month after a blow received upon the lower part of the back.[4]

Philip C. Knapp[5] has written a most careful paper upon the whole subject.

The pathology and etiology of *Friedreich's ataxia* are still obscure. According to Friedreich,[6] Möbius,[7] Grasset,[8] and others, it is merely a species of tabes dorsalis caused by primary degeneration of the posterior columns of the cord with secondary meningitis. Bourneville[9] and Ross,[10] however, associate it with insular sclerosis, whilst Gowers[11] finds a correlation between it and ataxic paraplegia.

In this probably the only ordinary form of tabes dorsalis seen in childhood,[12] intraocular signs of sensory disturbance have, according to the most careful analyses of recorded cases by Griffith,[1] been very rarely seen, if at all. Out of one hundred and forty-three cases, he found ophthalmoscopic examination noted in thirty-eight instances, and in none of these were there any changes of importance except in Power's cases,[2] in which the disks were rather white, and in one of Seguin's,[3] in which there was "partial atrophy of both optic nerves." Continuing, he says, "The condition of the *color-fields* might prove of interest in showing the possible relation of the disease to locomotor ataxia. Very little has been done in this direction, though Oliver made a careful examination of one of Sinkler's cases[4] and found narrowed fields, leading him to believe that there existed ocular changes allied to those of tabes." In this instance, which was most carefully studied, the writer found incipient optic-nerve degeneration, as evidenced by the ophthalmoscope and shown by slight subnormal color-perception for green, with marked contraction of the visual fields, more pronounced on the left side. These sensory changes in association with a pronounced horizontal nystagmus induced him to conclude that he was dealing with ocular conditions which were related to some peculiar form of locomotor ataxia.

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[1] A Contribution to the Surgery of the Spinal Cord, 1889, p. 182.

[2] Op. cit., p. 178.

[3] Transactions of the Clinical Society of London, 1875, p. 80.

[4] Could disturbed menstrual function have been a factor in the production of the optic-nerve change?

[5] Boston Medical and Surgical Journal, November, 1888.

[6] Virchow's Archiv, 1888, xxvi.

[7] Schmidt's Jahrbücher, 1884.

[8] Traité pratique des Maladies du Système nerveux, 1881.

[9] Nouvelle Étude sur quelques Points de la Sclérose en Plaques disséminées, 1869.

[10] Hand-Book of the Diseases of the Nervous System, 1885, p. 527.

[11] A Manual of Diseases of the Nervous System, 1886.

[12] As an exception to this rule, see case at ten years of age by Eulenberg (Ross, Diseases of the Nervous System, p. 213). Another, from Erb's practice (Ziemssen's Cyclopædia, xiii. 524), is quoted by Sinkler (Medical News, July 4, 1885).

Through the kindness of Dr. J. P. Crozer Griffith, and with the assistance of Dr. H. W. Cattell, the writer has been able to study the ocular conditions of two of the personal cases mentioned in Dr. Griffith's "Contribution to the Study of Friedreich's Ataxia" (*supra*). In the case S. J. he was enabled to verify Dr. Turnbull's original statement, "The result of the ophthalmoscopic examination of the eye-ground . . . was entirely negative." In the third case (Annie C.), where Dr. Turnbull two years previously had reported "ischæmia of the retina and venous pulsation in both eyes, but no other alteration of the eye-grounds," there was decidedly less capillarity to the temporal portions of the optic-nerve substance, though the disks appeared otherwise healthy; this loss of capillarity being more pronounced in the left eye. Vision with the right eye was reduced to two-thirds of normal, whilst that with the left eye was but one-third of normal. Fields of vision for form were contracted to one-fifth (right) and one-sixth (left) respectively; there being a series of indentations in the periphery of the color areas, especially down and in. Tension was normal in each eye. Media were clear.

It would be both interesting and instructive to have a careful ophthalmoscopic record with a painstaking perimetric examination made in every case falling under observation, so that if the optic-nerve changes be constant they may be included in the symptomatology of the disease.

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[1] Transactions of the College of Physicians of Philadelphia, February 1, 1888.

[2] St. Bartholomew's Hospital Reports, 1882, p. 305.

[3] New York Medical Record, March 13, 1885, and British Medical and Surgical Journal, October 15, 1885.

[4] Philadelphia Medical News, July 4, 1885.

In *lateral sclerosis* ophthalmoscopic changes are said by Gowers[1] to be very rare. He nevertheless notes an instance of repeated transient attacks of amblyopia followed by optic-nerve atrophy; the symptoms appearing in an adult male whom he supposed to be suffering from the disease. In a nineteen-year-old girl seen by the writer through the courtesy of Dr. G. Betton Massey, repeated ophthalmoscopic examination extending over several years showed that the right optic nerve continued slightly the healthier in appearance[1] and gave somewhat better visual results through the entire period; though the visual acuity, color-perception, and visual fields of the left eye were always but slightly below normal.

Unclassified Neuroses.—As *hereditary optic-nerve atrophy* is significant of a form of lesion which most certainly appears to be associated with heredity, there can be no doubt of the correctness of the use of the term in this connection. As early as 1817 it was recognized by Beer,[2] who gave a detailed account of three generations in whom the females became blind about the time of menopause, and where, curiously, the brunettes of the family were those affected. Travers[3] instances another series in two generations, whilst Sedgwick[4] gives a most peculiar family grouping, where the blindness appeared at sixty in the father and where the sons became successively earlier and earlier affected. Habershon[5] gives an exhaustive account of the literature and a *résumé* of a large number of personal instances. Four generations of the disease are noted by Haswell.[6] Wardrop[7] says that he has "known several instances of this kind." Weller[8] cites an interesting example where an autopsy showed that the "medullary matter of [the optic] nerves had been completely removed." Thomsen's[9] case, which developed melancholia later in life, is interesting by reason of a supposed seventeen years' remission. Jacobson[10] attempts to explain the condition in a child born of a seemingly normal-eyed mother, as dependent upon an intra-uterine pathological process that had disturbed the already finished optic nerve. Leber (*loc. cit.*), who has given us a systematic study of the whole question, believes that a peculiar neuropathic tendency, such as neuralgias, dizziness, anæsthesias, and even epileptiform seizures, exists in all these cases. He thinks that it generally manifests itself a short time after adolescence, though it has been observed quite early in life. Consanguinity does not seem necessary, and in fact it is not usually found.

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[1] Medical Ophthalmoscopy, 1882, p. 168.

[2] Lehre von den Augenkrankheiten, etc., 1817, ii. 442.

[3] See Leber's paper, Archiv für Ophthalmologie, xvii. 2, S. 249.

[4] Medical Times and Gazette, March 22, 1862.

[5] Transactions of the Ophthalmological Society of the United Kingdom, viii. 190.

[6] British Medical Journal, December 3, 1887.

[7] Essays on the Morbid Anatomy of the Human Eye, 1818, ii. 189.

[8] Manual of the Diseases of the Human Eye, 1821, ii. 79.

[9] Münchener Medicinische Wochenschrift, March 27, 1888.

[10] Centralblatt für praktische Augenheilkunde, December, 1887.

Norris[1] has had the rare opportunity of making a most careful ophthalmoscopic study of seven cases (four boys and three girls), the children of an unaffected mother who was the sister of two affected brothers and one unaffected sister, the grandmother of this generation being affected. His youngest case appeared in a seven-year-old boy; the tendency, as shown in Sedgwick's series, being that the younger children should be attacked the earliest.

Most writers look upon the disease as one manifesting itself about the time of puberty, whilst others have failed to recognize it until later in life. Littell[2] notes an instance where "four or five children in one family were born blind, the parents themselves enjoying perfect vision."

The subjective symptoms are quite characteristic: more or less frontal headache; dizziness; attacks of "fogging of vision" during perspiration, without watering of the eyes; various-colored phosphenes (red and blue stars, etc.) appearing in the centre of the visual field; gradual diminution of power of central vision; ocular pain upon exposure to light; visual fields markedly and irregularly contracted, with varying sizes and densities of central scotomata; gradually decreasing normal color-perception, passing through the various colors, green, red, blue, and yellow, until at last nothing but equal intensities of color are laid side by side, without reference to tint.[3]

Ophthalmoscopically, the changes in the optic nerve which are so well described by Norris (*supra*) may be summed up as follows: "1. The stage of cloudy and œdematous swelling," where the disk is still capillary and hazy, its normal outlines partly or entirely hidden, and its substance slightly swollen. "2. That of lymph reflexes, where the haze and swelling have lessened, and the disk has become slightly grayer." In this stage there appear numerous silvery and yellowish-white reflexes situated in front of the retinal vessels, mostly marked in the macular region, which, according to the author, are "probably partly due to capillary vessels of the retina which have become visible by the thickening and clouding of their walls, partly to the enlargement of the lymph-channels of the retinal tissue." "3. That of general death of the nerve-tissue;" here the reflexes lessen, the retinal vessels dwindle, and the tissue of the disk gradually becomes atrophic and assumes a greenish tint.

It will thus be seen that its early recognition in children depends upon carefulness of clinical study and the understanding of slight but significant changes in the fundus. From what has been said, it is fair to assume that the disease is as truly progressive in its early stages as later on, when even almost incompetent study cannot fail to expose its existence. The child of seven or eight years is as truly a sufferer, and in need of as much hygiene and therapy to endeavor to prolong the life of one of its most valuable organs, as the man of fifty years. The child of three or five years belonging to such a family, even though not presenting any macroscopic signs of disease to our ordinary instruments of precision, is even more to be protected and cared for, in the hope of averting such a calamity, than the one of older years. The conditions seen in youth are just as indicative of this terrible heirloom as are the grosser lesions and their more dire consequences when found in the related adult. Early youth and infancy are the only times in the individual's life when we may hope by most careful and even extraordinary regimen and studied prophylaxis to mitigate or suppress the manifestation of this dread disorder. As each year in life is passed, the greater the changes become, and the less likelihood is there for repair or betterment of condition. What its true pathology may be—axial neuritis or vascular disturbance—autopsy will alone show. So far we are in the dark. Many theories might be proposed, each holding a grain of truth, but the knife and the microscope can alone give the answer.

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[1] Transactions of the American Ophthalmological Society, v. 662.

[2] Manual of the Diseases of the Eye, 1846, p. 215.

[3] This series of color-change and loss, which the writer has been so fortunate as to study in Dr. Norris's grouping of cases, is exceedingly interesting, and is of value, in the pathological sense at least, in the study of the evolution of the color-sense.

Strychnine, as advocated by Mooren, Leber, and Norris, probably acts, as the last-named author states,[1] by increasing arterial pressure, so as to give greater flow of nutriment through shrivelled optic-nerve capillaries.

Whether it be true or not that *exophthalmic goitre* has its pathogenesis in the cerebral mass,[2] yet according to Gowers[3] the fundus in this disease is, with the exception of arterial pulsation and œdema of the disk, usually quite normal; elsewhere[4] he says that "the retinal arteries participate in the general arterial dilatation which occurs so uniformly in the disease and is ascribed to a paralysis of the sympathetic vaso-motor fibres." Berry[5] asserts that in his experience "pulsation, at any rate, is much less frequent than is assumed by some writers on this subject, and than might perhaps be expected from the evident want of tone in the arteries of the head and the excited state of the heart's action." Norris[6] says that "ophthalmoscopic examination usually shows a slight thickening of the fibre-layer of the retina in and around the disk, with dilatation and tortuosity of the veins, a state of affairs which may often be fairly attributed to a venous stasis caused by the swelling tissues." He further remarks, "In addition to these symptoms, there is sometimes, as Becker has pointed out, a dilatation of the arteries, which may almost equal the veins in calibre. At times there is an arterial pulse." This arterial pulsation, which has been seen and described by Becker,[7] is both spontaneous and variable.

In an analysis of thirty-two cases of Graves's disease occurring at the

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[1] Transactions of the American Ophthalmological Society, 1882.

[2] See article by Leplaine, Gazette des Hôpitaux, 1889, No. 5.

[3] Manual of Diseases of the Nervous System, 1888, p. 811.

[4] Medical Ophthalmoscopy, 1882, p. 170.

[5] Diseases of the Eye, 1889, p. 386.

[6] Pepper's System of Medicine, vol. iv.

[7] Klinische Monatsblätter, January, 1880.

Manchester Royal Eye Hospital, Hill Griffith[1] found but three cases under twenty years,[2] in one of which, although the eye-grounds were designated as normal, with no arterial pulsation, yet the disks were hyperæmic. Spontaneous arterial pulsation was not found in a single case. West[3] denies any retinal changes "except some fulness of the veins in a few." Lang and Pringle[4] give a case in a fourteen-year-old boy whose fundus was said to be normal.

There can be no doubt that in the majority of cases, when carefully studied with the upright image, there can be detected a true venous stasis, associated with pronounced retinal striation. In some instances, where there is but little associated intraocular change, this venous tortuosity and dilatation may be fairly attributed to general vascular disturbance,—thus forming an intraocular guide, as it were, to the condition of the patient. In the notes of the few examples that the writer has seen, there is no record of spontaneous arterial pulse or enlargement of the arteries. These fundus-changes, of course, need no special treatment, as they merely represent some of the minor and least important symptoms in the disease. In fact, they form, as it were, a part of the natural history of the disorder.

Numerous cases of probable reflex sensory disturbance of the second nerve or its internal prolongation from *irritation or injury* of large adjacent nerve-trunks, such as the *trigeminus*, have been recorded. The writings of the older ophthalmologists, such as Beer,[5] Wardrop,[6] and Middlemore,[7] abound in curious instances. The last-named author reports a remarkable though questionable case.[8] De Wecker,[9] Hutchinson,[10] Widmark,[11] Galezowski,[12] and others relate instances, both in adults and among children, where amblyopia is supposed to have arisen from dental irritation.

All such cases should be most carefully studied before a diagnosis is vouchsafed. Accurate ophthalmoscopic records, with repeated examination of the visual fields and color and form perception, should be made in every suspected instance, so that more accurate data as to the ophthalmic groupings may be obtained. These studies, in association with careful exploration of the general system, may prove of the utmost value in determining a question which must remain undecided until all doubtful points of differential diagnosis have been definitely settled.

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[1] Transactions of the Ophthalmological Society of the United Kingdom, 1886, p. 60.

[2] Two cases only can be found in the table.

[3] Op. cit., p. 76.

[4] Op. cit., p. 108.

[5] Lehre von den Augenkrankheiten, etc., 1817, i. 195.

[6] Essays on the Morbid Anatomy of the Human Eye, 1818, ii. 179.

[7] Quoted by Lawrence in his Treatise on the Diseases of the Eye, American edition by Isaac Hays, 1843, p. 109.

[8] Treatise on Diseases of the Eye, ii. 364.

[9] Annales d'Oculistique, 1866.

[10] Royal London Ophthalmic Hospital Reports, iv. 381.

[11] Annales d'Oculistique, September–October, 1888.

[12] Revue Générale d'Ophtalmologie, October, 1888.

LESIONS DEPENDENT UPON DISORDERS ORIGINATING IN THE SECRETORY AND EXCRETORY SYSTEMS.

Temporary diminution of vision with an ophthalmoscopic picture of optic-nerve congestion has been found as a sequel of *parotitis*. Brundt[1] has noticed tortuosity of the veins of the retina. Noyes[2] quotes a very interesting case of optic-nerve tumor seen by Liddell in a young woman of twenty years, which first manifested itself by dimness of vision five months after a protracted attack of mumps, followed by exophthalmus and blindness six months later. "The patient was in good health five years later, with no return of the growth." Metastatic choroiditis is also said to follow the disease sometimes, this condition being most probably the result of embolic infarction with resultant localized inflammation.

Very rarely retinal changes indicative of embolism have been noted in association with *tonsillitis:* Von Graefe is said[3] to have once seen such a case.

Gastric hemorrhage, as, for instance, in Asiatic cholera or in any organic lesion of the stomach, may give rise to sudden loss of visual power, just as might occur in any other form of hemorrhagic dyscrasia. Galezowski[4] claims to have seen grave disturbance of vision following atrophic changes in the optic nerve from gastric troubles; asserting improvement of the ocular condition by attention to the stomachic disorder.

Hepatic disease, especially of the icteric type, not only produces conjunctival discoloration and xanthopsia from bile-pigment deposition, but may also give an ophthalmoscopic picture of apparent yellowish discoloration of the blood of the retinal and choroidal vessels. Jaeger has seen this latter condition, which Gowers believes to have been merely an appearance caused by the tint of the media. Both Junge[5] and Buchwald[6] have noted instances of retinal hemorrhages in organic disease of the liver: these hemorrhages are believed by Litten[7] to be present only in cases of jaundice.

Intestinal disease associated with general wasting and blood-impoverishment from local hemorrhages or profuse diarrhœa is at times productive of blindness, as noted in Ziegler's case[8] of a man who died after a severe duodenal hemorrhage, where ophthalmoscopic symptoms of thrombosis were seen; in Schweigger's instance,[9] where the optic disks were pale and "cloudy," followed by degenerative changes; and in Von Graefe's case[10] of increasing pallor of the nerve-head and lessening of retinal-artery calibre:

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[1] American Journal of the Medical Sciences, 1886.

[2] A Text-Book on Diseases of the Eye, 1890, p. 668.

[3] See Gowers, Manual and Atlas of Medical Ophthalmoscopy, 1882, p. 250.

[4] Journal d'Ophtalmologie, March, 1872.

[5] See Gesammelte Schriften (Müller), 1874.

[6] Berliner Klinische Wochenschrift, xvii.

[7] Deutsche Medicinische Wochenschrift, 1882, S. 179.

[8] Fortschritte der Medicin, 1887.

[9] Hand-Book of Ophthalmology, 1878, p. 544.

[10] Ergebnisse der Untersuchung mit dem Augenspiegel, 1876.

or it may be caused by irritants, such as impacted fæces, as seemingly, though doubtfully, shown in Wishart's case,[1] where a nine-year-old boy was made completely blind in the left eye for some months "by a loaded state of the bowels," the patient being cured by clearing the alimentary canal;[2] or by the presence of worms, as related by Vandermonde,[3] in which instance a girl is said to have lost both vision and speech, or, again, where strabismus and "amaurosis" were doubtfully due to the presence of ascaris lumbricoides, as in the case mentioned by Hogg.[4] It is also of interest to mention Immermann's adult case,[5] where a patient, believing himself to have a tape-worm, took such enormous doses of cathartics as to bring on exhaustion from excessive diarrhœa. There was no intestinal hemorrhage. At first ophthalmoscopic signs were negative, though optic atrophy soon manifested itself.

The blindness which rapidly takes place does not generally ensue until several days after the loss of blood: this fact militates very much against the belief in mere anæmia as the causative factor, and possibly suggests, as Von Graefe[6] taught, that it is in a measure dependent upon some disturbing process in the optic nerve itself. Moreover, as shown by the same author,[7] other signs of anæmia were not present in such cases. In fact, the whole subject is still *sub judice*, and until more accurate clinical studies have been made, with proper post-mortem examination, answer must be looked for not only in the vascular but also in the nervous system.

As can be readily understood, no special treatment is of any value, except in the prevention of rare local complication. All efforts should be directed towards the amelioration of the general condition and the removal of the exciting cause.

In many cases of *organic renal disease*, no matter of what variety, both the choroid and the retina share in the general vascular disturbance found: especially is this true of the interstitial form of inflammation. While it is certain that the cirrhotic kidney is eminently a disease of middle adult age, it must not be forgotten that instances of this condition have been reported in the young. Moreover, as Tyson[8] has shown the powerful influence of heredity as a causative factor, and cites a remarkable grouping of related cases, in which it is noted that not only a twenty-year-old brother of his patient had Bright's disease for six years, but also that two children of another brother had the disease when respectively four and seven years of age, this factor must not be forgotten.

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[1] Edinburgh Medical and Surgical Journal, xxiv. 64.

[2] Hocken, A Treatise on Amaurosis, 1842, p. 127.

[3] Journal de Médecine, tome x.

[4] British Medical Journal, July 21, 1888.

[5] Deutsche Medicinische Wochenschrift, 1887.

[6] Archiv für Ophthalmologie, xii. 2, S. 149.

[7] Ibid., vii. 1, S. 150.

[8] A Treatise on Bright's Disease and Diabetes, 1881, p. 161.

Be the initial pathology of the disease what it may,—lesion of the ganglionic centres[1] or changes in the vascular intima,[2]—it is certain that in the chronic form there are localized changes through the entire blood-vessel system which lead to degenerative changes of a fatty nature.

In spite of the fact that Brailey and Edmunds[3] have found constant alterations in the retinal vessel walls where there were no apparent ophthalmoscopic changes before death, it must be conceded that these pathological pictures can be nowhere better seen ante mortem than in the retina. The membrane laid open to the careful ophthalmoscopist will frequently show alterations and peculiarities of appearance that will enable a competent observer to note them as pathognomonic. Care should be taken to lay sufficient stress upon minute and apparently insignificant changes. The writer has a distinct recollection of a young girl of eighteen, supposed to have been suffering from general cold, who was sent to him from one of the wards of a large general hospital into the ophthalmic dispensary service for the purpose of ordinary ophthalmoscopic study. In each macular region he discovered a few very faint and questionable flecks and points, which seemed to him to be sufficient to excite grave suspicion of renal disease. Upon so informing the attending physician, he was rewarded with a sceptical shrug of the shoulders. Careful examination and re-examination of the urine, with negative results, made the writer more and more uncomfortable. Six months later, he had the great satisfaction—scientific, at least—to receive a note of the result of an autopsy upon the patient, which had become necessary by reason of an uncontrollable attack of uræmia,—granular kidneys, with cardiac hypertrophy.[4]

Roughly, we should generally expect to find evidences of early œdema, points and areas of fatty degeneration, hemorrhagic extravasations, and even true neuro-retinitis, with consecutive atrophy, in all manner of variations and intensities. At first one or more of these conditions is so marked as to cause some authors to designate it as the peculiar form of retinal change, but later in the affection the other conditions begin to show themselves, and even outbursts, as it were, may give rise to sudden attacks of neuro-retinitis with hemorrhagic extravasation: again, the inflammation of the nerve-head, with its immediate consequences, may appear primarily, and mislead an observer who does not take the precaution to search the entire fundus for other related macroscopic changes. The star-like radiate patches so generally seen in the macular region should be carefully searched for in every suspected instance. Local atrophic changes now appear, vessel-calibre lessens still more, vascular walls become more thickened and more opaque, blood-supply diminishes, the nerve becomes more pallid and shrunken, and the fatty degenerations pass away, until at last the entire color of the fundus pales, and the ground assumes an appearance of optic-nerve atrophy with degeneration. Usually this stage is seldom reached, the patient, as a rule, rapidly succumbing, as shown by C. S. Bull,[1] to the general disease after the retinal changes of "chronic endarteritis" have been made sufficiently gross to be visible by the ophthalmoscope.

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[1] See Da Costa and Longstreth, American Journal of the Medical Sciences, July, 1880.

[2] A. V. Meigs, Transactions of the College of Physicians, 1888, p. 411.

[3] Transactions of the Ophthalmological Society of the United Kingdom, p. 14.

[4] Gowers (Medical Ophthalmoscopy, 1882, p. 184) mentions the rarity of early retinal changes without albuminuria in granular kidney-disease.

At times the choroid may be involved, showing atrophic patches from hemorrhages produced by breakage of the choroidal vessels. According to Gowers,[2] Poncet has figured "a peculiar choroid degeneration of the vessels of the choroid in old cases of albuminuric retinitis," which condition leads on to thickening of the tissue of the choroid. Liebreich (*loc. cit.*) has called attention to changes in the epithelium, appearing as "small angular gray spots of pigment," these being grouped, and "appearing first in the periphery."

If careful inquiry be not made into associated symptoms, and special attention not drawn to a few differential points, the ophthalmoscopic symptoms may at times be confounded with those that are dependent upon other causes, especially cerebral disease.

Treatment, which of course is to be directed towards the hygiene, etc., of the general system, need only be supplemented by protection of the irritated organs of vision from undue exposure and strain.

In the *acute form* of the disease, where a single blow, as it were, from a renal congestion alone, without involvement of the arterial system itself, apparently causes disk-choking with a few isolated "plaques," or where sudden blindness ensues with no evident intraocular lesion, it is most probable that either the presence of nitrogenous matter in the blood, with consequent uræmic poisoning, acting locally upon the optic-nerve and retinal elements or upon the visual centres of the cortex themselves, or causing simple œdema from watery extravasation in the blood, is the direct cause of the conditions. This is the form of intraocular disturbance from nephritis so generally found as a factor in the various exanthemata and general dyscrasiæ of children, rendering it, as can be easily imagined, the most likely form of retinitis to be associated with childhood. Fortunately, however, it apparently much more rarely affects the optic nerve and retina than the chronic variety, though it is most probable that were the eye-grounds of all the children affected with the various dyscrasiæ and exanthemata to be studied in a routine manner, there would be found a definite amount of retinal and optic-disk œdema in every case of any moment,—an amount which could be realized only after long experience and study among such cases,—an objective symptom which, by long training of the clinician, might often be of immense collateral advantage in deciding the

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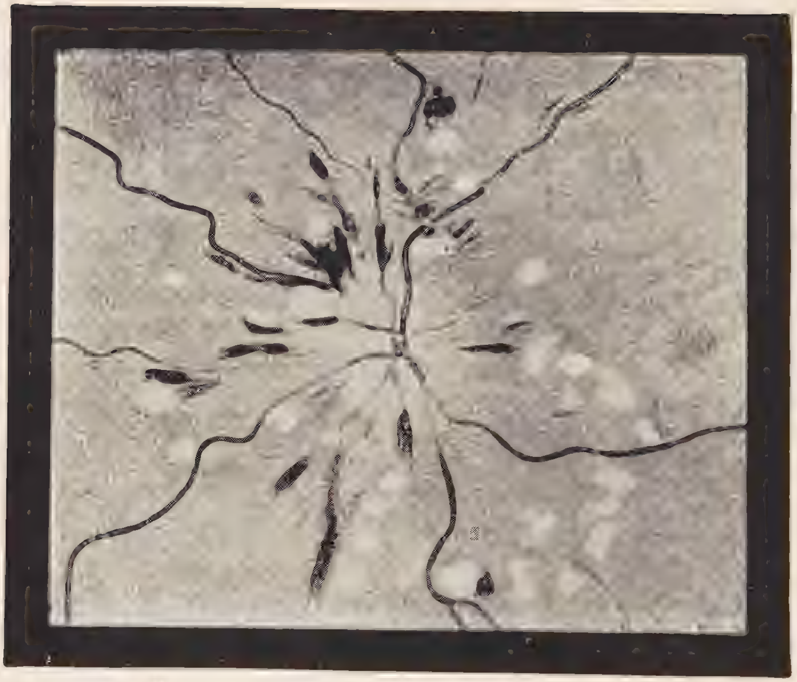
[1] Transactions of the American Ophthalmological Society, v. 190.

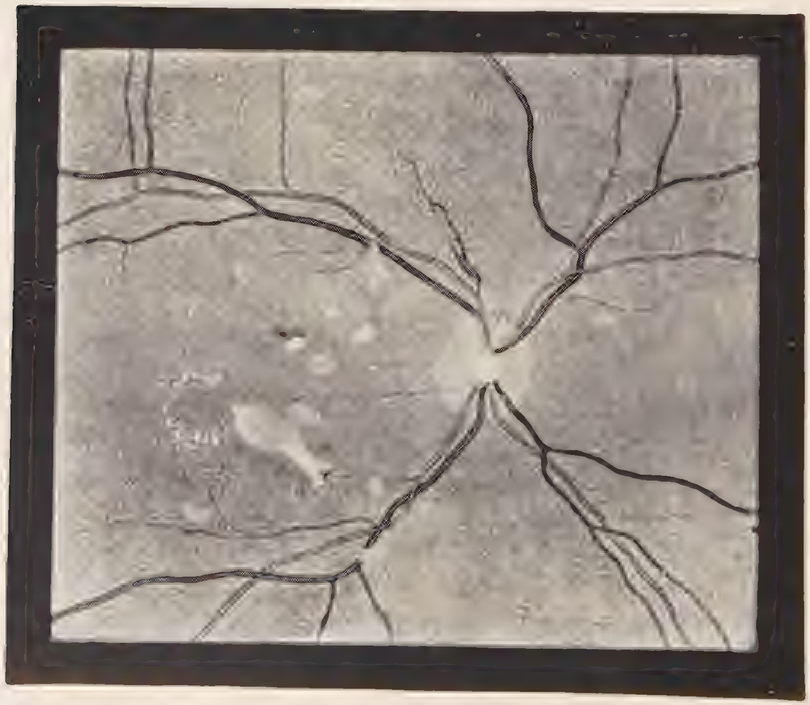
[2] Medical Ophthalmoscopy, 1882, p. 190.

PHOTO. XIII.



Acute Nephritic Retinitis. (Gowers, Medical Ophthalmoscopy, 1882.)

PHOTO. XIV.



Chronic Retinal Changes in Albuminuria. (Gowers, Medical Ophthalmoscopy, 1882.)

question of the gravity of the special disease under care at the time. Transient decreases of vision, too, expressive of either cortical, retinal, or even conducting-fibre œdema or irritation, may be of value in the question of prognosis and treatment. Ophthalmoscopic signs of optic-nerve and retinal irritation should be sought in every such case. That they are present in the majority of instances there can be no logical doubt, and were they searched for in every case, finer differentiation of retinal and optic-nerve disorder would be better known.

Here the treatment of the local symptoms, which must, of course, be directed towards the general condition of the patient, must, as can be easily understood, be both heroic and prompt. In every instance Tyson's words[1] should be borne in mind: "There is no doubt that many cases of acute nephritis recover while the conditions of *rest*, *quietude*, and *warmth* are maintained."

The first of the accompanying monotints, taken from Gowers, furnishes, in the author's words,[2] "a good example of the diffuse retinitis of Bright's disease occurring in the last period of chronic, supervening an acute, nephritis." The case illustrated by the second monotint presents[3] a typical example of the two forms of amblyopia of Bright's disease: 1, uræmic amaurosis, sudden in onset, accompanied by other evidence of uræmia, soon passing, and unattended by visible changes in the fundus oculi; 2, amblyopia due to the special changes in the retina, gradual in onset, persistent, increasing. The retinal changes could be followed from their commencement, and ran a subacute course. It is noteworthy that congestions of the disk, hemorrhages, and small, soft-edged patches were the earliest appearances, and preceded the zone of dots around the macula.

Davidson[4] reports a most instructive case of renal retinitis with total detachment of the retina (very much like Anderson's case described under the paragraph upon Rubeola) in a fourteen-year-old girl. The urine, which failed to reveal casts, was albuminous. Each fundus gave characteristic lesions of renal disease. Upon the increase of albumen, blindness from retinal detachment came on. Death followed after a convulsive seizure one month later. Upon post-mortem examination, both kidneys were found to be contracted, that of the right side being extremely atrophic and weighing but one ounce. Both capsules were adherent, leaving a granular surface. Granular changes in the cerebrum were also visible.

It is exceedingly doubtful whether Mooren's statement,[5] that when "*chronic skin eruptions* have their seat in the scalp they favor the occurrence of retinitis by maintaining a constant hyperæmia of the meninges," can be fully accepted. Gowers[1] believes that "if such a sequence occurs, it is possibly by the production of a local orbital cellulitis."

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[1] A Treatise on Bright's Disease, 1881, p. 113.

[2] Medical Ophthalmoscopy, 1882, p. 323.

[3] Loc. cit., p. 326.

[4] Transactions of the Ophthalmological Society of the United Kingdom, 1881, p. 57.

[5] Ophthalmologische Mittheilungen, 1874, p. 93; quoted by Norris, in A Practical System of Medicine by American Authors, vol. iv.

Before any definite opinion can be given as to the probability of the relationship between the two conditions, more data are necessary. The co-operation of those interested in dermatology will be of great assistance in obtaining proper statistics upon this all-important though as yet extremely vague subject.

LESIONS DEPENDENT UPON DISORDERS ORIGINATING IN THE GENERATIVE APPARATUS.

As the physiological action of the generative system remains inert until puberty, it is seldom, if ever, that we find any ophthalmoscopic symptoms which can in any way be said to have relation with the sexual apparatus before the adolescent period of life. At this time, however, the whole being changes, and every portion of the organism seems to enter into profound relationship with the generative health and function of the individual. The difference of mature development and the peculiarities in the activity of the sexual organs of the two sexes seem to exert special mysterious influences upon ophthalmic symptomatology; these being by far more profound in woman than in man.

Disorders of Menstruation.—In spite of Allbutt's inability to associate any ocular disturbance with menstrual derangement, there can be no doubt that disorders of menstruation are not infrequently the cause of retinal irritation and optic-nerve inflammation. Kollock[2] gives a brief account of a number of cases where, besides asthenopia, ocular pains, etc., which occurred during female puberty, retino-choroiditis appeared. Norris[3] cites an instance in a thirteen-year-old girl, where, in addition to the ordinary external congestion, with inability to use the eyes, "the retinal fibres were swollen and œdematous, hiding the outlines of the disk, while the lymph-sheaths of the retinal vessels at their point of emergence from the disk presented an almost snow-white appearance." Moreover, he says, "the disks and the retinæ have never quite recovered a normal appearance." Gowers[4] says that "in chronic menstrual irregularities optic neuritis of chronic course has been found, and occasionally other disturbances, such as retinal hemorrhages." The writer has at the present time a most interesting example of sudden intense neuro-retinitis, with great reduction of vision and limitation of color fields, appearing in a young amenorrhœic girl, which rapidly subsided, leaving both fair central and excentric vision, through prompt re-establishment of the menstrual function. R. H. Derby[5] cites an instance of an intense monocular neuro-retinitis which appeared in a twelve-

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[1] Medical Ophthalmoscopy, 1882, p. 222.

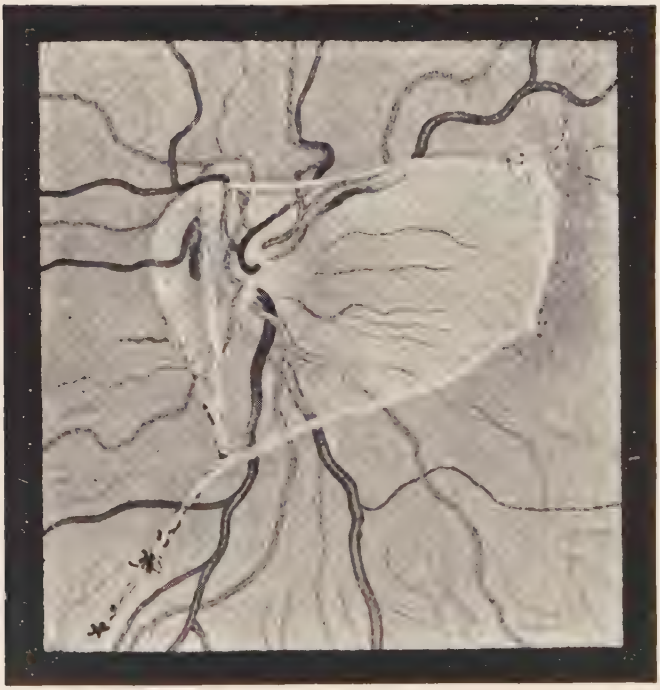
[2] Gaillard's Medical Journal, June, 1888.

[3] Pepper's System of Medicine, vol. iv.

[4] Medical Ophthalmoscopy, 1882, p. 221.

[5] Transactions of the American Ophthalmological Society, 1888, p. 106.

PHOTO. XV.



Partial Absorption of Hemorrhagic Opacities in the Vitreous. (Powers, Transactions of the Ophthalmological Society of the United Kingdom, 1888.)

year-old girl who had never menstruated. In concluding the report of his case, he says, "That the optic neuritis bore some relation to the efforts of nature to establish the catamenial period seems more than probable." In most of the cases the fundus-changes are very slight, and would probably pass unnoticed by a careless or an incompetent ophthalmoscopist. As the case advances, however, the conditions become more marked, and may eventually lead to actual inflammation and degeneration. Thaon[1] records a well-marked case of neuro-retinitis with "numerous white patches along and superjacent to the vessels, with some large muscæ in the vitreous. Vision was abolished in the upper third of the field. Treatment directed to the restoration of the menses was followed in this case by great improvement, if not perfect recovery." Sometimes profuse intraocular hemorrhages appear and reappear at every catamenial epoch. Wardrop[2] credits Pechilini with having seen "a young woman who had amaurosis during a suppression of the menstrual discharge, which was removed the moment menstruation returned." St.-Yves[3] quaintly says that "it generally attacks *maids* that are not regular." Dor relates[4] a most instructive case, whilst Power (*supra*) gives an exquisite chromo-lithograph of a similar condition in a thirteen-year-old girl suffering from menorrhagia. The accompanying sketch shows the condition of the fundus "after the opacities in the vitreous occasioned by the breaking up of the blood-clot behind the iris had broken up and diffused through the vitreous."

Prognosis is favorable in due proportion to the amount of resultant pathological change.

In the majority of instances the treatment must be given to the gynæcologist, leaving dangerous local complications to the therapy of the ophthalmologist. In the hands of the writer, varying proportions of the ingredients of Dewees's mixture have proved of great advantage in a few cases of dysmenorrhœa and amenorrhœa with ocular symptoms that he has seen.

As a matter of curiosity, it may be worth while to state that Finkelstein[5] asserts that a concentric narrowing of the field of vision for both form and color takes place during the ordinary menstrual period, which reaches its greatest degree during the height of the epoch, this being accompanied at times with faulty perception of yellow for green, and a slight diminution of central vision without change of refraction.

Masturbation.—This vice, which is probably of greater frequency in the male sex, is undeniably shown to be at times associated with optic-nerve and retinal change. Cohn's[6] seven cases, three of which were girls, gave pronounced photopsiæ as subjective evidences of retinal irritation, these symptoms rapidly subsiding upon cessation of the habit. Fitzgerald[1] gives the details of the cases of two young women who were victims of the habit, in both of whom the optic disks were slightly hazy, associated with pallor, which condition was followed by "total atrophy" of the nerve in one of the cases; whilst Power[2] has seen with MacKinlay a hemorrhage upon or in the retina in a nineteen-year-old lad, whose only causative symptom seemed to be frequent masturbation.

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[1] Thèse de Paris, reported by Power, in Transactions of the Ophthalmological Society of the United Kingdom, 1888, p. 14.

[2] Essays on the Morbid Anatomy of the Human Eye, 1818, ii. 171.

[3] A New Treatise of the Diseases of the Eyes, 1741, p. 194.

[4] Recueil d'Ophtalmologie, 1884, p. 164.

[5] Inaugural Dissertation, St. Petersburg, 1887.

[6] Archives of Ophthalmology, 1882, p. 428.

The diagnosis, which is often extremely difficult, especially in the female sex, is to be determined by allied symptoms. In contradistinction to the results of Fitzgerald's and Power's cases, the prognosis ordinarily may be said to be favorable. Treatment should be directed towards the moral and physical hygiene of the patient, and care should be taken in all inveterate cases to institute search for mechanical irritants, such as phimosis, vulvitis, vaginitis, etc.

The blindness of *hysteria*[3] is but another among the multitudinous real and fanciful symptoms which are so frequently spoken of by a most unfortunate class of subjects. As with all other forms of disease which are more or less closely related with the condition of the sexual apparatus, the male sex is certainly freer than the female, although, as Charcot says,[4] hysteria in the male is far from being rare. Harlan[5] gives the details of the case of a boy of eleven years who persisted in supposed blindness of one eye for more than eighteen months. St. John[6] notes an almost identical instance, though only of five weeks' duration, in a ten-year-old boy with decided neuropathic antecedents. Another case by Harlan[7] of ten years' duration had its exciting cause in the assertion of an ophthalmic surgeon of a "paralysis of the optic nerve" from traumatism; the whole delusion being dispelled by a few moments' careful ophthalmic examination. Moore[8] gives an instance of a fifteen-year-old lad with blindness in the right eye, which occurred after disappointment at school. Under ether and electricity recovery was both rapid and permanent. Schweigger[9] associates the monocular type, as just given, with unconscious psychic deceit upon the part of the subject. Glascott[10] and Snell,[11] under the title "Amaurosis fugax," each describe instances of the bilateral variety. Griffith[12] reviews the subject and adds some clinical data. Both Jackson[1] and Marlow[2] refer to cases in the male sex; the former's case being that of a delicate colored lad of twelve years. Charcot[3] describes a variety in a boy of sixteen years, in whom, in association with anæsthesia in patches and blunting of hearing, smell, and taste on the left side, there was double contraction of the visual field, more pronounced on the right side, upon which side the patient did not distinguish violet; the fields for red were larger than those for blue. In spite of all treatment, the convulsive crises, the hysterical stigmata, and the sensorial and sensitive anæsthesias continued. A second and somewhat similar instance, in an eighteen-year-old boy, is cited by the same author.[4]

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[1] Transactions of the Ophthalmological Society of the United Kingdom.

[2] Ibid., 1888, p. 7.

[3] Although it is doubtful whether this functional disorder should come under the above category, yet, as the associated conditions are so symptomatic of sexual derangement, it has been thought best to place it there.

[4] Clinical Lectures on Certain Diseases of the Nervous System, 1888, p. 10.

[5] American Journal of the Medical Sciences, October, 1873.

[6] Transactions of the American Ophthalmological Society, 1889, p. 330.

[7] Op. cit., p. 328.

[8] Medical Chronicle, August 2, 1889.

[9] Klinische Monatsblätter für Augenheilkunde, November, 1881.

[10] British Medical Journal, 1879.

[11] Ophthalmic Review, 1882.

[12] Transactions of the Ophthalmological Society of the United Kingdom, vii. 292.

In the female these symptoms are more common, as has been incontestably shown at La Salpêtrière, at which place Charcot finds frequent coincident or causative ovarian derangement. Applying the significance of Weir Mitchell's expressive words, that "the symptoms of real disease are painted on an hysterical background,"[5] to the reasoning of the causation of the many curious subjective ocular changes seen in such cases, the special symptomatology of the organ as seen in ordinary disease may be said to be absolutely and entirely given. Varying contractions and perversions of the visual fields, temporary losses of perception of certain colors, marked lowering of direct vision, appear,—all probably, in a measure, the result of sensory fatigue in association with the ordinary motor impairment of both the intraocular and the extraocular series of muscles. Even retinal hyperæsthesias, with colored phosphenes at times, in conjunction with the almost innumerable variety of clonicisms, frequently occur, whilst crossed amblyopias and hemianæsthesias seemingly appear. Many cases could be given, but it hardly seems necessary to cite in detail what so many neurologists and ophthalmologists see so frequently. An interesting case, however, of this character in a young girl has recently come under the observation of the writer, in which, in addition to the ordinary symptoms of greatly-lowered vision for form and color (the former of which could not seemingly be improved by careful correction of an existent refraction-error) and the general characteristic conditions, there was a peculiarity of the fields of vision, which at first seemed to tally with the so-called "perversion of color-fields" so frequently noted by various authors, but after repeated and painstaking trials at several hours' intermission proved to be nothing more than an abnormal fatigue of the sensory structures, by which at every trial any order of fields could be obtained,—this order always coinciding in extent with the first colors tried. One day the green, which was tried first, gave a much larger area than white, blue, yellow, and red, in the order named; on the following morning green was made the smallest area and red much greater in extent than any other color-field, simply by trying the green last and the red first. In other words, the color first tried gave the largest area. On the third and fourth days the colors were tried at fifteen-minute intervals, at which examination the usual order of white, yellow, blue, red, and green was followed, though in every instance the areas were more and more contracted, just as the direct vision for form and for color was found more greatly impaired after repeated trials. Fleeting scotomata for every color could be obtained in any part of the visual fields, but at no time were the colors miscalled or confounded with one another. The questions thus arise, May not many of the so-called perverted color-fields in this disease be simply due to improper field-taking, and cannot the order of sequence of color-areas be obtained much more frequently by carefulness to avoid sensory fatigue? In a second though still uncertain case, seen in the practice of Dr. Robert M. Girvin through the courtesy of Dr. John H. Musser, in which there was blindness upon the left side, with a perverted order of irregularly contracted and excentric color-fields upon the right side, the *yellow*[1] was persistently designated as "pale green" in every part of its visual field except at one small point in the centre of the combined color-areas up and out, where it was properly named. In this case the retina of the left (blind) eye showed a slightly œdematous condition, with some tortuosity of its veins, as described by Landolt. De Schweinitz[2] also reports "a somewhat distended and slightly tortuous retinal vein, with undue prominence of the central lymph-sheaths," in a sixteen-year-old girl, with complete hysterical analgesia and aphonia. In this case the visual fields were normal.

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[1] Transactions of the American Ophthalmological Society, 1888, p. 85.

[2] New York Medical Journal, February 9, 1889.

[3] Clinical Lectures on Certain Diseases of the Nervous System, 1888, p. 131.

[4] Op. cit., p. 143.

[5] Quoted by Gowers, Manual of Diseases of the Nervous System, 1888, ii. 907.

In two cases recently studied through the kindness of Dr. William Goodell, careful perimetric examination showed more than one-thirtieth reduction of both form- and color-vision from normal, this in each instance being more pronounced upon the left side. The same is true of another example of the same type seen in conjunction with Dr. Wharton Sinkler. All three cases were in young female adults. In no instance was there any perversion of the order of the fields of vision.

From these groupings it can be seen that with the concomitant symptoms the diagnosis is comparatively easy, though when the manifestations are limited to the ocular apparatus alone the disease becomes difficult to discover. Enough, however, has been said to show that the very mutability of the special symptoms affords an excellent criterion for the recognition of "that domestic demon which has produced untold discomfort in many a household, and, I am almost ready to say, as much unhappiness as the husband's dram."[3]

With the exception of retinal-vessel dilatation and œdema, true objective fundus-changes have never been noted, although it may be fair to presume that in the hystero-epileptic form of the disease, where the seizures have been both many and severe, physical alterations like those so constantly found in old epileptics (even in young subjects) may become visible.

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[1] This is both remarkable and highly symptomatic, as being opposed to the ordinary color-loss seen in organic change of the second nerve.

[2] American Journal of the Medical Sciences, November, 1889.

[3] S. Weir Mitchell, Wear and Tear, or Hints for the Overworked, 1874, p. 29.

No absolute data can be given as to the prognosis. Some cases recover vision spontaneously without any assignable cause; others are almost momentarily freed from this disturbance by profound psychic impression; others, again, linger for a long time without any apparent gain, in spite of all hygiene and judicious care; whilst a number seem pushed, as it were, into absolute darkness, to remain forever blinded.

All that contributes to the general welfare of the patient (not forgetting Dr. S. Weir Mitchell's highly successful "combination of therapeutic measures,"[1] which "consists in an effort to lift the health of patients to a higher plane by the use of seclusion, which cuts off excitement and foolish sympathy; by rest, so complete as to exclude all causes of tire; by massage, which substitutes passive exercise for exertion; and by electrical muscular excitation, which acts in a somewhat similar manner to massage, and with it by depriving rest in bed of its essential evils,"[2] in any case that has resisted the advantages of out-door exercise in a new environment) should be conscientiously tried, and ofttimes apparently grave ocular symptoms will disappear as the physical and the moral tone of the patient improve. Simulation of both the conscious and the unconscious types can be readily detected by repeated and careful testing.

LESIONS DEPENDENT UPON SPECIAL DYSCRASIÆ.

In *enteric* or *typhoid fever,* which is especially an affection of early adult life and adolescence, ophthalmoscopic lesions are not wanting. Hutchinson[3] gives an instance of a boy who developed double optic neuritis two or three weeks after an attack of fever which was complicated by marked diarrhœa and cephalalgia,—a condition of eye-nerve no doubt due to an associated meningitis. Fortunately, however, as Wilson tells us,[4] "actual meningitis is exceedingly rare, notwithstanding the comparative frequency of symptoms suggestive of its presence."

Cases of secondary atrophy without ophthalmoscopic appearances of previous inflammation of the nerve-head are on record, as, for instance, the interesting one noted by Haddaeus[5] and others. The present writer has seen a ten-year-old girl in the general wards of St. Agnes's Hospital, who, two weeks after the cessation of all active symptoms of typhoid fever without any seeming complication, showed extremely pallid disks, especially to the temporal sides, with marked reduction in the calibre of both the arterial and venous currents. The query arises, Was a low-grade meningitis the mischief-maker in this instance?

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[1] Lectures on Diseases of the Nervous System, especially in Women, 1885, p. 269.

[2] Op. cit., p. 270.

[3] Royal London Ophthalmic Hospital Reports, ix. 125.

[4] This Cyclopædia, i. 471.

[5] Klinische Monatsblätter für Augenheilkunde, August, 1865.

As cardiac thrombi are present at times, it is not difficult to understand how intraocular vascular changes, such as embolism of the central artery of the retina or of one of its retinal branches, may at times be found. Galezowski[1] cites such a case.

Typhus fever, which is usually mild in children, is not very apt to have marked intraocular changes associated with its symptoms. Where optic nerve-head changes have been found, it is most probable that they have been either the results of meningeal inflammation or the consequences of lodgement of embolic or thrombotic massings: thus, the cases of Chisolm[2] and Teale,[3] quoted by Gowers,[4] probably belong to the former class. According to Norris,[5] Larionow's statistics of fifty-seven patients with typhus exanthematismus show one instance of neuro-retinitis and two cases of contraction of the field of vision. Pepper,[6] who has had opportunity to study the affection during an epidemic in Philadelphia, says that eye-symptoms were very rarely seen.

Independently of the presence of a definite spirillum in the blood of patients suffering from *relapsing fever*, which of itself may be productive of disturbance in the vascular channels of the eye, metastases from splenic abscess, septic peritonitis, or even hemorrhagic infarcts from various viscera, etc., which are at times found in this disease, may all give rise to profound inflammation of the more deeply seated tissues of the uveal tract. Choroiditis with consequent hyalitis, cyclitis with pus in the posterior chamber,[7] and retinal and optic-nerve complications, may all appear. Trompetter[8] has determined the presence of these conditions in nearly six and a half per cent. of over three hundred cases seen by him.

Scarlet fever, or *scarlatina*,—the dread disease of the young,—is so apt to have renal derangement as one of its symptoms that it is not infrequent to have some form of intraocular expression of the complication. More especially is this noticed with the amblyopic form of disturbance, where, without warning, at the time of the utmost severity of the attack, double blindness suddenly appears, to last but a few days. The ophthalmoscope seemingly tells nothing of the cause, except a possible slight obscuration of the border of the head of the optic nerve. Ebert,[9] quoted by

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[1] Traité Iconographique, p. 188.

[2] Royal London Ophthalmic Hospital Reports, vi. 214.

[3] Medical Times and Gazette, May 11, 1867.

[4] Medical Ophthalmoscopy, 1882, p. 240.

[5] A System of Practical Medicine by American Authors, vol. v.

[6] System of Medicine, vol. i.

[7] Schweigger (Hand-Book of Ophthalmology, 1878, p. 419), in speaking of an epidemic of recurrent fever which occurred in the Charité Hospital in Berlin, says that there was simple unilateral iritis in nearly one-half of the cases.

[8] Klinische Monatsblätter für Augenheilkunde, April, 1880.

[9] Veränderungen des Augenhintergrundes.

Norris[1], gives a series of clinical histories of such instances. In the discussion of these cases, Graefe[2] makes the important observation that as there is always proper motion of the irides to light-stimulus, the lesion must be posterior to the quadrigemina: the symptoms of such a condition thus may prove of immense collateral value in the prognosis of vision. Pflüger's note[3] of a ten-year-old child who became gradually blind in three days' time three weeks after an attack of scarlet fever, is interesting in showing "double papillo-retinitis" without the presence of albumen in the urine. In five months sight had returned to almost normal. It is probable that meningitic inflammation was the cause, as was partially evidenced by "considerable headache" during the fever. Bayley[4] gives the histories of progressive failure of sight in two sisters who had uncomplicated attacks of scarlet fever. In each instance pigment-massings in the fundus oculi were visible, whilst the optic disk is noted as merely "pale." Cases of direct implication of the optic nerves, with seeming consecutive atrophy from descending optic neuritis, are on record. More rarely, inflammatory changes in the choroid, with liquid effusion between the choroid and the retina, giving rise to grave intraocular destruction, have been noted.

In all these latter forms of ocular disease the prognosis is truly grave. Lubinski[5] is said to have found evidences of irido-cyclitis with vitreous opacities in twenty-two out of six hundred and forty patients. The ocular affection occurs as a late sequela of the fever. In the lighter form hyperæmia of the disk is noted. All the cases seen were in male adults.[6] Alterative and absorbent treatment, with attention to the general hygiene of the little patient, is all that can be judiciously recommended for the preservation or improvement of the remaining sight.

In marked cases of *diphtheria*, especially where multiple paralyses ensue, ophthalmoscopic pictures of grave optic-nerve disease with consecutive atrophic changes have been seen. Bouchut[7] has found such instances, one of which was unilateral in type. Gowers[8] says, "The congestion and œdema are usually bilateral, but may be more intense on one side than on the other." In partial confirmation of this statement, the writer has published[9] an account of a twelve-year-old boy who, five years after an attack of diphtheria and paralysis which confined him to bed for three months,[10] whilst presenting an ophthalmoscopic appearance of double chorio-retinitis with partial degeneration of the optic nerve, associated with curious lymph-extravasation into the retina and vitreous, more pronounced on the right side, gave but one-fiftieth of normal vision with the right eye and nearly one-fifth with the left. At that late time the patellar tendon reflexes were almost abolished. An examination of the urine failed to reveal any abnormal deposits.

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[1] Pepper's System of Medicine, vol. iv.

[2] Quoted by Norris.

[3] Archiv für Ophthalmologie, vol. xxiv., mentioned in Gowers's Medical Ophthalmoscopy, 1882, p. 243.

[4] The Lancet, September 15, 1877.

[5] Wjestnik Ophtalmologie, 1887.

[6] Thomson and Gould, Annual of the Universal Medical Sciences, vol. iii., 1888.

[7] Quoted in Gowers's Medical Ophthalmoscopy, 1882, p. 249.

[8] Medical Ophthalmoscopy, 1882, p. 249.

[9] Transactions of the American Ophthalmological Society, 1887.

[10] During the time of his illness he was unable to speak, and his eyesight became bad,—the latter condition persisting.

It is probable that most of the fundus-lesions sometimes met with in *rubeola* are dependent upon meningitis or some other form of cerebral complication. Wadsworth[1] reports three such instances. Stephenson[2] cites a case of double optic neuritis which was accidentally discovered in a four-year-old boy who had just recovered from an uncomplicated attack of measles. Whether there was any causal relationship between the two in this case it is impossible to say, although there was an entire absence of any other apparent organic disturbance. Coggin[3] gives the history of several cases. He says, "The lesion is supposed be be a circumscribed basal meningitis, and non-tubercular, though rarely tubercular deposits are present." Both Von Graefe[4] and Nagel[5] give cases—especially the latter—which are expressive of cerebral disturbance.

Should nephritis set in, the fundus-lesions may become quite pronounced: thus, a rare case of isolated retinal detachment from subretinal effusion subsequent to a severe hemorrhagic neuro-retinitis is reported by Anderson[6] as having been seen in a six-year-old girl who was suffering from chronic nephritis, which seemed to follow an attack of rubeola, with subsequent bronchitis and "consumption of the bowels," at eighteen months of age. Uræmia terminated the patient's life three months after the ophthalmoscopic signs became manifest. The necropsy revealed advanced fibroid contraction of the kidneys. The case is worthy of record not only on account of its comparative rarity, but also as most probably having had the exanthem as its starting-point.

Here, as shown in all the acute exanthemata, the solution of the whole problem is to be found in pathological changes which affect the nervous and vascular systems.

In view of Edwards's observation[7] that he has seen "one instance of tubercular meningitis develop as a complication of *rubella*" (*rötheln*), fundus-oculi lesions indicative of this condition may at times be expected. As renal disturbance has also been met with by Curtman (*loc. cit.*), ophthalmoscopic expression of such an occurrence may not be wanting; in fact, all the intraocular expressions of other exanthemata, which are visible to the ophthalmoscope, may be safely asserted to have proper placing among the possible ophthalmic symptoms seen from time to time.

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[1] Boston Medical and Surgical Journal, 1880, p. 636.

[2] Transactions of the Ophthalmological Society of the United Kingdom, viii. 150.

[3] American Journal of Ophthalmology, January, 1890.

[4] Archiv für Ophthalmologie, xii. 2, 138.

[5] Behandlung der Amaurosen, S. 24–30 (quoted by Norris).

[6] Transactions of the Ophthalmological Society of the United Kingdom, 1888, p. 141.

[7] This Cyclopædia, i. 697.

The sudden blindness which sometimes appears during the convulsive stage of *pertussis* is *a priori* generally associated with hemorrhagic extravasation into the interior of the eye from vessel-rupture during a paroxysm. Case III. of Landesberg's series[1] most probably belongs to this grouping. Knapp[2] found all the symptoms of "ischæmial retinæ" in a three-year-old boy who suddenly became blind during an attack of whooping-cough. Curiously, "both pupils, however, responded promptly to changes of light." A double paracentesis seemed to restore both optic nerve and retinal circulation to a sufficient degree not only to give objective signs of betterment, but even to allow the patient to recognize ordinary objects. Six weeks after the operation the child died from lobular pneumonia: a confirmation of a remark made to the observer by Prof. Loomis, of New York, who informed him "that blindness in whooping-cough had been observed, but almost exclusively in children who afterwards died from lobular pneumonia." Case I. of Landesberg's list[3] reads very much like a case of embolism of one or two of the upper arterial branches in the right retina: vision is said to have been restored to one-half of normal. Alexander[4] contributes two examples. The first, which was followed by death, gave proper pupillary reaction; here cerebral œdema between the occipital lobes and the quadrigeminate bodies is supposed to have been the cause. The second showed optic neuritis with consecutive partial atrophy. In this instance the irides were immobile. Meningitis is named as the cause.

Except by direct infection or extension of purely local changes, deep lesions of the eye as complications or sequelæ of *variola* are very infrequent. Gowers[5] instances a man of fifty who had atrophy of the right optic disk dating back to an attack of small-pox at twelve years of age. When seen, the patient exhibited some general and local signs of ataxia. He also cites Leber[6] as observing diffuse neuro-retinitis during the stage of drying of the eruption.

Varicella, the most benign of all the acute exanthemata, fails to present any lesion of the optic nerve or the intraocular tunics except in the gangrenous variety of the disease. In this class of cases Hutchinson[7] has seen loss of sight from purulent irido-choroiditis, a condition no doubt dependent upon metastasis of pathogenic material from some infected organ, causing the formation of a local inflammation somewhere in the uveal tract, with consequent abscess.

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[1] Medical and Surgical Reporter, 1880, p. 249.

[2] Archives of Ophthalmology and Otology, iv. 448.

[3] Op. cit., p. 248.

[4] Deutsche Medicinische Wochenschrift, No. 11, 1888.

[5] Medical Ophthalmoscopy, 1882, p. 348.

[6] Op cit., p. 244.

[7] Quoted by Jennings in vol. i. of this Cyclopædia, p. 763.

As mentioned by Hirschfelder in vol. i. of this Cyclopædia, p. 778, "Amaurosis has been produced by retrobulbar abscess," in *erysipelas* both of the face and of the head, this being caused by extension of the external inflammation into the tissues of the orbit, with involvement of the optic nerve. Both Knapp[1] and Jaeger report cases where the eye-grounds presented pictures indicative of thrombosis. In some of the worst cases the veins of the orbit become pus-bearing, leading to brain-complications, which cause the patient's death. Weiland[2] has had a marked case where recovery was extremely slow. At times there is no other decided symptomatic evidence of pus-formation in the orbit with extension, or proof of pressure from inflammatory material, than a rapid loss of vision. This decrease of sight generally begins either both centrally and peripherally, leaving the so-called annular field, or centrally alone. In such cases intra-ocular changes of consecutive atrophy are apt to manifest themselves objectively later.[3] Oeller's[4] case is of much interest.

The peculiarity of the ophthalmoscopic picture, in every instance, is merely indicative of the kind of offending inflammatory material, and is dependent upon the amount and position of the pressure-changes.

As endocardial disease is more prone to appear in the *rheumatism* of childhood than it is in that of adult life, ocular symptoms expressive of the complication should be more frequently expected among children than among older patients. These changes, as might be expected, belong to the vascular type of disease: thus, embolism of the central retinal artery, or even choroiditis of the metastatic type, may appear. Direct implication of the post-ocular portion of the optic nerve from irritation in the orbit may give rise to pressure-signs or inflammation-symptoms in the interior of the eye.

Not infrequently, when a new or a weakly subject is pushed, as it were, into a paludal district, the series of symptoms indicating *malaria* produced by the specific blood-poison are increased by an ocular grouping which not only embraces the superficial form of inflammation but also includes changes in the deeper and denser motor and sensory nerve-structures of the organ. These changes in the eye are probably due either to the passage of some of the foreign substance in the blood into the ocular tissues themselves, or to the lodgement of pathological products in such a position in the intraocular apparatus as to cause undue pressure upon some important part, with irritation and inflammation of adjacent material. In more marked cases brain and spinal-cord disturbances may ensue, the former giving rise to complications which make themselves known by various peripheral neuroses. Rarest of all these affections is that of the optic nerve itself. At times its disturbance is made apparent by attacks of transient amblyopia, which probably by frequent repetition causes gradual degeneration of nerve-material. Such symptoms generally manifest themselves very soon after a severe attack of the fever, especially if the patient be in a hot climate. In these cases the capillary circulation of the optic disk is almost or quite gone, leaving the substance very pallid and white. At times the retinal vessels are small, whilst limitation of the visual field, even of the hemianopsic variety, and great diminution of central vision, for both form and color, manifest themselves. C. S. Bull[1] describes two such cases in adults.

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[1] Archives of Ophthalmology, 1884.

[2] Deutsche Medicinische Wochenschrift, 1887.

[3] Stillé (International Encyclopædia of Surgery, Ashhurst, 1881, p. 185) quotes Parinaud (Archives Générales de Médecine, June, 1879, p. 641) as saying, "Besides the sequelæ common to all the forms, there is one that is indeed rare and seems peculiar to erysipelas of the face. It is blindness due to an atrophic degeneration of the optic papilla, which sometimes affects only one eye, and sometimes both eyes. The impairment of sight, when it occurs only in one eye, begins towards the close of the attack, or when the swelling of the eyelids has subsided sufficiently to permit them to be raised. When both eyes have been involved, the impairment of sight appears not to have been noticed before the complete subsidence of the erysipelas, and, after varying in degree, to have left a permanent defect of vision, sometimes, however, in regard to certain colors."

[4] Münchener Medicinische Wochenschrift, 1889.

In another class of cases, which occurs almost exclusively in the tropics, and in which hemorrhagic retinitis with perineuritis exists, the fundus oculi shows numerous striated and stellate hemorrhages either situated along the larger retinal stems or scattered in small areas between the disk and the fovea, the nerve-head itself varying in degree of swelling and serous infiltration. De Mussey[2] relates one such instance.

Mackenzie[3] gives the descriptions and drawings of the eye-grounds of two young men with the quotidian type of the disease. In each instance large superficial hemorrhages were found, the first having a number of pin-point opacities scattered about the eye-ground. A third case, of the tertian type, in a patient aged eighteen years, also showed hemorrhagic extravasation into the retina. In none of the instances was the optic nerve very much disturbed. In all the retinal-vessel calibre appeared normal.

According to Gowers,[4] Ramorius[5] has had the good fortune to study the vascularity of the fundus oculi during a series of paroxysmal attacks of periodical amblyopia in two cases. During the paroxysm the nerve-heads were pallid and the arteries of the retina were thread-like and almost bloodless, whilst the veins were nearly invisible. Curiously, this attack was accompanied by "great congestion of the face and ears and a sensation of heaviness in the head." Other vaso-motor and sensory symptoms were present. Quinine rapidly cured both of the cases.

Accepting Guitéras's conclusion,[6] "that the foci of endemicity of *yellow fever* are essentially maintained by the creole infant population," and agreeing with his belief[7] "that yellow fever in its native haunts is essentially a disease of childhood, the adult native being protected by a previous attack," it at once becomes apparent that the ocular symptoms of the disease, modified by the age of the patient and the gross peculiarity of the general symptoms, are to be found here just the same as in the adult.

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[1] American Journal of the Medical Sciences, April, 1877.

[2] Journal d'Ophtalmologie, 1872.

[3] Medical Times and Gazette, 1877.

[4] Medical Ophthalmoscopy, 1882, p. 247.

[5] Annali di Ottalmologia, 1877.

[6] This Cyclopædia, i. 854.

[7] Op. cit., p. 857.

Although the conjunctival injection and ecchymotic spots are very rare in this class of subjects, yet their very presence, just as in older patients, may serve to explain in some instances rapid failure of sight as possibly caused by intraocular extravasation. In graver cases, though fortunately, from the nature of the disease, its pathology, and the age of the patient, very rarely, emboli or thrombi from hemorrhagic foci in the cardiac and pulmonary cavities may at times appear and give rise to pronounced visual and ocular disturbance. Again, temporary blindness and dimness of vision at the onset of the disease, or pupillary dilatation and extraocular muscle enervation seen later in the disorder, as shown by Rush[1] to have been found in adult cases, may at times be expected in some cases where unusual nerve symptoms followed by coma ensue.

Were the ophthalmoscope employed regularly in every instance of the pyretic stage of *dengue* as seen in children, ophthalmoscopic symptoms indicative of cerebral change might at times be manifest. Should endocarditis or even arthritis appear, intraocular manifestations, as previously mentioned in speaking of some of the other fevers and dyscrasiæ, may show themselves. According to Thomas,[2] "glaucoma, amaurosis, . . . and other evidences of the profound impression of the poison . . . are more deserving of attention."

In *cholera*, as Norris says,[3] "the retinal arteries are much diminished in size, and the veins, although not dilated, are filled with blackish blood." Continuing, he tells us that, "owing to the great feebleness of the circulation, the slightest pressure with the finger on the eyeball produces arterial pulse." Von Graefe[4] has found instances where artificially induced increased intraocular tension caused the arterial blood-currents to disappear, this being especially noticeable in cases where the cardiac action was so enfeebled that the ordinary radial pulsation could not be determined. He has also found the optic nerve head reddish blue in appearance.

In spite of the declaration of some authors that they have been unable to find a special form of retinitis in *diabetes*, the affection is of such great frequency in the glycosuric variety of the disease and the changes are so peculiar that there can be no reasonable doubt of the existence of the association. If it be true, as Ellis[5] says, that both forms of the disorder are excessively rare in children, and if clinical results support Fenwick's assertions[1] that when present in early life the disease "is seldom protracted beyond three or four years," and that most patients "die within a year or eighteen months," it may be fairly presumed that ocular disturbance should be expected in the great majority of such cases,—a presumption that can only be made a certainty, however, by painstaking study of every case. Rolland[2] goes so far as to say that he has never found a glycosuric who did not have some visual disturbance.

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[1] An Account of the Bilious Remitting Yellow Fever as it appeared in the City of Philadelphia in the Year 1793, 1794.

[2] Quoted by Matas, this Cyclopædia, vol. i. p. 891.

[3] A System of Practical Medicine by American Authors, vol. iv.

[4] Archiv für Ophthalmologie, xii. 2, 210.

[5] A Practical Manual of the Diseases of Children, 1879, p. 157.

In a number of instances there is nothing more than a binocular dimming of central vision, with the retention of fair peripheral sight, and without any appreciable ophthalmoscopic lesion. In others, following this condition, the nerve-head generally loses some of its capillarity and appears partially atrophic. In some of these cases, as shown by the great disposition to hemorrhagic extravasation,[3] it is possible that the degenerative changes that have been found post mortem in the optic nerve[4] are the results of aneurismal dilatation with breakage of the vessel-walls of the capillaries of the optic nerve in such a position as to give rise to the appearance of central scotomata.

In another variety, especially where probable nephritic change has taken place, as expressed by the presence of albumen in the urine (though by no means invariably), the hemorrhages seem to take place intraocularly; here the ophthalmoscopic picture is totally different: instead of a comparatively healthy eye-ground, the retina is puffed and swollen, the disk is hidden, whilst numerous hemorrhagic striations in the retina and masses in the vitreous prevent any useful sight. Again, small circummacular dottings have been noticed, apparently bearing no relation to albuminuria. Relapses are apt to occur. At times the influence of a cerebral tumor producing either direct or indirect pressure upon the fourth ventricle may be suspected as the causative factor, especially if other localizing symptoms, such as ocular paresis and paralysis, be present.

Prognosis is always bad. Treatment is to be directed towards the systemic trouble alone.

Ocular changes in the insipidus variety of the disease are very much rarer than in the mellitus form. The only unequivocal case that the writer has had opportunity to study failed to present any coarse ophthalmoscopic changes whatever. The patient was a young girl who suffered intensely from general symptoms of the disorder. Her mother died of diabetic coma following the birth of a child,—a low-grade female imbecile,—who died of organic heart-disease eight years later.

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[1] Outlines of the Practice of Medicine, 1880, p. 366.

[2] Recueil d'Ophtalmologie, 1887.

[3] See photo-lithograph of Gowers's preparation of capillary aneurism and varicose capillaries from the retina in a case of diabetes with retinal hemorrhages (Medical Ophthalmoscopy, 1882, p. 376), from a case under the care of Mackenzie and Nettleship (Royal London Ophthalmic Hospital Reports, ix. 150). Here, as Gowers says (op. cit., pp. 197, 198), "the chief change beyond œdema was a peculiar hyaloid degeneration of the intima of the arteries and numerous capillary aneurisms."

[4] See article by Nettleship and Edmunds in the Transactions of the Ophthalmological Society of the United Kingdom, i. 124.

Although acquired *syphilis* in the young is rather infrequent, yet as the early recognition of its symptoms and their treatment are so essential to the future welfare of the patient, a brief mention of the various ophthalmoscopic signs will be made here.

In this disease, as in all other dyscrasiæ, the uveal tract seems extremely prone to disturbance. Inflammation of the choroid of either a disseminate or a macular variety rapidly involving the retina, and associated with fine vitreous opacities, is the most common form; that this should be so is very natural, as the choroid being practically a part of the uveal tract, and rich in vascularity, like the iris and the ciliary body, this tunic is very apt to be compelled to bear the brunt of the ravages of this dread disease. Should the inflammatory changes in the choroid be primary and extend forward into the ciliary and iris regions, gummatous swellings, descemetitis, and turbidity of the aqueous humor, with the usual pupillary and iritic changes, may all manifest themselves.

Prognosis in all these cases is bad. Treatment should be directed towards the dyscrasia; taking care to prevent the patient from employment of the eyes and to keep them at rest by the use of mydriatics and dark glasses.

Diffuse retinitis (probably at times choroidal in nature), showing itself as a fine translucent veil-like opacity in the retina, extending far out into the retinal periphery, with slight obscuration of the optic disk, is often seen. Here the retinal circulation is somewhat disturbed, as shown by venous tortuosity and distention, with arterial narrowing at times. Frequently, new blood-vessel formation, either with or without the association of whitish membranous bands, extends as irregular loops and net-work forward into the vitreous. Sometimes faint, almost invisible opacities can be seen in the vitreous humor; at other times innumerable flocculi float with every movement of the globe.

Vision is generally affected, but as a rule, in the minor cases, rapidly rises to normal in young and sthenic subjects under judicious alterative treatment.[1] Phosphenes, micropsia, and metamorphopsia have all been noticed. Prognosis, especially if the case be seen early and promptly treated, is favorable. Relapses, unfortunately, are apt to take place.

When the poison attacks the optic-nerve tissue itself, either peripheral neuritis appears or simple atrophy takes place. In these cases there is generally some cerebral or spinal-cord involvement. Such cases, however, are almost unique in children.

In the congenital type of syphilis, choroidal and retinal changes expressive of inflammation as a part of intra-uterine ocular disturbance may be recognized in addition to the other permanent sequelæ, such as posterior synechia, complicate cataract, etc.

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[1] At the present time the writer has under his care an eighteen-year old girl, who when first seen, three weeks ago, had a vision of $\frac{5}{15}$, which is rapidly approaching the normal under the use of mercurial inunctions. In this case the choroid does not appear to be involved.

In the hereditary form of the disease, where the ophthalmic symptoms appear in a seemingly healthy organ some time after birth, coarse changes in the choroid, retina, and optic nerve have all been noted by many observers. Irregular atrophic areas bordered by blackish pigment at times; dull, dirty red-gray disks, with diminished calibre of the retinal vessels; proliferation of pigment epithelium into the lymph-channels of the vessels of the retina, giving pictures closely simulating pigmentary retinitis,[1] have all been found.

Retinitis, not only as an extension from choroidal inflammation, as in Nettleship's most interesting case,[2] in a nine-year-old boy who gave an undeniable family history of contagion,[3] but also as a probable idiopathic disorder, has been recorded. Optic atrophy, without other gross ophthalmoscopic change, probably the result of some intracranial lesion or disturbance of the optic nerve, has also been seen.

In opposition to Cohnheim's assertion[4] that he has failed to detect tubercles in the choroid in similar conditions of the intestines and lungs, the same observer has incontestably shown their presence in *miliary tuberculosis;* this latter assertion being substantiated by both Manz[5] and Busch.[6] After most careful and painstaking investigation, Lawford[7] has even demonstrated the presence of the bacillus tuberculosis in the choroidal deposits.[8] Wadsworth also reports bacilli in a similar growth which had its initial point in the ciliary region. From this it is probable that the failure to find them by so many writers is dependent either upon the character of the tubercles, the peculiar idiosyncrasy of the patient examined, or the fault of the observer.

Probably the first ophthalmoscopic description of the presence of tubercles in the choroid was made by Von Jaeger,[9] followed by Von Graefe.[10]

They appear as nodules with whitish summits, gradually fading into a yellow tint, and at last assuming the color of the choroid. In size they are extremely variable, ranging from less than a millimetre to an area larger than that of the disk itself. Their number is very inconstant, ranging, as

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[1] At the present time the writer is studying such a case at Dr. Wm. F. Norris's clinic at Wills Eye Hospital. In this case the upper incisors are characteristic.

[2] Transactions of the Ophthalmological Society of the United Kingdom, ii. 60.

[3] In this case, Nettleship says that "the appearances in the left were like those in Plate V., Fig. 1, of Liebreich's Atlas; those in the right resembled Plate IV., Fig. 1."

[4] Archiv für Ophthalmologie, xiv. 1.

[5] Ibid., ix. 3.

[6] Archiv für Pathologische Anatomie, xxxvi. 448.

[7] Transactions of the Ophthalmological Society of the United Kingdom, vi. 348.

[8] Transactions of the American Ophthalmological Society, 1883.

[9] Oesterreichische Zeitschrift für Praktische Heilkunde, January, 1855.

[10] Archiv für Ophthalmologie, xiv. 1.

Cohnheim[1] has shown, from a single spot to more than fifty. Large aggregations are often present. If such massings be large, as in the accompanying monotint, taken from Lawford's case,[2] the retina will be markedly bulged forward by the overlying retinal vessels, whereas the smallest ones are invisible upon account of the overlying epithelium. As they grow, however, the superimposed tissues degenerate, and thus allow the tuberculous mass to become more and more visible, until at last from a doubtful shimmering area the nodule bursts into view as a prominent unpigmented spot. As a rule, the macular region is preferred (see the monotint).

In some instances these fundus-changes may antedate the appearance of the general inflammatory symptoms: thus, Fraenkel[3] reports such an exception, and Steffen[4] has seen them six weeks before the outburst of a tubercular meningitis. In view of these facts it is positive that were intraocular search instituted in all cases of incipient meningeal disease in young children, the diagnostician would much more frequently be able, by the knowledge of their presence, to determine if this were the true character of the intracranial disturbance. In such instances among children, very little is to be expected from subjective symptoms, because vision is not necessarily affected, and, when affected, is so only when the adjacent choroidal and retinal elements begin to suffer. Should the child have an acute attack of meningitis, the ophthalmoscopic picture may be quite different; the neuro-retinitis quickly establishes itself, the retinal arteries become pressed, the venous channels, as shown in Lawford's case, choke, and fine striated hemorrhages may even appear throughout the ground. This, however, is by no means a necessary accompaniment, as in a case of tubercles of the choroid diagnosed ophthalmoscopically one day before the patient's death from acute tuberculosis, Wells[5] found an almost utter want of intraocular disturbance. In this case Vernon[6] demonstrated the presence of the tubercles in the choroid by the microscope. Bearing in mind Stricker's observation (quoted by Gowers and Norris), that they may become recognizable in from twelve to twenty-four hours, we should not rest content with a single study of the fundus when it is important to note their presence.

Whether it would be wise to follow McHardy's example[7] in enucleating an eye containing a rapidly-increasing intraocular growth supposed to be a localized tuberculosis of the choroid, in order to prevent general infection, it is impossible to say. Both Mules[8] and Eperon[9] strongly advocate this

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[1] Berliner Klinische Wochenschrift, 1869, No. 4.

[2] Transactions of the Ophthalmological Society of the United Kingdom, 1883, p. 348.

[3] Jahrbuch für Kinderheilkunde, Bd. ii.

[4] Op. cit., 1870.

[5] A Treatise on the Diseases of the Eye, Amer. ed., by C. S. Bull, 1883.

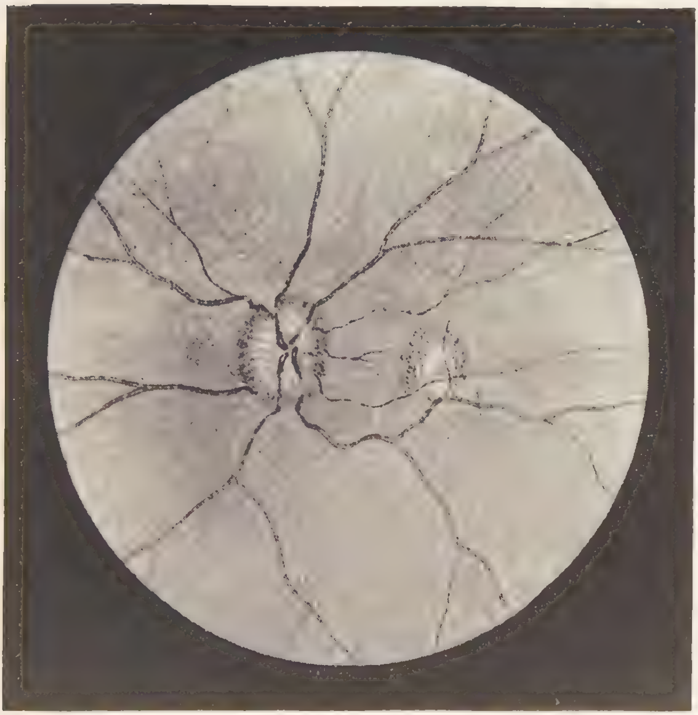
[6] Royal London Ophthalmic Hospital Reports, ii. 163.

[7] Transactions of the Ophthalmological Society of the United Kingdom, viii. 197.

[8] Ophthalmic Review, January, 1885.

[9] Archives d'Ophtalmologie, 1883, p. 485.

PHOTO. XVI.



Tuberculosis of the Retina and Choroid. (Lawford, Transactions of the Ophthalmological Society of the United Kingdom, 1886.)

procedure, the latter deeming it lost time to resort to either medicines or hygienic measures. At any rate, one must be very careful in attempting to decide this most difficult problem, which seems, to the writer's mind at least, a question that can be decided only by obtaining a consensus of opinion from several authoritative persons in each individual case.

Of much interest is Mules's instance in a ten-year-old girl.[1]

Very frequently the presence of the *gray tubercle* has been demonstrated in tissues of the *retina* and the *optic nerve.* One case is noted by Perls[2] in which the uveal tract seemed to be partially implicated. Chiari[3] reports an instance where the disk-tissue appeared infiltrated, causing the nerve-head to become quite prominent. Both Cruveilhier[4] and Hjort[5] show second-nerve deposition as far back as the chiasma. Lawford[6] cites a case of a five-year-old boy suffering from tubercular meningitis (see the case in greater detail in the section on the Choroid), where, in spite of the optic neuritis and the large aggregation of tubercular material in the choroid at the situation designated in the monotint, the retina and the optic nerve were uninvolved.

Leprosy.—This dread disease, with its definite bacillus, fortunately is so rare in this country that in reality it is known to us as a curiosity only. Its eye-symptoms, as a part of the general destructive conditions through which the tissues pass, are as multitudinous as the structures of the organ itself. Nothing seems to escape; nerve-substance becomes infiltrated with the peculiar cell-form, as shown by Virchow;[7] corneal tissue becomes invaded, as exhibited beneath the microscopes of Bull and Hansen;[8] whilst the choroid and even the retina, according to the researches of the same observers, do not escape. Although Pollock[9] justly tells us that "the disease of the eyeball is largely ciliary in origin," yet by continuity of tissue the choroid and retina at last become infiltrated, and in some instances total loss of sight and even destruction of the globe itself are brought about. In most cases, however, the degenerative processes in the anterior segment of the organ cause the ordinary sequelæ of shrinkage to appear, while in some others increased intraocular tension, with all its evil consequences, may ensue.

Upon account of the early disorganization of the tissues in the anterior part of the eye, the ophthalmoscope often fails to give any of the initial appearances of infiltration in the choroid and retina. Pollock,[10] however, asserts that no atrophic or pigment spots in the choroid have ever been observed ophthalmoscopically. Bull and Hansen[1] state that they often saw "a light grayish obscuration of the parts of the retina which surround the optic disk, with a relative tenuity of the retinal arteries, at post-mortem examination."

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[1] Medical Times, 1884, ii. 80.

[2] Archiv für Ophthalmologie, xix. 1.

[3] Wiener. Med. Jahrbücher, 1877.

[4] Anat. Path. Gén., 1862.

[5] Klinische Monatsblätter für Augenheilkunde, 1867, S. 166.

[6] Transactions of the Ophthalmological Society of the United Kingdom, 1886, p. 346.

[7] Krankhaften Geschwülste.

[8] The Leprous Diseases of the Eye, 1873.

[9] Leprosy as a Cause of Blindness, 1889, p. 76.

[10] Op. cit., p. 66.

LESIONS DEPENDENT UPON TOXICS INTRODUCED INTO THE SYSTEM.

In countries where *quinine* is used extensively, cases showing the toxic effects upon the second nerve have been noted. Thus, Knapp[2] gives an account of a female child of seven years, suffering from malaria, in whom frequently-repeated enemata of ten-grain doses of the drug caused blindness in six days, followed by phosphenes. Upon cessation, central vision gradually returned, but peripheral vision remained somewhat impaired. He cites two additional cases in boys, aged seven and eight years, where blindness soon supervened upon large doses of quinine, the loss of vision only gradually and imperfectly returning. E. Williams (*loc. cit.*) reports a similar instance after a single ingestion of a large dose, in a fourteen-year-old lad, who became totally blind in four days.

The blindness, which is sudden, is generally associated with a temporary deafness with tinnitus, which lasts about a day. The blindness, which is much more persistent, gradually lessens until good central and fair peripheric vision are obtained. Curiously, during the convalescence central color scotomata are said to appear.[3] At first the ophthalmoscope shows a retinal condition which closely simulates embolism.[4] The retinal vessels (both arteries and veins) are greatly contracted, and their contents can be removed by very slight pressure. Vorhies (quoted by Gowers)[5] asserts that he has found the choroidal vessels also empty. Gradual, though incomplete, restoration of both form- and color-vision takes place, the first appearing to be regained much the sooner. What the *rationale* may be it is not possible to say, though most likely it is dependent upon vaso-motor disturbances, giving rise to local changes. We must remember, however, as Norris[6] says, that "in many of the reported cases it is difficult to decide positively how much of the amaurosis is due to the quinine and how much to the disease for which the patient is under treatment;" he believing that "this is especially true where the patient has been suffering from severe intermittent fever or from exhausting hemorrhages complicating uterine disease, which are well known frequently to produce complete atrophy, with shrinking of the vessels."

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[1] Op. cit., p. 71.

[2] Archives of Ophthalmology, x. 220.

[3] Neul, Traité complet.

[4] See Gruening, Archives of Ophthalmology, x. 81, and Brown, Transactions of the Ophthalmological Society of the United Kingdom, vii. 199.

[5] Medical Ophthalmoscopy, 1882, p. 238.

[6] Pepper's System of Medicine, vol. iv.

One peculiarity of the disorder as spoken of by Gowers[1] is that, "whilst the symptoms are passing off, relapses may be produced by insignificant doses of quinine."

As amblyopia from *tobacco*, comparatively so common among male adults, may at times be found in younger subjects, though of course very rarely in children, a brief description of the symptoms will be given.

From the time of Mackenzie's assertion that most cases of amaurosis were caused by tobacco, which was considerably modified by Hutchinson,[2] to the latest analyses by Browne,[3] much has been written for and against it. The latest and most important researches by Uhthoff[4] show that the lesion is an axial inflammation with consecutive atrophic changes of the retro-bulbar portion of the optic nerve, generally close behind the globe itself.

In the incipiency of the attack, the optic nerve head appears somewhat œdematous and the veins of the retina are apparently tortuous. As the case progresses, signs of atrophic degeneration become manifest, the disk appears of a peculiar red-gray, with a decided loss of capillarity to the temporal side, and the retinal vessels diminish in calibre.[5]

Negative color scotomata, especially for green and red, either centrally or somewhat excentrically placed, are found early in the case. Later, they become larger, and either unite with the blind spot of Mariotte or extend some distance around the fixation-point. Both blue and yellow now rapidly disappear, until at last the scotomata become positive in type and cause a conscious defect[6] in the visual field. At this time the concentric limitation of the various fields, which has been progressively increasing, becomes very great for both form and the remnants of color-perception left.

Prognosis is always good when the case is seen early. Treatment consists in total abstinence from tobacco. Strychnine and the best of hygiene should be employed.

Alcohol amblyopia is rarely, if ever, seen in children. The ophthalmoscopic signs and the visual symptoms are almost identical with those of tobacco. In fact, this is to be expected, since the toxic effects of the drug are most probably upon the same strands of optic-nerve fibres.

Prognosis is favorable. Treatment consists in abstinence from the employment of the toxic agent.

A doubtful case of *theine* amblyopia is reported to have been seen in Russia.[7]

Salicylic acid, either by itself or in conjunction with some base, is said by Reiss to have given ocular symptoms simulating those of quinine. Gatli[1] notes an instance of temporary dimness of vision from a dose of one hundred and twenty grains of salicylate of sodium. The patient, a sixteen-year-old girl, was suffering from an acute attack of articular rheumatism. The fundus-oculi changes were limited to undue fulness of the retinal veins. Both pupils were dilated. Schiffer[2] gives an account of hallucinations of vision, lasting one day, following an enema of seventy-five grains of the same drug.

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[1] Medical Ophthalmoscopy, 1882, p. 239.

[2] Medico-Chirurgical Transactions, 1867.

[3] Liverpool Medico-Chirurgical Journal, January, 1888.

[4] Archiv für Ophthalmologie, 1886, iv., and 1887, 1.

[5] In many cases as seen in adults there are an indescribable tint and appearance of the nerve-head which seem almost pathognomonic.

[6] Infrequently, as, for instance, when the study of color-changes constitutes the work of the patient, trifling changes in green and red are recognized quite early in the case.

[7] Annual of the Universal Medical Sciences, 1888, iii. 126.

It frequently occurs, and in fact constitutes one of the physiological proofs of the assimilation of the drug, that *santonin*, given ordinarily as a vermifuge in children, produces xanthopsia, which lasts several hours. Of some importance, however, are the results upon the fundus oculi which are occasioned by much larger doses of the drug. In such instances, subnormal color-perception, associated with marked diminution of central vision, is more profound and lasting.

Mittendorf[3] reports an instance of visual impairment, with central scotomata for green and red, in an adult patient who had been taking from forty to sixty grains of *chloral hydrate* daily for six months. The nerve-head appeared "muddy." Upon the patient's ceasing to use the drug, and after the administration of strychnine, vision rapidly improved and the scotomata disappeared.

Noyes[4] mentions that "great and sudden amblyopia" has been caused by *osmic acid.*

Mytilotoxine, the poisonous ptomaine said to originate in diseased mussels (with physiological effects strongly resembling those of curare), is said by Dutertre[5] to produce visual troubles.

A very curious instance of blue discoloration of the eye-grounds has been seen by Litten[6] in a patient poisoned by *nitro-benzol* containing *aniline.* In this case the entire surface of the body was similarly discolored.

The toxic action of *lead,* like that of many of the other poisonous agents, produces either temporary blindness without any visible fundus-lesion, optic neuritis, or simple atrophy. The first, which is generally both sudden and extremely transient, fails to show any distinctive ophthalmoscopic signs. The second, which is quite pronounced on both sides as a rule, with numerous feathery hemorrhages from both contracted arteries and distended veins, is frequently recurrent in its intensity. At times there may be almost total annihilation of sight, lasting for a day or two. These losses of vision appear to have no relation to any visible changes in the fundus oculi, and seem to be associated with the first form of visual disturbance. Wadsworth[1] reports a very interesting case of double optic neuritis with ophthalmoplegia from lead-poisoning (complicated by typhoid fever) in a nine-year-old boy. Hutchinson[2] reports several instances of optic neuritis which were followed by atrophy. The third form, which may be either simple or consecutive, is manifested by a gradual decrease of the capillarity of the head of the nerve, with lessening of the retinal-vessel calibre, until sight is absolutely lost. Wells[3] gives the notes of a case of complete blindness from the consecutive type in a young woman, a worker in lead. The writer has at present a most interesting case of this type in an adult, who is fast becoming blind. Careful analysis of definite volumes of the urine, the saliva, the nasal mucus, and the tears, by Dr. John Marshall, revealed the presence of certain percentages of lead in every excretion but the last. The description of the fundus-changes and the transient peculiarities of the fields of vision in this case will be reserved for future publication.

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[1] Gazzetta degli Ospitali, Milan, 1880.

[2] Archives Mensuelles de Médecine et Chirurgie, 1887.

[3] Ophthalmic Review, October, 1888.

[4] A Text-Book on Diseases of the Eye, 1890, p. 634.

[5] Recueil d'Ophtalmologie, 1887.

[6] Centralblatt für Praktische Augenheilkunde, April, 1881.

In all the cases, cephalopathic symptoms are so frequently apparently visible that the utmost caution must be exercised before any absolute diagnosis is given. In fact, the presence of lead in the tissues and excreta, and the other pathognomonic symptoms of saturnine intoxication, such as wrist-drop, colic, etc., are the only certain evidences of the causative factor.

In many cases of prolonged poisoning, the renal apparatus is made to suffer to such a degree that both albumen and tube-casts can be readily detected in the urine. From this fact the query arises, Does lead-absorption mean endarteritis, with all its dire consequences, just as is found so frequently to be the case in so-called Bright's disease?

In both the neuritic and the atrophic form the prognosis is very bad. The amblyopic variety can be frequently bettered by the use of iodide of potassium.

The symptoms of the action of volatilized *mercury* upon the nervous system are well known. As a part of these, both optic neuritis[4] and atrophy[5] have been observed. The doubtful influence of acute hydrargyria upon the kidneys must be borne in mind in these cases.

True *argyria*, independently of the beautiful pictures of conjunctival staining as shown by Grossman[6] to have been produced by topical applications of strong solutions of nitrate of silver, has been found by Reimer[7] to have existed in the sclerotic sheath of the optic nerve. Gowers[8] states that "silver-poisoning is said to be accompanied by amblyopia in addition to the other symptoms of argyria." Again, it must be remembered that kidney-affections and vascular disease, which are so prevalent in certain forms of chronic metallic poisoning, may play important parts in the production of visual disturbance and optic-nerve inflammation.

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[1] Transactions of the American Ophthalmological Society, 1885, p. 54.

[2] Royal London Ophthalmic Hospital Reports, vi. 1, and vii. 1.

[3] A Treatise on the Diseases of the Eye, 1883 (American edition), p. 600.

[4] Galezowski, Des Amblyopies et Amauroses toxiques.

[5] Gatli, Gazzetta degli Ospitali, Milan, 1880.

[6] Ophthalmic Review, June, 1888.

[7] Quoted in Gowers's Medical Ophthalmoscopy, 1882, p. 239.

[8] Op. cit., p. 239.

Arsenical poisoning, like lead-poisoning, is apt to give rise to marked nervous symptoms. As with mercury, the usual mode of entrance of arsenic into the system is by inhalation. Dana, however,[1] reports a case of optic neuritis with other nervous groupings following an acute poisoning by the drug. Seguin,[2] in speaking of chorea and its treatment by arsenic, says that he has never seen symptoms of multiple neuritis or of optic neuritis from the medicinal use of arsenic.

In addition to the ordinary toxic effects of the constant inhalation of the vapor of *bisulphide of carbon* and *chloride of sulphur*, ophthalmic symptoms have been noted. Nettleship[3] cites an instance of a young man of twenty years who had been employed in an india-rubber works for ten months, whose vision had failed to $\frac{5}{70}$, with a central defect for red. The visual fields were said to be normal, whilst the optic disks were pale and slightly hazy, these symptoms being partly relieved by discontinuance of work. Huguenin[4] gives two cases, the first in a fifteen-year-old boy, who, after four months' exposure, had failure of vision and was unable to recognize green. Nine months later vision was bettered. The second case, a girl who had been exposed for two years, had "chromopsia" (red and green), with failure of sight. Becker[5] notes a most instructive instance.

In nearly every instance the patient gazes as it were through a fog, this symptom becoming more pronounced when the patient is tired or fasting. Central scotomata have been found, whilst the optic nerve itself frequently shows signs of a low grade of chronic inflammation. Prognosis is, as a rule, good. Treatment consists in the avoidance of the fumes of the drugs, together with the use of both local and general hygiene. During the attack, Nettleship employed strychnine in his case, seemingly to advantage, after having unsuccessfully tried "the constant current." Lavigerie[6] claims good results from the use of strychnine and iodide of potassium.

UNCLASSIFIED.

As shown by Leber,[7] *retinitis pigmentosa* is a chronic disturbance, which consists in proliferation of connective material associated with degeneration of nerve-tissue and wandering of pigment-massings into the substance of the retina. From this last objective condition of the disorder, which in reality is sometimes wanting (?), the disease receives its name.

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[1] Brain, ix. 456.

[2] New York Medical Journal, April 5, 1890.

[3] Transactions of the Ophthalmological Society of the United Kingdom, v. 149.

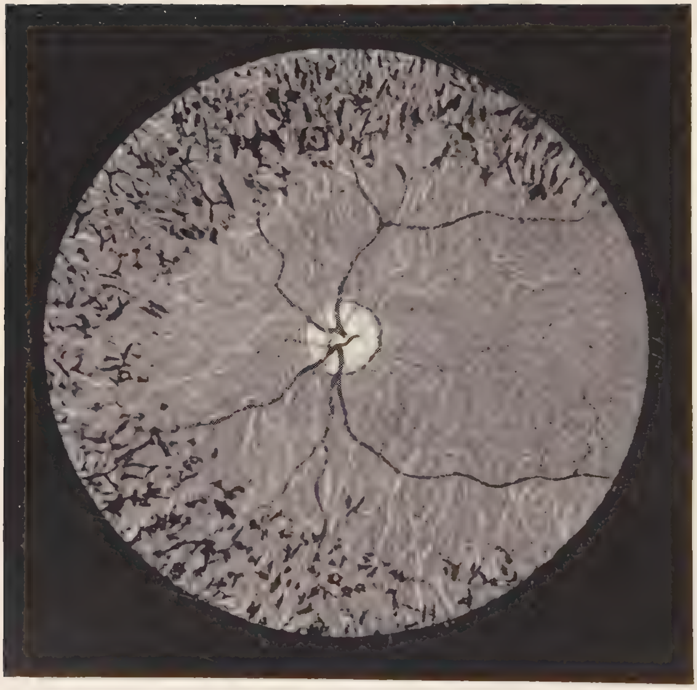
[4] Thèse de Paris, 1874.

[5] Centralblatt für Praktische Augenheilkunde, May, 1889.

[6] Jour. de Médecine et Chirurgie, 1887.

[7] Graefe und Saemisch, Handbuch der Gesammten Augenheilkunde, Band v.

PHOTO. XVII.



Retinitis Pigmentosa. (Jaeger, Beiträge zur Pathologie des Auges, Plate XXXVII.)

These conditions, which are more extensive in the peripheral portions of the retina, gradually advance towards the region of the yellow spot. Atrophic degeneration of the optic nerve extending outwardly soon shows itself ophthalmoscopically, whilst the degeneration areas even pass inwardly beyond the point of intracranial crossing.

Ophthalmoscopically, the fundus-oculi changes are very apparent. As shown in the accompanying monotint, taken from Jaeger's Atlas, the pigmentary deposit lies far out in the periphery of the ground and stands comparatively well forward in the retinal layers,—the massings, as a rule, having a much less decided preference for the temporal side of the ground. The pigmentation itself, as can be readily seen, assumes a distinctly bone-corpuscle-like appearance. Ofttimes these massings seem to follow the course of the main retinal stems. Throughout the eye-ground, though less pronounced in the macular region, there is marked absorption of the pigment epithelium, which allows the larger underlying choroidal vessels to be plainly visible. At times the retina itself between the pigment-aggregations appears as a grayish film. Both series of retinal vessels are small, with thickening and opacification of their walls.[1] Curiously, the tissues in the macular region seem to remain intact for a long time.[2] The nerve-head, which at first is reddish gray in tint, becomes more and more gray, until at last it assumes a dull-white appearance.

In the early stages of the disease, an increasing inability to see properly in dim light manifests itself, this incompetency of vision frequently showing itself by the patient's stumbling over large objects situated in unaccustomed places. Again, the patient will assert that there is a gradual lessening of the area of vision. As the case grows worse, both peripheral and central vision fade, until at last absolute (or almost complete) blindness ensues.[3] Fortunately, in some cases the condition remains stationary after reaching a certain point.

The disease is said at times to be congenital, or it may appear early in life. There can be little doubt that it is distinctly hereditary. The writer has notes of six cases in four generations, the order of sequence being from an affected mother to three affected grandsons and one affected grand-daughter by an unaffected father; another (the first) daughter (unaffected) of this father having one boy and one girl,—the boy being affected. Consanguinity existed in this grouping.

Prognosis as to betterment of sight is always bad. Although treatment has been found of little or of no value, yet Hasket Derby's and Myles

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[1] The sketch does not show this.

[2] Even in the earliest stage this is not strictly true, because if careful testing for central color-perception be made at that time, evidences of slight green and red subnormal perception can be determined in most instances. In contradistinction to this, however, hyperæsthesia has been asserted in a few cases,—possibly from primary abnormal excitation through slight exacerbations of chronic low-grade inflammatory changes.

[3] At times these clinical groupings may be interfered with, giving rise to all manner of anomalous symptoms.

Standish's suggestions[1] for the use of electricity should be conscientiously tried in every instance. Alteratives in association with the best hygienic measures must always be employed.

The works of the older writers teem with imperfect histories of cases of so-called *hemeralopia*. Wonderful, however, is the accuracy with which intraocular conditions were guessed at without the use of the ophthalmoscope, and most interesting are the accounts of sailors, soldiers, workers before bright glares, travellers under the tropic sun and through the arctic snows, who are supposed to have been subjects of the disease.

The conditions sometimes appear connected with scurvy, interference with the functions of the liver, starvation, etc. Forry[2] thinks that it is rare in the United States, and that it is much more prevalent in the Southern States than in the Northern.

In the great majority of cases ophthalmoscopic signs are wanting, though Wells[3] has seen a slight dilatation of the retinal veins. The pupils are said to be dilated, and the irides are generally noted as sluggish to light-stimulus.

The patients declare an inability to recognize objects by feeble illumination. At times both peripheral vision and central color-perception seem to be below normal.[4] Both negative and positive scotomata have been found in the visual fields. Phosphenes and subjective after-colors have been noted. Both eyes are affected.

The disease appears suddenly. Prognosis is good. Treatment consists in tonics, taking care to give the patients the best of hygiene and to place them under the influence of subdued light, as, for instance, in cool, comfortable, darkened chambers, or, better, to protect their eyes by smoked glasses.

Nettleship[5] notes the case of a patient with "stationary night-blindness with minute white spots at the fundus." In this case the fundus oculi seemed to be studded with small non-pigmented white dots, these being less pronounced in the macular region. The patient, aged twenty-one years, complained that he had had difficulty in seeing in the dark for as long a time as he could remember. Gayet[6] has described two similar instances under the supposititious title of "Retinitis Pigmentosa."

On account of the elasticity of the ocular tissues and the freedom of fluid interchange, *glaucoma* in childhood is very rare. Most of the few instances recorded show either progressive myopia with stretched ocular walls, or incarceration of inflammatory material in situations where constant secretion and excretion are taking place.

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[1] Transactions of the American Ophthalmological Society, 1887, pp. 555, 556.

[2] American Journal of the Medical Sciences, April, 1842.

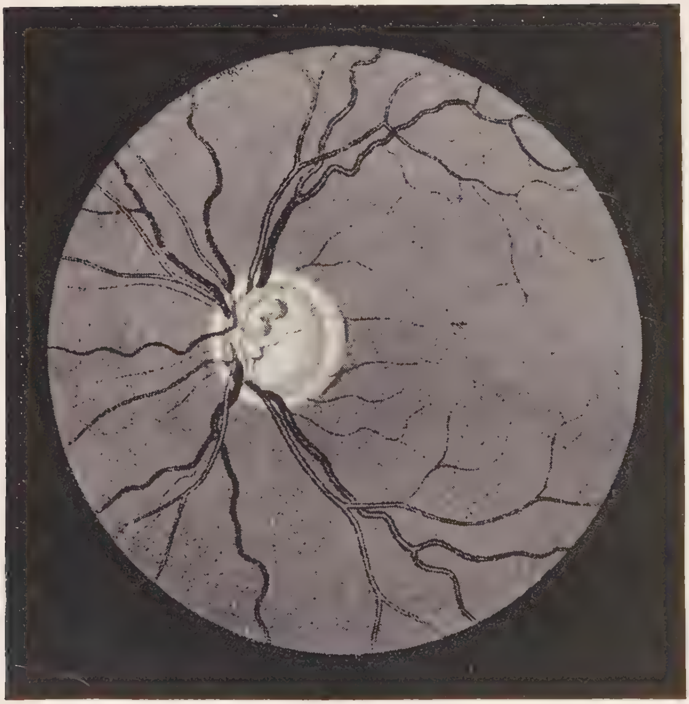
[3] A Treatise on the Diseases of the Eye, 1883 (American edition), p. 606.

[4] Foerster (Ueber Hemeralopie, 1857) has found that blue, violet, and red are the colors that are the most difficult to recognize by these cases.

[5] Transactions of the Ophthalmological Society of the United Kingdom, 1888, p. 163.

[6] Archives d'Ophtalmologie, 1883, p. 386.

PHOTO. XVIII.



Glaucomatous Excavation. (Jaeger, Beiträge zur Pathologie des Auges, Plate XVIII.)

The writer has seen two instances, one of the secondary type and the other of uncertain origin, at Dr. Wm. F. Norris's out-patient service department at Wills Eye Hospital. Both patients were boys, one aged eleven and the other thirteen years. In the younger child the changes in the anterior portion of the globe were so pronounced[1] that it was very difficult to study accurately the pathological excavation in the nerve-head. Tension was increased.

In the second case, without any history of accident, the sight of the right eye was said to have been "always poor." Here also intraocular tension was increased (+ T 1). The field of vision was markedly contracted to the nasal side: the pupil was larger than that of the left eye, and the iris was somewhat sluggish to light-stimulus. Vision with this eye equalled one-ninth, which was increased to one-sixth by a correction for a low amount of mixed astigmatism. A shallow[2] though well-marked glaucomatous excavation, more pronounced to the temporal side of the disk, could be plainly seen with the ophthalmoscope. No marks of traumatism or of opacity were discernible. The fellow-eye had a vision of one-half, which was bettered by a weak convex cylinder. The visual field was contracted in the same way as that of the opposite eye, but not to the same extent. Accommodation was fair, though, as in the other eye, it plainly indicated spasm. No glaucoma cup could be determined positively. The intraocular tension was apparently normal. The better eye was the fixing organ, the fellow-eye wandering out during the act.[3]

Many of the cases of ciliary and corneal staphylomata and buphthalmos seen in the young are mere expressions of what would be glaucoma in older subjects under similar circumstances.

The ophthalmoscopic picture of glaucoma, which is so well represented by the accompanying reproduction of one of Jaeger's plates, is typical of increased intraocular tension. The soft substance of the nerve-head is pushed backward against itself, exposing the sclerotic ring with its sharply-cut edge, against which the retinal vessels are forced. In the bottom of the pathological excavation the flattened veins can be dimly seen with the lens that renders the scleral edge of the nerve plainly visible. By gradually weakening the focussing power of the ophthalmoscopic lenses, the details of the bottom of the pit can at last be brought into distinct view. Upon account of the increase of the intraocular tension giving greater obstruction to the arterial current as it enters the globe, the arteries appear small, whilst the corresponding veins, for the same reason, seem wider and more flattened.

Often the excavation is only partial; in such cases, as a rule, it is to the temporal side of the disk. Again, an original physiological cupping with an underlying or a circumscribing pathological excavation may give a terraced appearance to the depression. Spontaneous venous pulsation is often present, and an arterial pulse may be easily obtained by pressure with the finger upon the eyeball.

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[1] Anterior leucoma with ring-like opacities in the lens, the sequelæ of a traumatism when the patient was six or seven years of age.

[2] One diopter.

[3] Careful examination showed that this was dependent upon insufficiency of the interni.

The visual field is frequently characteristic, the contraction, as a rule, first appearing to the nasal side.[1] Visual acuity is usually very faulty.

Prognosis is certainly bad, and treatment to be of any value must consist in iridectomy. This, however, should not be done without the advice of some competent authority and without giving the patient's friends a clear understanding of both the immediate and the remote dangers of the operation.

In countries such as North Germany, where various preparations of raw pork are eaten, the parasite *cysticercus cellulosæ* has been found between the retina and the choroid. When in this position the retina rapidly becomes detached and the overlying tissues grow opaque and turbid, whilst surrounding inflammatory change and atrophic degeneration soon take place. In all such cases, even before any gross local changes have appeared, the presence of the parasite cannot be determined with any degree of accuracy, although Stellwag[2] says, "In some rare cases the head and neck are rarely seen through the opacity as a movable, deeply-clouded mass." Ofttimes the entozoon becomes encapsulated. Becker,[3] Schweigger,[4] Jacobson,[5] and Devencentiis[6] have all seen such cases.

Prognosis is always bad, even though a considerable number of successful attempts at extraction of the parasite have been made by Alfred Graefe and others.

As gross *congenital subnormal color-perception* (*color-blindness*) exists among us in so great a degree, a few words are necessary for its study and recognition. The colors most frequently confounded are green and red; for instance, a red berry is not so distinctly separated from the green leaves by its color-difference as it is by the comparative intensities or strengths of the colors themselves and the differences of form of the two objects. The proper color-designation may be given in each case, but should color-comparison be attempted, as, for instance, with a number of red and green berries of the same intensity of color, or a quantity of red and green leaves of similar intensities, both the berries and the leaves would be hopelessly confounded, since here, in each case, color-perception alone is called into play.

Many accurate accounts both of historic and of scientific interest could be given, as, for instance, Huddart's case[7] of the shoemaker Harris, who when a child could distinguish ripe cherries from green leaves in no other way than by their difference in size and shape, and Nicholl's case[1] of a boy who was found to possess subnormal color-perception for both green and red.

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[1] This test cannot always be depended upon, as there are exceptions.

[2] A Treatise on the Diseases of the Eye, 1868, p. 474.

[3] Zeitschrift der Wiener Aerzte, 1865.

[4] Archiv für Ophthalmologie, vii. 2, 53.

[5] Ibid., xi. 2, 148.

[6] Annali di Ottalmologia, vol. xvii., No. 1.

[7] Philosophical Transactions, London, 1777, lxvii. 260.

As examination of the color-sense has not been made a routine measure in any of our public or private educational institutions, the complaint is most frequently brought to our notice by the subject himself. Examination by some one of the plans of loose-wool selection, if conscientiously done, will soon reveal the defect. Treatment is of no avail; but careful training among colors and shades should be given to all young persons who are known to have such a defect, in order that a compensatory power, in an ability to recognize color-differentiations by the finer and more delicate choices of shades and intensities, especially in employments which require such discrimination, as, for instance, photographing, engraving, etching, etc., may be imparted to them,—a training that will render them better able to perform such work than those who possess ordinary color-perception.

It will be remembered that whilst speaking of hysteria and its blindness the unintentional type was noted as of frequent occurrence. Here, however, pure malingering, or conscious *simulated blindness*, which is more difficult to detect, will be briefly considered. Odd as it may appear, scholars desirous of escaping routine school-work, and children with a wish for sympathy or condonement, have calmly but strenuously persisted in the assertion of either complete or partial blindness, without any detection by ordinary means at hand. If the child be old enough, various procedures with prisms which produce double and erroneous projections of objects, or convex lenses and mydriatics which either so alter the focussing power of the two eyes as to render binocular fixation impossible, or exclude the avowedly good eye from action, should be made by some competent person. Artificial anæsthesia may also be tried, so that an attractive object may be offered to the supposed malingerer before he has sufficient command of his intellect to continue the fraud.

As it may be not only of interest, but possibly of medico-legal value, to have additional means of recognizing the certainty of *general dissolution* in the young beyond the persistence of muscular excitability to electric stimuli and the failure of the ordinary signs of decomposition, it has been thought fit to insert a few words giving some of the most important *ophthalmoscopic changes which can be seen in such cases.* Should careful study be made, the retinal arteries will be found to decrease steadily in size during each successive weaker impulse of the heart; the disk-capillaries will rapidly disappear; the substance of the nerve-head appears more and more blanched; the choroid pales; the blood in the retinal veins breaks into beaded currents and disappears; and, lastly, the retina becomes rapidly opaque. This last change, which may be made more noticeable for several hours after death by constantly moistening the surface of the cornea, is unequivocal in its significance. At this point, however, the media become so opaque that further examination of the fundus is impossible.

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[1] Medico-Chirurgical Transactions, 1818.

Gayet[1] has been so fortunate as to notice a red spot at the macula lutea of the same character as that which can be seen in cases of embolus of the central artery of the retina,—this condition, no doubt, being dependent upon the increasing haze of the retina permitting the reddish-yellow reflex of the underlying choroid to be last seen at the thinnest portion of the sentient membrane, the fovea centralis. Kyerson,[2] who was enabled to study the fundus-changes in the eye of an injured man who was dying, gives a most interesting account of the ophthalmoscopic details. In this instance it is noted that there was occasional venous pulsation. Careful studies have also been made by Bouchut,[3] Poncet,[4] Schreiber,[5] Arlidge,[6] and others.

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[1] Annales d'Oculistique, 1875, lxxiii. 1.

[2] Canada Lancet, April, 1888.

[3] Traité des Signes de la Mort, 1863.

[4] Archives Générales de Médecine, 1870, 6, xv. 408.

[5] Deutsches Archiv für Klinische Medicin, xxi. 100.

[6] West Riding Asylum Reports, 1871, i. 73.

PART III.

HYGIENE.

PHYSICAL DEVELOPMENT.

By J. M. KEATING, M.D.,

AND

J. K. YOUNG, M.D.

In considering the subject of physical development in children, the normal development of the child, the physical type of man, increased physical development, and the influence of physical development in the treatment of deformity and disease, will all be included.

M. Laine, Darwin, Chaillé, and others have investigated the dawn and development of the intellectual faculties; Russow, Hähner, Zeising, Stephenson, and others have observed the general increase in stature and weight; the physiology of infancy has been dealt with elsewhere. There remains the normal physical development through the succeeding periods of infancy, childhood, and youth.

"If we are to devote our attention, before all things, to what can be measured and weighed, the living man is the first object which demands our investigation." (Carl Vogt.)

MEASUREMENT OF THE HUMAN BODY.

This leads naturally to the consideration of the physical proportions of the body, of the measurement and strength of different individuals, or of those of the same individual at different periods of life. This investigation, or the study of anthropometry, does not at this early period assume the importance which it has in adult life. Later, the political aspect of the subject, bearing on the recruiting for the army and navy, the scientific importance of the effect of climate, seasons, and peculiar hygienic conditions, and the classification of the various races of mankind, are to be considered. It is, however, of distinct social importance in ascertaining the proportions most favorable to health, longevity, and physical endurance, in the diagnosis of diseases, accidents, and deformities, and in the insurance of lives and the fitness for certain duties.

Naturalists have concluded that the best means of classifying the races and varieties of mankind are measurements of the different dimensions of the body, and, in a more restricted view of individual interest, measurement constitutes the best means of ascertaining the changes occurring in the conformation of the body under the influence of age, sex, nurture, occupation, sanitary conditions, and the general effects of physical culture and athletic sports upon the economy.

Anthropometry has at the present day a voluminous literature of its own.[1] Its entire history is an attempt to establish a standard by which to determine and compare the proportions of the human body. From prehistoric antiquity the hand, foot, and cubit (forearm) have been convenient standards, possessed by every one and of sufficient accuracy to enable man to adjust his dealings with his fellow-man and construct his places of residence or of worship. These rude standards served for ages, and are still retained by civilized nations, notwithstanding the precise standard which they now possess through the perseverance and skill of the French mathematicians.

The early attempts made to ascertain the average foot and average cubit for the purposes of trade led to series of measurements, which, being admitted as standards, naturally induced artists and sculptors, in the earliest days of art, to endeavor to deduce the exact proportions of the perfect human form.

In the "Silpi Sastri," or Treatise on the Fine Arts, the earliest known Sanscrit manuscript, the human figure is divided into four hundred and eighty parts, the head being nearly a seventh part, conforming to the best standards of the present day:

PROPORTIONS OF THE HUMAN BODY, FROM THE "SILPI SASTRI."

The hair	15 parts.	From the umbilicus to the pubes	53 parts.
The face	55 "	From the pubes to the knee	90 "
The neck	25 "	The knee itself	30 "
The chest	55 "	The leg and foot	102 "
From the chest to the umbilicus	55 "	Entire height	480 parts.

It is highly probable that the Egyptians early possessed a standard of proportion, for Rosellini and Lepsius, after a careful examination of the figures found upon Egyptian monuments, showed that the artists conformed rigorously to a definite scale of proportions, and Lepsius has deduced three canons of measure in use at different periods.

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[1] See for complete bibliography Dr. J. H. Baxter's Statistics, Medical and Anthropological, of the Provost-Marshal-General's Bureau of the United States, vol. i., and Roberts's Manual of Anthropometry.

While one naturally turns to the matchless works of the Greek artists for examples of perfect symmetry, it is to be regretted that no writings of Greek authors treating especially upon the proportions of the human body have been preserved. It is known that at a very early period a system, rigorously minute in detail, had been introduced from Egypt, for Diodorus Siculus informs us of the construction of the Pythian Apollo, the two halves of which were executed by two sculptors in different cities, one being at Samos and the other at Ephesus. So exact were the details of the system that upon uniting the separate portions the statue proved to be a marvel of perfection and symmetry.

The statue of the celebrated sculptor Polykleitus known as "The Canon," but called also, from the subject, Doryphoros, or "The Spear-Bearer," was constructed upon an admirable theory of proportion, and was believed by the sculptor and his pupils and admirers to be absolutely perfect in form. Its effect upon Greek and Roman writings was marvellous and long-continued, although neither the statue nor a copy of the treatise describing it remains.

Vitruvius, the Roman writer on architecture, has incidentally given a partial account of proportions which were long considered authoritative. He writes:

"The human body, as nature composed it, has this proportion, that the face, which includes the space from the chin to the top of the forehead, where the roots of the hair begin, is a tenth part of the whole height; it is the same length from the wrist to the tip of the middle finger. The head, from the chin to the top of the skull, is one-eighth part; the same to the pit of the neck. From the top of the chest to the roots of the hair is one-sixth part, and to the top of the head one-fourth. The third part of the face is from the bottom of the chin to the lowest part of the nostrils; one-third from there to between the eyebrows; one-third from this latter to the roots of the hair, where it begins on the forehead. The foot is one-sixth part of the whole height, the cubit one-fourth, the chest (across the shoulders?) the same.

"The other members have each their measures and proportions, by which the greatest of the ancient painters and sculptors who have won signal honors have guided themselves. In the same way the parts and body of a temple have definite laws of proportion.

"So, too, the navel is naturally the centre of the body; for, if a man be laid upon his back, with hands and feet extended, and his navel be taken for the centre, the circumference of a circle so drawn would touch the extremities of his fingers and toes.

"Not only is the scheme of the circle found in the body, but also the scheme of the square; for, if the distance from the soles of the feet be taken to the summit of the head, and be applied to the hands outstretched, it will be found that the length and breadth are equal as a perfect square."

Though serving as the groundwork for subsequent works, many of these measurements are undoubtedly incorrect; notably, the position of the umbilicus as the centre of height in the adult, and the distance from the top of the sternum to the summit of the cranium.

The statement that the perfection of Greek statuary was due to the superiority of the living models from whom they were designed receives in the exact statistical data of man-measurements in our day a more decided denial than æsthetic criticism could ever have produced.

We have the authority of M. Quetelet for the statement that the physique of man to-day compares favorably with that during the time of the early Greeks. After a careful comparison of the dimensions of the best masterpieces of antiquity with the average results of modern statistical research upon the living, he declares, "It is, then, wrong to suppose that man in our clime differs essentially from the structure observed in the Greek statues. The delicacy and beauty of feature, the expressiveness of countenance, the elegance of form, may be inferior without the proportions of figure being different on that account. Everything tends to establish, on the contrary, that the human type in our clime is identical with that deduced from observation of the most symmetrical ancient statues."

The early authors employed but few models to determine the size and conformation of the parts, but took infinite precautions to unite exactitude of form with elegance of proportion. Phidias employed twenty models, it is said, to arrive at elegance, selecting from each the most beautiful parts, and arranging them according to his knowledge of the human form.

During the era of the Renaissance the canon of Polykleitus retained its influence, as is shown by the artificial nature of all the systems propounded. A part of the body, the cubit, hand, foot, head, face, or nose, was selected as the unit or basis of calculation, and every other part had a forced relation thereto.[1]

The character and limits of this sketch will not permit more than an allusion to the artists, sculptors, anatomists, mathematicians, and others who since the Renaissance have contributed theories or treatises upon the proportions of the human body, but a short description of the models and treatises of Alberti, Albrecht Dürer, Reynolds, Carus, Story, and Quetelet will serve to illustrate the progress of the subject to the present day.

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[1] "The standards of the proportions of the body employed by ancient and Renaissance sculptors and artists were taken from different parts of the body; and, although they are not of much value to science, they are full of interest to those who appreciate their incomparable works of art. They were the *cubit* of the Egyptians, or the distance between the elbow and the extremity of the fingers; it forms the fourth part of the height of man. The *foot*, which forms the sixth part. The *head*, which, according to Vitruvius, forms the eighth part; but, properly speaking, the head is contained seven and a half times in the height. The *face* (volto), which is equal to the length of the hand, and is the ninth part of the total height." (Roberts, loc. cit.)

Alberti may be said to have followed the canon of Vitruvius in taking the foot to be one-sixth of the entire height, and thus rendered his scheme defective. The adoption by him of the average or mean and of a decimal system of division is, however, noteworthy.

The peculiar feature of the measurements of Dürer is that they represent three points of view,—profile, front, and back; and Schadow, the author of the "Polyclet," expresses the opinion that his model figure was the result of calculation, and not of actual measurements of living subjects.

Science owes to Sir Joshua Reynolds the idea of the existence of a typical form in man, and the order which prevails in the apparent variations from that type. "All the objects," says he, "which are exhibited to our view by Nature, upon close examination will be found to have their blemishes and defects. It must be an eye long used to the comparison of these forms, and which, by a long habit of observing what any set of objects of the same kind have in common, has acquired the power of discerning what each wants in particular. By this means we acquire a just idea of beautiful forms; we correct Nature by herself, her imperfect state by her more perfect, and make out an abstract idea of forms more perfect than any one original. . . . From reiterated experience and a close comparison of the objects of Nature, the artist becomes possessed of a central form from which every deviation is deformity. . . . To the principle I have laid down, that the idea of beauty in each species of being is an invariable one, it may be objected that in every particular species there are various central forms, which are separate and distinct from each other, and yet are undoubtedly beautiful; that in the human figure, for instance, the beauty of Hercules is one, of the Gladiator another, of Apollo another, which makes so many different ideas of beauty. It is true, indeed, that these figures are each perfect in their kind; but still none of them is the representation of an individual, but of a class. And as there is one general form which belongs to the human kind at large, so in each of these classes there is one common idea and central form which is the abstract of the various individual forms belonging to that class. Thus, though the forms of childhood and age differ exceedingly, there is a common form in childhood and a common form in age, which is more perfect as it is more remote from peculiarities. But I must add further, that, though the most perfect forms of each of the general divisions of the human figure are ideal, and superior to any individual form of that class, yet the highest perfection of the human figure is not to be found in any one of them. It is not in Hercules, nor in the Gladiator, nor in the Apollo; but in that form which is taken from them all, and which partakes equally of the activity of the Gladiator, of the delicacy of the Apollo, and of the muscular strength of the Hercules. . . . There is, likewise, a kind of symmetry or proportion which may properly be said to belong to deformity. A figure lean or corpulent, tall or short, though deviating from the type, may still have a certain union of the various parts which may contribute to make them on the whole not unpleasing."

It remained, however, for M. Quetelet to reduce this artistic conception to a scientific basis, and demonstrate its soundness and usefulness by extensive and painstaking observation on living models.

Before considering the methods of M. Quetelet, which, being based on the inductive, strongly recommend themselves to every student, we must consider the work of Prof. Carus, a distinguished physiologist of Dresden, which is based on the deductive method of investigation.

He assumed the hand's length for his unit, dividing it into twenty-four parts, and considered the true key to our proportions to be the vertebral column, consisting of twenty-four free vertebræ,—"the true organic ell, divided into twenty-four inches." He confirmed his view by observing that in the egg of mammals the first indication of the future animal was a rayed line which subsequently becomes the spinal column, and also by observing that a ratio of length exists in the vertebral column of the new-born infant and the adult, the length of the former being exactly one-third the length of a line drawn perpendicularly from the spinous process of the atlas to the spinous process of the last lumbar vertebra in the adult. This standard, or "modulus" of a third of the length of the adult spine, furnishes, when divided by the authoritative measure of twenty-four, all the dimensions required to deduce the perfect form. Upon this he constructed a figure, a sexless statue, but one capable by a slight variation of being modified to represent either sex, and capable also, by the application of certain rules, of representing a dwarf or a giant, a poet, a philosopher, or an athlete. The "modulus" may, indeed, be theoretically correct, but for scientific purposes it renders no assistance to the knowledge of the progressive development of the body, as the different parts of the body do not develop with the same rapidity.

In 1866 Mr. Story, the sculptor, proposed a new canon, which in ingenuity of detail and beauty of result is equal to any of its predecessors. To obtain this canon he directs that one-fourth of the entire height of the intended figure be laid down as the side of an equilateral triangle. "The triangle being completed, from its apex a line is to be dropped, bisecting the base, and extending below it a distance equal to one-third of its length above it; this line forms the diameter of a circle, in which circle is inscribed a square. The diagram thus consists of a triangle and a square enclosed in a circle; and when the lines of these figures are divided into thirds, fourths, etc., a vast number of dimensions are obtained, and in them all the measurements of the intended figure are to be found."

From the "Silpi Sastri" of India to the "improved canon" of Story, it is instructive to observe what ingenuity and labor have been expended in these attempts to reduce human proportions to an exact system, and to notice the fallacy which pervades them all,—"that the key to the theory is to be found in the occult relation of numbers or in the parts of a geometrical diagram."[1]

It remained for observers like M. Quetelet, the distinguished mathematician, discarding theories, to recognize the existence of a central or typical form of man, *l'homme moyen*, as the *mean* result of large numbers of actual measurements of living men, combining the æsthetic conception of the "central form" of Sir Joshua Reynolds with the scientifically deduced "canon" of typical proportions of Prof. Carus.

"A large number of naturalists and philosophers," M. Quetelet says, "have attempted to prove, by a course of reasoning which is more or less conclusive, the unity of the human species. I believe that I have succeeded in demonstrating not only that this unity exists, but that our race admits of a type or model the different proportions of which can be easily determined.

"If there were an absence of type, and if men were unlike one another, not from the effects of accidental causes, but because no common law really existed among them, they might be measured, as regards height, for instance, without all the individual measurements offering any particular character or any definite numerical relation.

"If, on the contrary, all men have to a certain degree been cast in the same mould, and if they issue from it with differences which are purely accidental, the groups will no longer be formed in an erratic manner, but their numerical values, in accordance with the theory of probabilities, will be subject to pre-established laws, so that the numbers which represent each man can be determined *a priori.* There exist, therefore, for this entirely special case, characteristics by which we may recognize whether individuals belong to the same type and only differ owing to fortuitous causes. Another consequence of the theory is, that the greater the number of observations the more do fortuitous causes explain each other, and make the general type, which they at first tend to screen, stand out prominently. Thus, in the human race, when individuals only are considered, all heights are met with, at least within certain limits; those who come nearest the average are the most numerous; those who deviate the most from it form the smallest number; and the groups follow numerically a law (the binomial law of Newton) which may be laid down beforehand. In the case of man this law is verified not only in relation to the entire height, but also as regards his various members; and the same is the case with the weight, or strength, or any quality which can be measured and reduced to numbers."[2]

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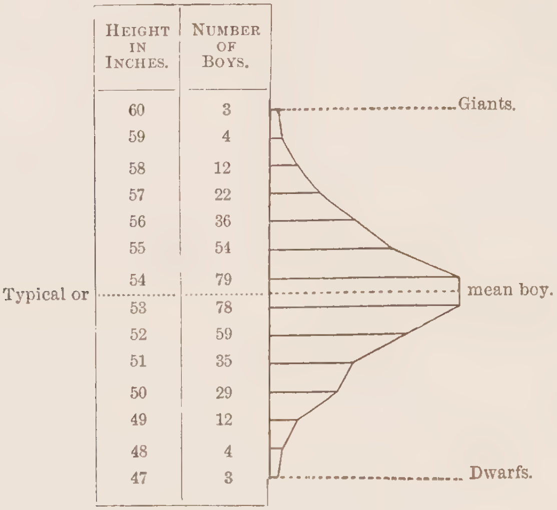
[1] Baxter, op. cit., p. lxix.

[2] *Mean and Average.*—The distinction between a mean and an average is often overlooked, or not clearly comprehended. Sir John Herschel so clearly exhibits it that the passage is worth quoting entire. Speaking of M. Quetelet's *homme moyen*, he says, "Now, this result, be it observed, is a *mean*, as distinguished from an *average*. The distinction is one of much importance, and is very properly insisted on by M. Quetelet, who proposes to use the word mean only for the former, and to speak of the latter (average) as the 'arithmetical mean.' We prefer the term average, not only because both are truly arithmetical

The following diagram, taken from Roberts's "Anthropometry," representing the heights from actual measurement of four hundred and thirty English public-school boys from eleven to twelve years old, will illustrate Quetelet's views:

Fig. 1.



Height in Inches.	Number of Boys.
60	3
59	4
58	12
57	22
56	36
55	54
54	79
53	78
52	59
51	35
50	29
49	12
48	4
47	3

"It will be seen," says Roberts, "that the numbers arrange themselves according to a very uniform rule; the most numerous groups are in the middle of the column, at 53 and 54 inches, while the groups at 52 and 55 inches are less in number, and those at 51 and 56 inches are still fewer, and so on till the extremely small number of the very short and very tall boys of 47 and 60 inches is reached. It is thus ascertained that the mean or typical boy of the class and age given is 53.5 inches, and, as representing the most numerous group, he forms the standard, from which the other groups of boys decrease in number as they depart further and further from his proportions.

"In the diagram, which has been drawn to a scale, the length of the horizontal lines (*abscissæ*) represents the numbers of boys in each group, and the curved line binding the ends together is the well-known 'binomial

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means, but because the latter term carries already with it that vitiated and vulgar association which renders it less fit for exact and philosophical use. An average may exist of the most different objects, as of the heights of houses in a town or the sizes of books in a library. It may be convenient to convey a general notion of the things averaged, but involves no conception of a natural and recognizable central magnitude, all differences from which ought to be regarded as deviations from a standard. The notion of a mean, on the other hand, does imply such a conception, standing distinguished from an average by this very feature,—viz., the regular march of the groups, increasing to a maximum and then again diminishing. An average gives us no assurance that the future will be like the past. A mean may be reckoned on with the most implicit confidence. All the philosophical value of statistical results depends on a due appreciation of this distinction, and acceptance of its consequences."—*Edinburgh Review*, vol. xcii.; Baxter, op. cit., p. lxxviii.

curve,' or the 'curve of the frequency of error.' Now, it will be seen that this curve is not quite uniform, and that the lower half (from the mean to the dwarfs) is less regular than the upper; and if the numbers are counted it will be found that there are ten more boys below the mean than above it, consequently the *average* (obtained in the usual way, by dividing the sum of the values observed by the number of observations) is lower than the *mean*, which is represented by the largest group. The difference in this instance is not very great, the average being 53.43 and the mean 53.5 inches, but in some instances it is much greater; and it is exceedingly important that the difference here indicated should be borne in mind, for in it consists much of the practical value of Quetelet's method."

Indeed, so exact are the methods of M. Quetelet that a curious fact has been discovered in relation to dwarfs and giants, which, though in the general estimate considered as monstrosities,—anomalies of the human species,—are found to fall into their places as necessary factors in completing the scale of human stature. Moreover, it would be possible, if a correct estimation of the mean proportion of a population were made, to declare the number of each, and even the actual stature.

While M. Quetelet's method of studying the proportions of the body is the only scientific one yet propounded, and is the one now employed by all statisticians, the number of observations made by him are too few to be of any practical value, and the tables of Mr. Roberts, those of Prof. Bowditch, and the statistical results of the observations of Dr. John Beddoe, of 17,000 observations obtained through the medical profession of Great Britain, the 12,740 Bavarian soldiers examined by Dr. Meyer, the extensive observations upon recruits of M. Boudin, and the examination of over one million men enlisted in the war of the rebellion in our own country (1,232,256), particularly the 23,624 men examined by the Sanitary Commission, have been employed in arriving at a correct scientific estimate of the "mean" man.

The greater bulk of these observations is upon men, and, while they have no direct bearing upon development in children, they improve and correct the observations made upon children, and furnish more accurate data upon which to estimate physical culture, perverted development, and treatment of disease at this early period of existence. To emphasize this fact, the statement of M. Quetelet that children of the same sex are of the same size at twelve, and its correction by Drs. Roberts and Bowditch by the statement that at thirteen and fourteen years girls are taller and heavier than boys, may be cited.

METHODS.—In recording systematic measurements of the human body certain rules must be observed, in order to secure exactness, and for subsequent reference and comparison. Prominent bony points—as the acromial extremity of the clavicle or the trochanter—are to be depended upon chiefly, although some soft parts, as the nipples in males, and the umbilicus, are sufficiently definite and fixed to be available. All measurements, if possible, must be taken upon the naked body, and heights taken without the shoes. The height is best obtained by measuring from the ground upward, although the various dimensions of the head are best obtained by measuring downward from the vertex. The diameters and circumference of the trunk are readily secured by means of callipers and a plain tape-measure marked in English inches and tenths of an inch. As the various trunk-measurements vary much with the state of the respiration, these are best determined when the chest is empty and at rest, a condition easily secured by directing the person to count ten slowly in a loud voice immediately before the measurements are taken. The strength is best determined by making with the dynamometer several (three or four) trials each of grasping, pulling, and lifting, and estimating the averages. For recording all measurements the best methods and chart are those given by Mr. Roberts in his "Manual of Anthropometry," to which the reader is referred, or the blank forms published by the Anthropometrical Committee of the British Association, 22 Albemarle Street, London.

The method extensively used in Germany, which consists in recording on a separate paper all the memoranda and observations relating to each individual case, reduces the labor of tabulation to a minimum and facilitates the estimation of any particular observation. It should, therefore, always be employed.

The following table may be taken as a good form upon which to record measurements. These should be repeated every three to six months, and comparisons made, or they may be plotted upon a percentage chart, such as are sold by dealers in general sporting goods, and compared with a standard or with hundreds of others similarly examined.

TABLE FOR MEASUREMENT.[1]

Date . . . . 189 . .
Name . . . .
Age . . . . . . years . . . . months . . . . days
Weight . . . . pounds
Height . . . . feet . . . . inches

Girth of head . . . . feet . . . . inches
“ neck . . . . “
“ right shoulder . . . . “
“ left shoulder . . . . “
“ cir. shoulder . . . . “
“ chest inflated . . . . “
“ chest uninflated . . . . “
“ lung capacity . . . . cubic “
“ waist . . . . “
“ hips . . . . “
“ right thigh . . . . “
“ left thigh . . . . “
“ right calf . . . . “
“ left calf . . . . “

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[1] From Physical Culture, A. J. Reach Co., Philadelphia.

TABLE FOR MEASUREMENT.—(*Continued.*)

Girth of right arm up	inches.
" left arm up	"
" right arm down	"
" left arm down	"
" right forearm	"
" left forearm	"

The relative and average growth in weight and increase in height have already been fully discussed in the article upon the physiology of infancy. There remain the general proportions and development of the head, neck, trunk, and extremities. It may be interesting here to observe that Quetelet has ascertained that in a large number of men (ten thousand, for example) of the same nation, age, and external surroundings, the same uniformity prevails as to weight that has been demonstrated to exist as to stature.

As in height and weight, in addition to a large number of accidental causes, there are at least three factors which directly affect the development of the different portions of the body in the growing child. These are nationality or race (Boudin), the occupation of the parents (Roberts), and the mode of life (Bowditch).

M. Boudin, after comparing the results of his very extensive observations, to determine the mean height of the inhabitants of different districts of France, with the researches of Broca, concluded that soil, local surroundings, and climate exercise little influence on height, which is always an affair of race or hereditary descent.

Mr. Roberts has demonstrated that the sons of English non-laboring classes are decidedly taller, and at most ages also heavier, than the children of the laboring classes; the difference amounting at thirteen years to nearly four inches, and this in a population comparatively stationary and homogeneous in character. In our own country, where the population is heterogeneous, movable, and without class distinction, this principle is much less marked. Dr. Bowditch has, however, pointed out its existence, but concludes that the influence of mode of life in determining the size of growing children is at least equal to and possibly even greater than that of race.

PROPORTIONS OF THE BODY.

The different portions of the human body develop with varying rapidity at different periods of life, an observation already made in referring to Prof. Carus's "canon" of proportions. Of all these the head is most completely developed at birth and varies least during subsequent growth, for which reason, doubtless, it was early adopted as a standard of measurement. In the adult its relation to the entire height is one-seventh. From birth to adult life it doubles its height, while the remainder of the body increases three or four times, the growth being almost terminated at the age of adolescence. At birth the antero-posterior diameter (occipito-frontal, 11¾ centimetres) is a little greater than the transverse (biparietal, 9¼ centimetres), and this ratio is maintained throughout life. The height varies little in the two sexes at different periods of existence, as shown in the following table, taken from Quetelet:

Age.	Men.			Women.		
	Total Height, Inches.	Height of Head, Inches.	Ratio.	Total Height, Inches.	Height of Head, Inches.	Ratio.
Birth	19.68	4.37	4.50	19.44	4.37	4.45
1 year	27.48	6.06	4.53	27.16	6.06	4.48
2 years	31.14	6.81	4.57	36.74	6.77	4.54
3 "	34.03	7.16	4.74	33.64	7.08	4.74
5 "	38 86	7.56	5.14	38.34	7.40	5.18
10 "	50.11	8.07	6.21	49.37	7.91	6.21
15 "	59.56	8.46	7.04	58.58	8.38	6.99
20 "	65.74	8.93	7.35	61.96	8.66	7.15
30 "	66.37	8.97	7.39	62.20	8.80	7.15

The lower part of the face appears relatively to grow more rapidly than the upper; the nasal incision, which at maturity divides the face into two equal parts, at birth is nearer the chin,—a circumstance which may be accounted for by the development of the alveolar processes and of the teeth, especially the molars.

Neck.—At birth the neck is about one inch in length, but later, owing to the fatness of the infant's chin, it apparently shortens, and does not appear to grow perceptibly till the sixth or seventh year, doubling its length by the time adolescence is reached. Its diameter develops rapidly during the first year, and again at puberty, and is practically the same in both sexes, being more rapidly attained, however, in the female, the diameter taken just above the clavicle measuring at birth about 1.81 inches, at six years 2.25 inches, and when fully developed about 4.75 inches.

The development of the larynx in the male increases somewhat the upper circumference, and the development of the thyroid body in the female the lower circumference.

Trunk.—The trunk includes the chest, abdomen, and pelvis, and is bounded above by the clavicles and below by a horizontal line on a level with the perineum. In length and transverse diameter the trunk-measurements from birth to maturity are tripled, while the head- and neck-measurements are doubled. The antero-posterior diameter, however, grows with less rapidity, increasing very slowly and regularly from birth, and doubling only at puberty. The circumference grows with the same rapidity as the transverse diameter: it is much modified after puberty in the two sexes, the shoulders being proportionately wider in the male, the pelvis in the female, the disproportion, which is, however, slight, being much enhanced in woman by the development of the breasts and by the modifying influence of her costume. This relative growth of the trunk in height and diameters is well shown in the diagrams Nos. 2 and 3 opposite pages 97 and 113 respectively in Roberts's "Manual of Anthropometry."

The circumference of the chest, on account of the important organs which it contains, has been selected, together with the height and weight, to determine the physical capacity of the individual for military, naval, and other public duties. The importance of these data in life insurance can hardly be over-estimated. From extended observation it has been ascertained that a direct ratio exists between the height, weight, circumference of chest, and age, and formulas have been arranged somewhat arbitrarily by which to ascertain from the stature the circumference of the chest. It is not well, however, to follow these formulas too dogmatically, as Baxter's conclusions prove that Mr. Hutchinson's and Mr. Brent's formulas do not correspond when applied to the examination of immense numbers of able-bodied men, representing the picked men of the nation. From these it is observed that the figures do not rise above the minimum size of the chest, *nor do they in any instance attain the medium size.*[1]

If the mobility of the chest be very limited, this should also be regarded as a disqualification for military service, or else should lead to more thorough physical examination. The degree of mobility varies much in different individuals, and is also modified by sex, the female chest, particularly the upper portion, being more active, but limited in extent. The healthy man's expansion, according to Hutchinson, is three inches (Baxter, 3.08), but may reach even seven inches. A remarkable instance was observed in a native of New Jersey admitted to the service, who was eighteen years of age, weighed one hundred and fourteen pounds, and was sixty-four inches in height; his chest at expiration measured twenty-nine inches in girth, and had an expansion of *seven inches.*

Upper Extremities.—The upper extremity, including the arm, forearm, and hand, varies much in development. The popular idea that the space covered by the extended arms is equal to the height is correct only for early life,—before puberty,—being the same in both sexes. After puberty, however, in man, owing to the increased breadth of the shoulders, the horizontal measurement exceeds the perpendicular, and in woman it is also slightly greater. The arm, measured between the acromion and the external condyle, increases in length from 3.5 inches in the infant to 13 inches in the adult, and in circumference from 4 to 12 inches, these measurements, however, being greatly influenced by exercise, sickness, etc.

The adult forearm (cubit) was the unit of measurement among the ancients. The growth of the forearm in length, measured from the external condyle to the styloid process of the radius, is from 2.25 inches in the infant to 9½ inches in the adult, and its increase in circumference from 2.5 inches in the child to 13⅜ inches in the adult. Its circumference is also much influenced by exercise.

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[1] For more detailed information the reader is referred to Baxter, vol. i. p. 43.

The hand, as employed by artists, sculptors, and others, represents the ratio of 1 to 9 of the entire height. It develops slowly, being doubled in length by the seventh year, and tripled at maturity. It is a little broader and plumper in proportion to length in women than in men.

The length of the entire upper extremity, measured from the acromion extremity of the clavicle to the tip of the middle finger, is doubled between the ages of four and five, tripled by puberty, and quadrupled by maturity, —growing with less rapidity than the lower extremity, which during the same period is quintupled.

Lower Extremities.—The lower extremity includes the thigh, the leg, and the foot, and is the most important factor in determining the height of the individual. The thigh, included between the trochanter and the patella, increases in length from 2.75 inches in the infant to 18 inches in the adult, an increase of nearly sevenfold, the greatest increase of growth observed in the body. After puberty the increase is relatively greater in the female, owing to the increased width of the pelvis and the development of the upper thigh. For the same reason, the angle formed laterally at the knee, between the thigh and the leg, is also relatively greater in the female.

The leg, included between the lower edge of the patella and the internal malleolus, increases from 3.4 inches in infancy to 15.3 inches at maturity, an increase which is much modified by exercise and somewhat also by sex.

The foot has been much employed as a unit of measurement, its length being in both sexes and at all ages from .15 to .16 of the entire height. The belief that its length corresponds to the height of the head and the circumference of the fist is fallacious. It grows in height from 1 inch at birth to 3.25 inches at maturity, and in length more rapidly than in height, from 3–3.5 inches at birth to 8–10 inches at maturity. Its width is proportionately greater in the male, and is much influenced by race-peculiarities and by dress.

The length of the lower extremity increases more rapidly than that of any other portion of the body, being doubled before the third year, quadrupled before puberty, and increased fivefold by maturity, the thigh growing more rapidly than the leg, and the leg than the foot.

In this connection it must be borne in mind that the human frame is subjected to many influences that tend to produce minor deformities, congenital and acquired, all, however, within the bounds of health, as unequal development of the lower extremities, transposition of viscera, excessive development of the right side, etc.

The relative proportions of a perfect female form, as deduced by modern sculptors from the Greek statues, may be stated as follows. With a height of five feet five inches, one hundred and thirty-eight pounds is the proper weight, which, however, could be increased ten pounds without greatly destroying the proportion. When her arms are extended, she should measure from tip of middle finger to tip of middle finger just five feet five, exactly her own height. The length of her hand should be just a tenth of that, and her foot just a seventh, and the diameter of her chest a fifth. From her perineum to the ground she should measure just what she measures from the perineum to the top of the head. The knee should come exactly midway between the perineum and the heel. The distance from the elbow to the middle finger should be the same as the distance from the elbow to the middle of the chest. From the top of the head to the chin should be just the length of the foot, and there should be the same distance between the chin and the armpits. A woman of this height should measure twenty-four inches about the waist, and thirty-four inches about the bust if measured from under the arms, and forty-three if measured over them. The upper arm should measure thirteen inches, and the wrist six. The calf of the leg should measure fourteen and a half inches, the thigh twenty-five, and the ankle eight.

When full development has been attained, the relative proportions of a perfect male may be summarized as follows:

TABLE[1]

Showing the Proper Weight, Height, and Measurement of a Fully-Developed Man.

HEIGHT.	WEIGHT.	NECK.	WAIST.	CHEST.	BICEPS.	FOREARM.	THIGH.	CALVES.
5 feet,	103–107	11⅛	29	32–33	Same measurement as for the neck.[2]	8⅞	15	Same measurement as for the neck.[2]
5 " 1 inch,	107–111	11½	29½	33–34		9¼	16	
5 " 2 inches,	111–116	12	30	34–35		9⅝	17	
5 " 3 "	116–121	12½	30½	35–36		10	18	
5 " 4 "	121–127	13	31	36–37		10⅜	19	
5 " 5 "	127–133	13½	31½	37–38		10¾	20	
5 " 6 "	133–140	14	32	38–39		11⅛	21	
5 " 7 "	140–147	14½	32½	39–40		11½	22	
5 " 8 "	147–155	15	33	40–41		11⅞	23	
5 " 9 "	155–164	15½	33½	41–42		12¼	24	
5 " 10 "	164–174	16	34	42–43		12⅝	25	
5 " 11 "	174–185	16½	34½	43–44		13	26	
6 "	185–196	17	35	44–45		13⅜	27	

EXERCISE.

The term "exercise" is applied physiologically to any exertion or action of the body for the maintenance of its organs or functions in a healthy condition, but in a more restricted and generally accepted sense applies to certain movements of the body effected by the contraction of the voluntary muscles made with sufficient force and rapidity to quicken the breathing and accelerate the circulation of the blood,—in other words, muscular exercise. The muscles acted upon during exercise are all of the striped variety,—the skeletal muscles, heart, diaphragm, etc.; but a marked distinction must be observed between these muscles, from the fact that, although practically of the same structure, the nervous control differs, the heart and diaphragm not being under the control of the will, and their action being only secured secondarily through the effects of the action of the voluntary muscles.

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[1] From Physical Culture, A. J. Reach Co., Philadelphia, p. 52.

[2] This rule has long been observed, especially among artists, but it is not true to life, for very rarely do we find either men, women, or children whose neck, upper-arm, and calf measurements are the same. As a rule, the upper arm is the smallest of the three.

The muscles consist essentially of the sarcous substance, with its nuclei or muscle-corpuscles enclosed within compartments, and surrounded by the sarcolemma and endomysium and perimysium, so as to consist of fasciculi or bundles of fibres surrounded by connective tissue. These fasciculi are connected ultimately by tendinous fascias or bands with the osseous structures upon which they are intended to operate. The sarcolemma through a single opening receives the axis-cylinder of a nerve-fibre, which after forming a flat protuberance—or motor end-plate—is distributed in fine fibrils throughout the cell-contents. Sensory as well as motor nerve-fibres are distributed to the muscle-substance, to convey impressions to the centrally situated nerve-cells. By a fine mesh-work of capillaries lying between and upon the fibres and cells, but without penetrating the sarcolemma, the muscles are bountifully supplied with blood.

Muscles are at rest neutral or slightly alkaline in reaction, and consist chemically by weight of three-fourths water and one-fourth nitrogenous and non-nitrogenous matters and salts. The most important nitrogenous element is the coagulated substance which becomes in dead muscle myosin, which is found in much smaller quantities in infants than in adults. Among the non-nitrogenous matters, paralactic or sarcolactic acid may be mentioned, while the salts are principally the alkaline compounds found most abundantly in the blood, these salts and other extractive matters being much more plentiful in infant than in adult life.

FUNCTION OF VOLUNTARY MUSCLES.—The characteristic physiological property of muscle is its contractility, by virtue of which all its acts are performed. The muscular system in infancy is very poorly developed, so that during childhood and youth the increase, both relative and absolute, is enormous. In its elongated condition of rest, muscle is still under a slight degree of tension (muscular tonus, or muscular tonicity), and under the influence of appropriate electrical or nervous stimulus it becomes shorter and thicker and its extremities are approximated. With increased stimulation the corresponding contraction will be found to increase, and finally will diminish until contraction ceases entirely through the muscle being fatigued from repeated stimulation. The more rapid the contraction the more quickly does the fatigue manifest itself and the longer is the period of rest required to recuperate its full power. As might also be expected, a muscle will become fatigued much sooner when it does work than when it simply contracts without doing work. The muscles in contracting must exert a force sufficient to elongate the opposing muscles, must overcome the force exerted by the tonicity of the antagonizing muscles, and must lift the weight of the portion of the limb to which they are inserted. When, on the other hand, this force, known as antagonistic force, is withdrawn by paralysis or section of the tendons, undue action is at once manifested in the opponents. The vibrations of muscular contraction can be distinctly heard if the stethoscope or myophone be applied over a powerfully-contracting muscle, as the biceps, or the heart during its first sound.

During contraction the reaction of the muscles becomes distinctly acid, from a considerable amount of carbonic or lactic or sarcolactic acid being set free. With use the muscles increase in size, in firmness or tone, and in strength, and respond more quickly to stimuli, good examples of which are exhibited in the bulky biceps of the blacksmith and the colossal calf of the ballet-dancer. When a single muscle or a group of muscles is exercised too much, it will, after increasing greatly in size, begin to waste. This, however, is not the case *when all the muscles of the body are exercised*, probably, as Parke suggests, because no one muscle can then be over-exercised.

MUSCULAR WORK.—The work performed by muscular contraction represents but a fractional part—about one-ninth, or over ten per cent.—of the entire work done in the body; the nervous energy, or internal work, as it is called,—the force required to regulate the various processes of digestion, assimilation, and secretion, for intellectual pursuits, etc., and the caloric work, or the force required in the production of heat to maintain the bodily temperature,—constituting the larger proportion. It must be remembered that the heat-units employed in the internal work of regulating the circulation and perspiration are only temporarily converted into mechanical energy, the latter being almost entirely reconverted into heat by the function developed by various obstructions offered to the movement of the blood and respiratory organs. However expended, or however it may vary in amount, the force is the same, and its amount may be estimated and its source be discovered. "The work done by a muscle in a given contraction, when it lifts a weight vertically against gravity, is measured by the weight moved multiplied by the distance through which it is moved."[1]

It has been found, from calculations made by different observers, that the amount of force expended daily by an adult weighing one hundred and fifty pounds in the performance of these three forms of work is about three thousand four hundred foot-tons,—or a force required to raise three thousand four hundred tons one foot in height,—the greater amount of which (two thousand eight hundred and forty) is employed in maintaining the animal temperature, the remainder being distributed between the internal force (two hundred and sixty) and muscular activity (three hundred).

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[1] Sargent, Physical Training Conference, 1889, p. 71.

Estimates made upon the actual labor expended in performing certain manual acts by men of the same weight, such as eight hours' pile-driving, turning a wrench, carrying a peddler's pack, paving, etc., demonstrate the same fact. From estimates of this character, and considering that the type of the most healthy life is that of a man engaged at moderate labor in the open air, at which work he will probably average between two hundred and fifty and three hundred and fifty foot-tons, Prof. Parkes has concluded that a healthy adult should bear without the risk of over-fatigue what would be equivalent to a walk of nine miles, from which must be deducted the exertion used in ordinary business pursuits.

The source of this force or energy must be sought in the molecular life or combustion going on through the entire economy. In every action of the human body,—whenever we make a movement, draw a breath, change a muscle of expression, or conceive a passing fancy,—molecular death has occurred, and a quantity of potential energy (force) has been liberated. This force, having previously entered the body in a latent state as food, was stored by means of the blood in the cellular structures awaiting the demand for its ignition with the oxygen circulating in the blood. These disintegrated cellular elements, removed by the blood to be eliminated from the system, are replaced by new active cells, and thus the unbroken circle of physiological phenomena—latent potential energy, motion or activity, cell-destruction, cell disintegration and elimination, cell-renewal, more latent force, and so the same succession of events—is maintained, and at every revolution vital, active (kinetic) force develops. This, in a word, constitutes "life;" and upon the celerity and thoroughness with which these processes are repeated—upon the "newness," as Maclaren has expressed it, of the individual cells—will depend the health and strength of the individual.

Motion and Heat.—This force, then, which is the result of the oxygenation, death, or burning up of the cellular elements of the body, suggests at once the relationship which exists between heat and motion, the correlation existing between them, and the conversion of one into the other. Conceived by Sadi-Carnot, clearly formulated by R. Mayer, brilliantly demonstrated by Joule, the theory of the equivalence of forces may now be said to be admitted by all physicists, and the mechanical equivalent of heat be considered as 425.

As before stated, nearly sixty per cent. of the work performed in the body is employed in the production of heat or in muscular exercise, and we shall now see that probably three-fourths of the heat developed is produced in the muscles at the moment of muscular contraction. Daily observation and special experiments teach us that the whole body is heated by muscular exercise, and Davy found that after a walk in the open air, the temperature of the surrounding atmosphere being 40° F., the temperature of the urine was elevated one degree,—from 100° to 101° F. This development of heat by muscular contraction is also well seen under certain pathological conditions,—for example, in tetanus rising, according to Wunderlich, to the extreme height of 112.5° F., although this extreme temperature can hardly be attributed entirely to muscular action.

The theory of the close relationship between calorification and combustion, poetically expressed by the philosophers of antiquity as the fire snatched from heaven, the flame of Prometheus, and described so accurately by Lavoisier more than a century ago, remains substantially the accepted view of modern times, the only modifications consisting in the implication of the theory of caloric, and in considering the lungs not alone the seat of the chemical interchange.

Natural philosophy furnishes numerous examples of motion converted into heat by friction, etc., and also of the conversion of heat into motion, of which the steam-engine is a practical example. In the human economy, the various systems, but particularly the muscular, may be regarded as so many machines for converting the potential energy derived from the food and oxygen into mechanical force, with this exception, however, that the muscles are more economical in their action than the finest engine could possibly be. Food and oxygen are taken into the system, potential energy and bodily temperature are produced, and it remains to estimate the heat-value and force-value of the different principles of food.

To ascertain the heat-units, and to deduce the force-value or units of motion from these, Favre and Silberman first, and later Prof. Frankland by means of the calorimeter, determined by experiment the amount of heat developed by the combustion of different articles of food within and outside the body. It was found that the fatty and carbohydrate foods were as thoroughly burned in the body as without, though more slowly, while the nitrogenous (albuminous) substances were but imperfectly consumed, corresponding exactly with what occurs within the system. Upon the same principle Ranke performed his admirable experiments upon himself. From these and the experiments of Vierordt and Voit a sufficiently accurate estimate of the force-value of the different elements of food may be formed.

According to the reduced table of Prof. Frankland as given by Ralfe,—

15 grains of dry albumen (fleshy matter) = 13,851 foot-pounds.
15 grains of fat of beef = 27,716 "
15 grains of starch = 11,720 "

"If, therefore," says Ralfe, "1800 grains of dry albumen, 1350 grains of fat, 4900 grains of starch, be daily consumed, an amount of force capable of lifting 7,910,045 pounds to the height of one foot, or 3530 foot-tons, will be derived from the food; a result closely corresponding with the calculated estimate of the total work done in the body,—viz., 3400 foot-tons." The former popular fallacy that the principal source of muscular force was from the combustion of the albuminous, fleshy principle has happily been dispelled, and the relative value of albumen, fats, and starches is appreciated.

It will be recollected that the strongest men are not always meat-eaters, and that the rhinoceros and the elephant, animals noted for their strength, are strictly vegetarians. Indeed, the opinion has been expressed by a good authority that different muscular qualities are developed by flesh and farinaceous food, the leopard and the deer being given among animals as illustrations: the leopard with the quick action engendered by fleshy food has the advantage of the deer, but the latter with the slow force developed by its farinaceous diet can outstrip the leopard in fair chase.

While the limits of this article will not permit of any discussion of general muscular movements, involving necessarily a detailed description of the muscles and general anatomical structures of the joints and the thorough consideration of animal mechanics, a short account of how the ordinary movements are accomplished would seem an appropriate introduction to a description of the varieties of exercises employed in physical culture. The skeletal muscles may be regarded for the most part as so many sources of power arranged to act upon the bones and cartilages as levers. The three forms of levers recognized in ordinary mechanics, from the relative position of the power, the weight to be moved, and the axis of motion or fulcrum, are all represented in the human economy, examples of the first kind being rare, and those of the third kind being more common than the second.

This preponderance of the third class of levers is probably due to the fact that the movements of the body are chiefly directed to moving comparatively light weights through a great distance, or through a short distance with great precision, rather than to moving heavy weights through a short space. A familiar example of the first order, where the power is at one end, the weight at the other, and the fulcrum in the middle, occurs when the body is raised from the stooping position by the action of the hamstring muscles attached to the tubera ischii. The second order, where the power is at one end, the fulcrum at the other, and the weight in the middle, is illustrated in the depression of the lower jaw in opening the mouth, the temporals and masseters representing the weight. The third order, where the fulcrum is at one end, the weight at the other, and the power in the middle, is best represented by the action of the biceps muscle upon the forearm.

All these orders of levers may be beautifully illustrated in the different movements of the foot,—the first where the heel is raised and the toe is tapped upon the floor, the heel representing the power, the ankle-joint the fulcrum, and the toe the weight; the second where the body is raised upon the toes, the ground representing the fulcrum, and the body the weight; and the third where the foot is flexed and elevates a weight resting upon the toes, the ankle being again the fulcrum.

There are few movements of the body in which one muscle only is concerned. In the majority of cases several muscles act together, nearly all the movements are co-ordinate, and generally the power is so disposed with reference to the fulcrum that in acquiring a greater range of motion the power is diminished.

Animal Mechanics.—Every movement of the body depends as much upon the proper co-ordination of the muscles for its accuracy, grace, and force as upon the strength of their contraction; and particularly is this true of the exceedingly complex movements of walking, running, jumping, etc. To appreciate these best, some knowledge must be had of the manner in which the upright position is maintained. In the erect posture the weight of the entire body is borne by the plantar arches, by a series of muscular contractions of the lower extremities, trunk, and neck, having for their object the maintenance of the body in such a position that the line of gravitation falls within the area of the feet. That this is the result of muscular action, at one time denied, is proved by the facts that a person standing perfectly at rest in a balanced position falls when unconscious, and that a dead body cannot be balanced upon its feet. The line of gravity passes from the vertex of the head in front of the occipital articulation, in front of the tenth dorsal vertebra, behind a line joining the centres of the two hip-joints, a little behind the knee-joints, to reach the earth in front of the centre of a line drawn between the two ankle-joints. The centre of gravity for the entire body is located at the end of the sacrum, and for the combined head and trunk about the level of the ensiform cartilage in front of the tenth dorsal vertebra. The somewhat unstable equilibrium of the erect position is maintained without much difficulty by keeping this line within the area of the basis of support; otherwise, when the line falls outside this area, the tendency of the body is to fall, unless overcome by strong muscular action.

In maintaining the body in this erect position almost all the muscles of the lower extremity, trunk, and neck are concerned. While the line of gravity remains within the area of the feet, the slight muscular effort required is little more than the tonicity contained in all living muscle. The head and neck are maintained from falling forward or backward by the combined action of the trapezius, levator anguli scapulæ, splenius, cervicalis ascendens, transversalis colli, trachelo-mastoid, spinalis colli, complexus, multifidus spinæ, interspinales, rectus capitis posticus minor, and obliquus superior, acting as backward flexors against the platysma myoides, sterno-cleido-mastoid, rectus capitis anticus major, rectus capitis anticus minor, longus colli, scalenus anticus medius and posticus, digastric, sterno-hyoid, sterno-thyroid, omo-hyoid, mylo-hyoid, and genio-hyoid as forward flexors; the position laterally being maintained by the platysma myoides, sterno-cleido-mastoid, trapezius, splenius, trachelo-mastoid, complexus, the three scaleni, rectus capitis posticus major, intertransversalis, and rectus lateralis.

The line of gravity falling in front of the occipital condyles, the tendency of the head is to fall forward, demanding naturally greater action of all the posterior group of muscles and ligaments (particularly the ligamentum nuchæ), which of necessity are better developed.

The trunk is maintained from falling backward by the action of the rectus abdominis, pyramidalis, obliquus externus, obliquus internus, and psoas magnus and parvus, assisted by the pectoralis major and minor, serratus magnus, transversalis, pectineus, adductor longus, adductor brevis, rectus femoris, sartorius, and all those muscles which pass from the pelvis to the internal condyle and shaft of the femur; and from falling forward by the action of the glutei (magnus, medius, and minor), latissimus dorsi, serratus posticus inferior, and all the larger dorsal muscles, assisted by the scapular muscles,—rhomboidei, inferior portion of the trapezius, etc., which fix the scapulæ backward and approximate them to the skeleton. It is maintained laterally by the obliquus externus, obliquus internus and transversalis, quadratus lumborum, longissimus dorsi, and sacro-lumbalis.

The rigidity of the body upon the thighs is secured by the contraction of the psoas magnus, iliacus, sartorius, pectineus, three adductors, gracilis, gluteus minimus, obturator externus, tensor vaginæ femoris, and rectus femoris, as flexors acting against the gluteus maximus and medius, pyriformis, obturator internus, gemelli superior and inferior, quadratus femoris, long head of biceps, posterior portion of adductor magnus, semi-tendinosus, and semi-membranosus acting as extensors; the position being secured laterally by the three glutei, tensor vaginæ femoris, pyriformis, gemelli, sartorius, and obturator internus, acting as abductors against the three adductors, psoas magnus, iliacus, pectineus, quadratus femoris, obturator externus, gracilis, semi-tendinosus, semi-membranosus, and long head of biceps acting as adductors.

The rigidity of the legs upon the thighs is maintained by the biceps, semi-tendinosus, semi-membranosus, gracilis, sartorius, gastrocnemius, plantaris, and popliteus acting as flexors, and the quadriceps femoris (rectus femoris, vastus externus, and crureus) acting as extensors.

The relation of the foot at an angle of ninety degrees to the leg is maintained by the gastrocnemius, soleus, plantaris, and peroneus longus, as extensors; the tibialis anticus, peroneus tertius, and extensor longus digitorum, as flexors; the tibialis anticus, tibialis posticus, and flexor longus digitorum, as adductors; and the three peronei (peroneus longus, brevis, and tertius), as abductors; the arch of the foot being supported principally by the peroneus longus, the interosseous ligaments (chiefly the inferior calcaneo-scaphoid and calcaneo-cuboid), and the plantar fascia.

"It may be instructive here to review briefly the anatomical construction and mechanism of the natural foot. The foot includes all that portion of the inferior extremity below the tibio-tarsal articulation, consisting of the tarsus, metatarsus, and phalanges, and in the adult has the form of two arches, an antero-posterior and a transverse, each with its convexity or dorsal surface above and its concavity or plantar surface below. The antero-posterior, the most important, is supported upon two piers or pillars, and has its summit at the astragalus and ankle-joint. This has been still further divided into two arches, an outer and an inner, by an imaginary line drawn posteriorly between the third and fourth metatarsal bones; the inner portion of the antero-posterior arch is much more curved than the outer, and forms the instep. The posterior pier, formed by the posterior parts of the astragalus and os calcis, is shorter, more curved, has but one joint, and is more solid, receiving the greater part of the weight of the body. The anterior pier, composed of the scaphoid, three cuneiform, and three inner metatarsal bones, is longer, less curved, has many joints, and is more elastic, serving to diminish the force of shocks transmitted to the arch. The head of the astragalus fitting into the concave surface of the scaphoid, and its postero-inferior surface articulating with the anterior surface of the os calcis, it may be regarded as the keystone, though differing in many respects from such bodies as usually employed. The weak part of the arch is strengthened by the interosseous ligaments, particularly the inferior calcaneo-scaphoid, which supports it from below, while those beneath the inner portion of the plantar fascia add additional strength. The outer portion of the antero-posterior arch consists of the outer portion of the os calcis, the cuboid, and the two outer metatarsal bones. It is strengthened by the calcaneo-cuboid ligaments and the outer portion of the plantar fascia. Both arches are still further maintained by the tibialis posticus and peronei muscles, particularly the peroneus longus. The transverse arch, formed in the inner and outer sides by the bones entering into the inner and outer antero-posterior arches respectively, varies in degree of curvature in different portions of the foot, being most marked across the cuneiform bones. It affords protection to the soft parts of the sole, and adds to the elasticity. The weight of the body is received by the astragalus as the highest part of the arch, and transmitted to the ground through the two piers of the antero-posterior arches. The foot in extension rests normally upon the heel, the tips of the metatarsal bones, and the outer side of the sole, the weight of the body in standing, walking, running, or dancing being transmitted through the heel, the ball of the great toe, and that of the little toe,—the natural tripod of the foot,—in the order named. Thus the direction of the weight upon the arches is constantly changing, and it is only through the actions of certain muscles that the normal arches are conserved. Thus, in flexion the antero-posterior arch is increased by the action of the tibialis anticus, peroneus tertius, and extensor longus digitorum; and in extension, by the action of the gastrocnemius, soleus, plantaris, and peroneus longus, both the curves are diminished and the foot is flattened. Then, also, when the foot is markedly flexed, the foot is adducted, in which position the arches are each respectively increased and diminished. In this connection Mr. Le Gros Clark says, 'In reviewing the actions of the various muscles around the foot, it is obvious that their attachment is designed to preserve the plantar arch, and that such healthy condition must depend in a great measure on the evenly-balanced action of those muscles upon their several attachments. Thus, the peronei and tibial muscles antagonize each other, and the expanded insertion of two of them into the tarsal bones is very instrumental in preserving the transverse as well as the antero-posterior arch.' "[1]

LOCOMOTION.—It is necessary here to refer briefly to the methods employed in the study of animal locomotion, in order to obtain a proper conception of this important subject. While the work of the Weber brothers and the earlier studies by means of the graphic method of MM. Marey, Carlet, and Mathias Duval were excellent so far as they extended, they gave but an imperfect idea of the complicated mechanism of animal locomotion. To remedy the defects of the graphic method, Marey first and Demeny later invoked the aid of photography, but even the results thus obtained give but meagre information of the lateral sway. The earlier authors employed the zoetrope—an ingenious optical instrument invented by Plateau, which presents to the eye a series of successive images so coordinated by rapid revolution as to bring before the eye all the phases of a movement—to overcome these defects; and Marey, well aware that his photographs gave no information of the direction or extent of the lateral sway, endeavored to remedy this by an ingenious application of the stereoscope to his photographic wheel. This addition to the pictures gave the impression of an undulating white band extending through space, the undulations being in three directions, forward, vertically, and laterally, but did not admit of detailed study of the curves.

It remained for Mr. Eadweard Muybridge, of Philadelphia, so to utilize photography and electricity as to produce results almost, if not entirely, perfect. These possess the decided advantage over all similar efforts of admitting of a detailed study not only of the curves, but also of the forward, vertical, and lateral movements of the various points of the body.

Notwithstanding the various slight sources of error in Mr. Muybridge's methods, pointed out by Dercum, these photographs have contributed more than anything else in modern times to facilitate the study of animal motion and locomotion. These photographs, when placed in the ingenious instrument, invented by Mr. Auschuetz, known as the tachyscope,—which consists of a series of pictures so placed on a circular glass plate revolving rapidly on its axis that whenever a picture appears before the eye of the observer it is lit up by an electric spark,—produce a bit of life with a degree of truth and accuracy that are absolutely bewildering. Take, for example, the hurdle jump of a race-horse, which occupies seventy-two one-hundredths of a second, or the slower movement of a man on a galloping horse: the illusion is perfect. One sees not only the legs move according to the gait, but the dust rise, the horse's mane and tail fly, the nostrils dilate, the rider urge his horse, pull the curb, and move back his leg to apply the spur.

Mr. Muybridge's method consists in making simultaneous serial photographs of a moving man or animal at from two to four points of view at right angles with one another. While the serial method gives slightly less accurate results regarding the rise and fall and onward movement of a limb, taken all in all it more than compensates for the slightly-varying angle at which the photographs are taken, by permitting the determination of the direction and amount of the lateral sway, and gives an opportunity for the study of a part which a wheel photograph could not furnish. The drawings from Dercum exhibit the squares and display the scientific character of the work, but in the other original drawings these have been omitted, though they are none the less correctly drawn.

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[1] Young, New York Medical Record, iii. 10.

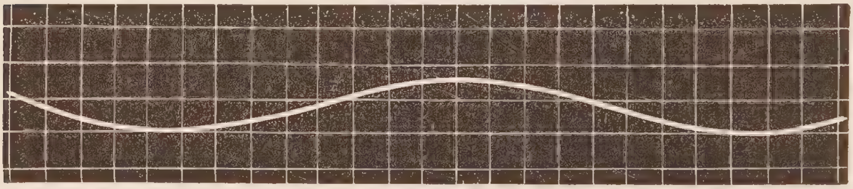
WALKING.—Walking has been described as being "a constant falling forward, where the weight of the body is received by each leg alternately." In walking there is in each step a moment when the weight of the trunk is supported upon the foot of one extremity (say the left) by the combined action of the flexors and extensors of the extremity and the abductors of the opposite side (see p. 261), while the other (the right) is inclined obliquely behind, the heel raised by the action of the gastrocnemius, soleus, flexor longus pollicis, flexor longus digitorum, tibialis posticus, and peroneus longus, and the toe resting upon the ground. The body balanced upon the left foot, by the combined action of the flexors and extensors of the hip, thigh, leg, and foot, and the abductors of the right side of the body, the weight of the body is thrown principally upon the glutei, inferior dorsal muscles, and abductors of the right side of the trunk. The right thigh is flexed by the psoas magnus, iliacus, biceps, gluteus medius, anterior part of the gluteus magnus, gracilis, sartorius, gastrocnemius, plantaris, and popliteus; to avoid contact with the ground the leg is swung forward pendulum-like by the relaxation of the flexors, and the contraction of the extensors of the thigh and of the knee, principally the quadriceps extensor femoris. This contraction of the extensors of the knee makes the pendulum-like motion of the right leg, and contributes to the forward motion in walking, the length of the swing varying with the length of the limb, and the right heel, the side of the foot, and the ball of the great toe are brought in contact with the ground.

On this right toe as a fulcrum the body is moved forward by the action of the extensors about the hip, particularly the gluteus maximus, and the lateral muscles of the trunk (see p. 261), describing both a vertical and a lateral curve, the right leg becoming straight and rigid by the combined action of all the muscles of the lower extremity.

With another forward movement of the pelvis the starting-point is again reached, the body supported vertically upon the right foot as before, and the left foot directed in an oblique position behind. This forward movement of the right foot carries the pelvis beyond the vertical, and in so doing swings the left leg forward by the action of the psoas magnus and iliacus, assisted by the sartorius, pectineus, gracilis, gluteus minimus, obturator externus, tensor vaginæ femoris, and rectus femoris, until its heel, side of sole, and ball of great toe in turn form the fulcrum on which the pelvis moves, and the right leg is again swung forward. In this manner the head and pelvis describe a series of concentric curves with their convexities upward. (See Photo. I.)

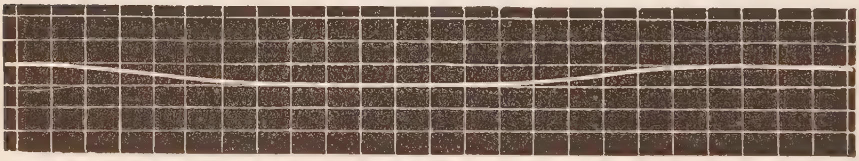
This regular and alternate movement of the support of the body from one foot to the other in walking necessitates a lateral displacement of the line of gravity, so that the centre of gravitation is constantly describing a consecutive series of horizontal (lateral) curves in addition to the vertical ones already described. This is particularly noticeable in the gait of a person walking slowly away from the observer.

Fig. 2 *a*.



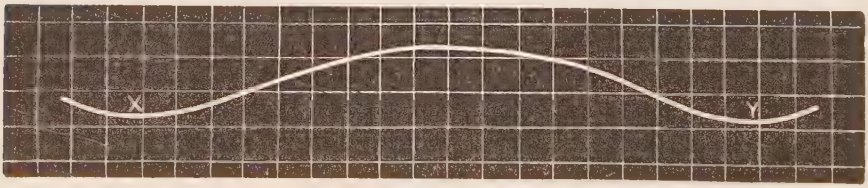
a, rise and fall and onward movement of head.

Fig. 2 *b*.



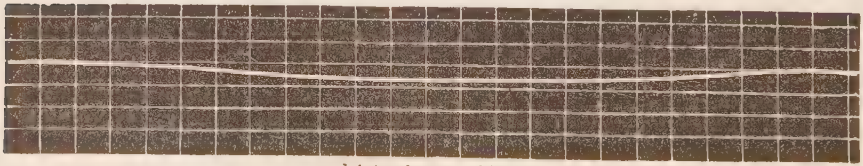
b, lateral sway of head.

Fig. 2 *c*.



c, rise and fall and forward movement of right hip.

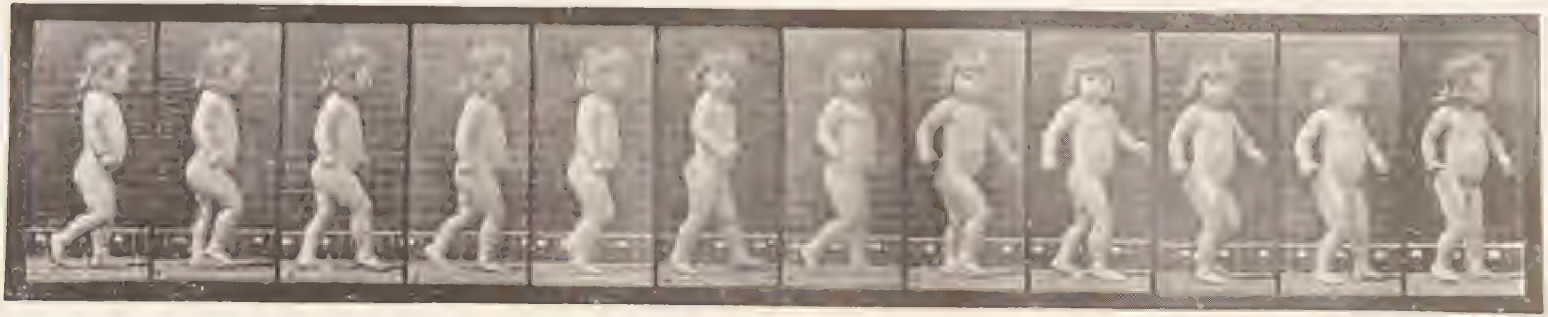
Fig. 2 *d*.



d, lateral sway of hip.

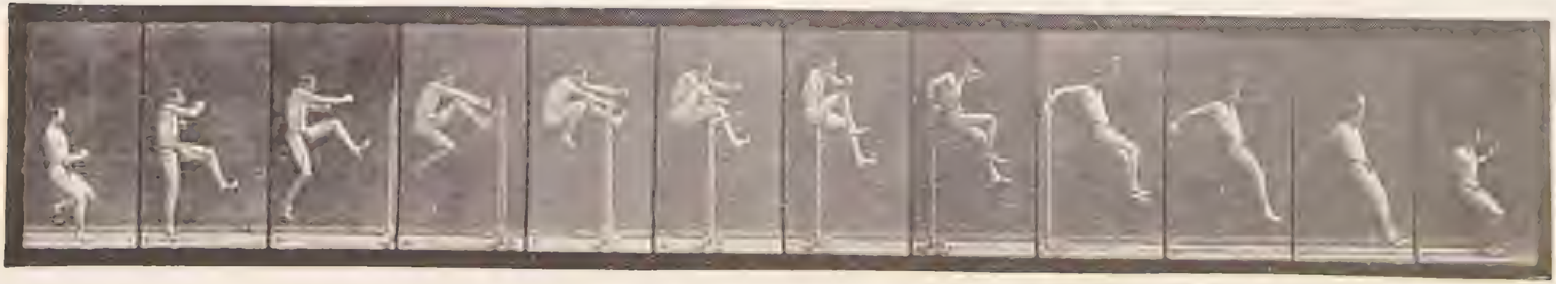
These curves are best understood by observing (see Fig. 2) the line *a*, which represents the rise and fall and onward movement of the white button in the cap worn by the subject, and the line *b*, which represents the direction and amount of the lateral sway of the same, while the line *c* represents the

PHOTO. I.



Walking Child.

PHOTO. V.

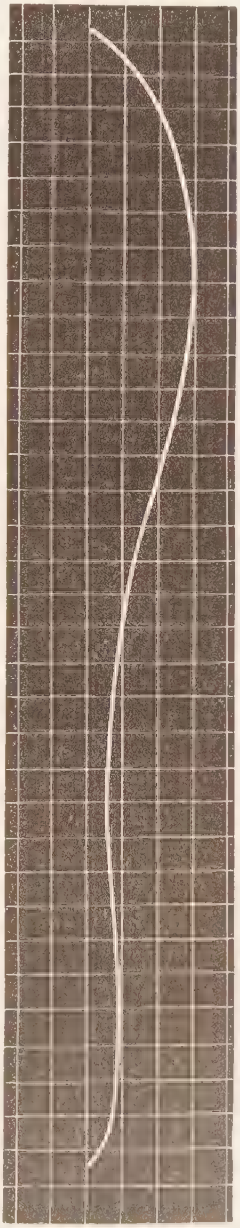


Running High Jump.

Copyright 1887, by Eadweard Muybridge.

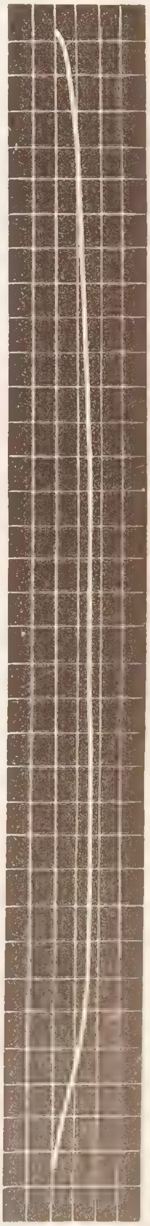
rise and fall of the right anterior superior spinous process of the ilium, which is observed to be similar in general course to the line *a*, but the amplitude of the wave is greatly increased. The spinous process has been selected in preference to the pubis employed by Marey, as being more definite. Of greater interest still is the line *e*, which represents the rise and fall and forward movement of the external malleolus of the left foot. This curve really consists of a number of elements, as elaborated by Dercum, in whose own words it is best given: "The curve begins, *e*, by the malleolus sweeping upward on an arc the radius of which centres in the ball of the great toe. At the next instant the centre of rotation is transferred to the tip of the great toe. The balance of the curve up to its highest point is the result of the flexion of the leg and of the forward movement of the pelvis; thence to the final impact of the heel upon the ground the curve is the resultant of a complex movement, in which three principal elements are distinguishable: first, a pendulum movement; second, a fall; and, third, a forward movement, the latter being due to the movement forward of the body as a whole. The first two elements are those of a cycloid, and the foot therefore falls to the ground, other things being equal, along the line of swiftest descent.

FIG. 2 *e*.



e, rise and fall of left foot.

FIG. 2 *f*.



f, lateral sway of left foot.

"A fourth element is observed in the slight secondary rise occurring in the curve just previous to its termination. The significance of this rise is as follows. We notice that the heel of the passive leg in swinging forward in its cycloid-like descent does not immediately strike the ground, but that just previous to the impact it again makes a slight ascent. This is shown not only in the curve, but also in all the plates illustrating the normal walk."

From a careful study also of the lateral sway of the foot, *f*, its extent was found to be much less than was expected, and, being the least possible with the working of the limb, shows a conservation of energy. It is thus found in the normal walk, taking all the trajectories together, that the three movements forward, laterally, and vertically are correlated, and that the greatest economy of force and time results when the secondary (lateral and vertical) movements are reduced to a minimum. This conclusion is still further strengthened by observing the advent of fatigue in ordinary marching and when particular methods of marching, demanding increased vertical or lateral movements, are attempted.[1]

In slow walking there is a period when both feet are on the ground together, a circumstance which does not exist in fast walking, in which one foot leaves the ground the moment the other touches it, which accounts for the fact that slow walking fatigues more quickly than rapid walking. The length of the step depends upon the length of the swinging leg, though this may be diminished or increased by direct muscular effort, as when soldiers of unequal height keep step,—a mode of marching obviously fatiguing and involving an unnecessary expenditure of energy.

RUNNING.—The short interval described in slow walking when both feet touch the earth at the same time is in running replaced by an interval when both feet are off the ground at the same moment. To prevent the body from falling during this interval, a quick short leap, or kind of jerk, is given to the body by quickly flexing the active leg at the commencement of the step and forcibly extending it. The duration of the pressure of the feet upon the ground is less than in walking, this being proportioned to the energy with which the feet tread. These two elements which characterize running—force and brevity of pressure—increase generally with the speed, as does also their frequency, though the extent of space travelled may depend upon the extent of each fall rather than upon their number, as in some forms of running.

From the vertical trajectories furnished by the graphic method of Marey, he believed that the suspension of the body at each impulse of the feet was not effected by a quick leap, as is generally supposed. These show that the body executes each of its vertical elevations *during* the downward pressure of the foot, and begins to rise as the foot touches the ground, "attains its maximum elevation at the middle of the pressure of the foot, and begins to descend again in order to reach its minimum at the moment when one foot has just risen, and before the other has reached the ground." From the relation of the vertical oscillations to the pressure of the feet, he plainly shows that the time of suspension does not depend on the fact that

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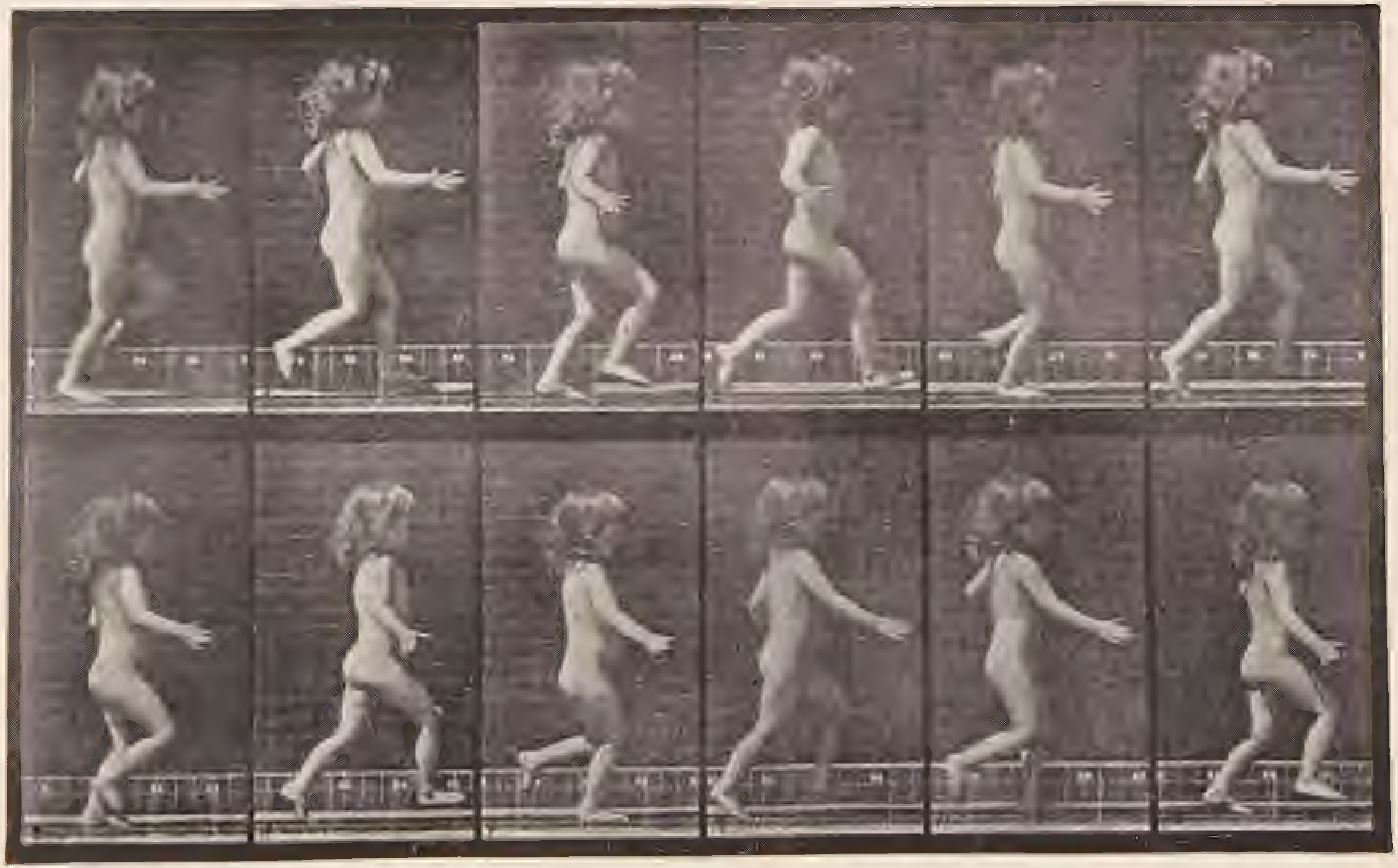
[1] See Russian Imperial Guards' March, Park's Hygiene, p. 392.

PHOTO. II.



RUNNING CHILD.

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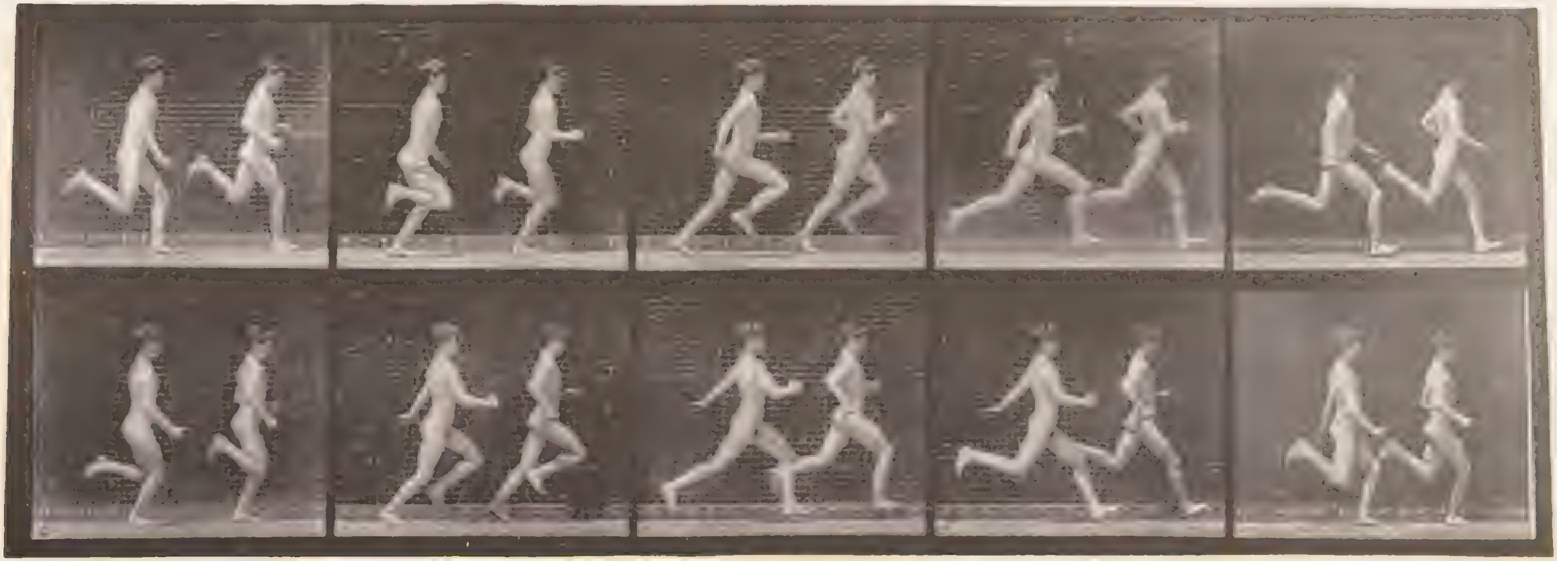
PHOTO. III.



RUNNING BOYS.

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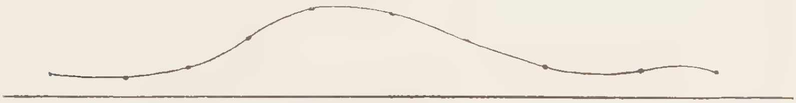
the body, projected into the air, has left the ground, but that the legs have withdrawn from the ground by the effect of their flexion, which takes place at the moment when the body is at its greatest elevation. (See Photos. II. and III., Running.)

Fig. 3 represents the rise and fall and onward movement of the right malleolus of the first running boy. The curve is similar in its general course to the line *c* in walking, except that its amplitude is much less, showing that the upward movement is more quickly accomplished than in walking. Its comparative height is also greater. Compared with line *c*, it displays clearly the difference between these two modes of progression. The slight rise just before the completion of the movement has been added: this rise, on account of its great rapidity, is lost in running; but it is always present in all slower movements, as in walking. It is found, likewise, in the motion of the horse.

FIG. 3.



Rise and fall and onward movement of right foot of first running boy.

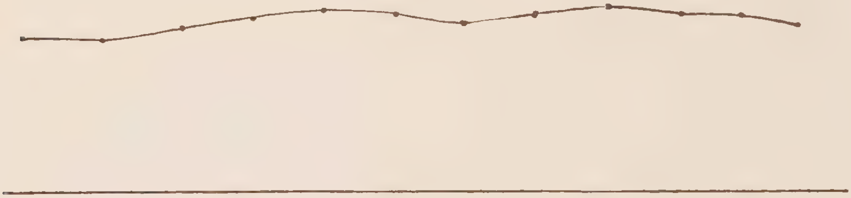
The muscles concerned in running are those described under Walking, the action being more severe and extensive.

ROWING.—Rowing is one of those exercises which call into play almost all the muscles of the body, those of the trunk, as well as those of both the lower and the upper extremities. In sitting upright the body rests upon the tubera ischii, upon which, as pivots, it swings backward and forward. The handle of the oar being grasped by both hands, the first action is a compound one, involving first the movement of the trunk or body, and secondly that of the entire upper extremity on the trunk: the body is swung forward to an angle of forty-five degrees, and the arms are fully extended. The second action consists in the recovery of the trunk simultaneously with the retraction of the shoulder and the flexion of the arm, supplementary to which, by a rapid extension of the wrist by the three extensors of the thumb, feathering is accomplished. Simultaneously with the commencement of the forward swing the abdominal muscles contract, and the body is drawn forward by the psoas and iliac muscles, supplemented by the sartorius and tensor vaginæ femoris. The extension of the arms to their fullest length is accomplished by the combined action of the serratus magnus and pectoralis minor, the triceps and anconeus. The oar is now lowered into the water and the boat is propelled by the retraction of the scapulæ or shoulders by the trapezius, latissimus dorsi, and rhomboidei, aided in some degree by the pectoralis major, the drawing backward of the entire trunk from the angle of forty-five degrees to the upright (ninety degrees) by the powerful glutei muscles, and the flexion of the arm at the elbow by the triceps and the brachialis anticus. It will be observed in this description that but little action is attributed to the erector spinæ and other muscles of the back: these appear simply to render the spinal column inflexible, as their greatest range of action cannot exceed from one to two inches.

The important action of the muscles of the lower extremity as adjuvants in good rowing is exhibited in the following manner. In the forward swing the extensor quadriceps and leg flexors are slightly relaxed and the knees bend a little outward. Then follows the contraction of the biceps, semi-tendinosus, semi-membranosus, gracilis, and sartorius, and, later, at the commencement of the recovery, in conjunction with the glutei, first the quadriceps and then all the muscles of the thigh and leg are powerfully contracted, fixing the foot and leg firmly against the "stretcher" and providing a fulcrum upon which the powerful glutei can act. A considerable number of small muscles of minor importance are also continuously brought into play. (See Photo. IV.) From this brief sketch it will be observed that the muscles of the hips, of the thighs and legs, of the lumbar region, of the upper and dorsal region of the chest, of the arm and forearm, and of the interior pectoral region, are all exercised in rowing, their importance being in the order named.

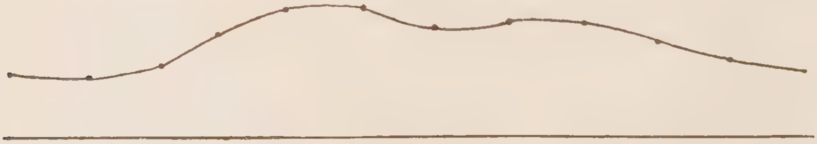
The first trajectory of rowing (Fig. 4) represents the rise and fall of the top of the head. It commences just as the body is thrown farthest forward, and gradually rises until in number five, when the arms are fully extended and the greatest force is being exerted, the highest point is reached. It is lowered slightly during the next three figures, when the final effort is made, and is again elevated as the recovery is effected during the forward movement, reaching the low initiatory point again.

FIG. 4.



Rise and fall of head in rowing.

FIG. 5.

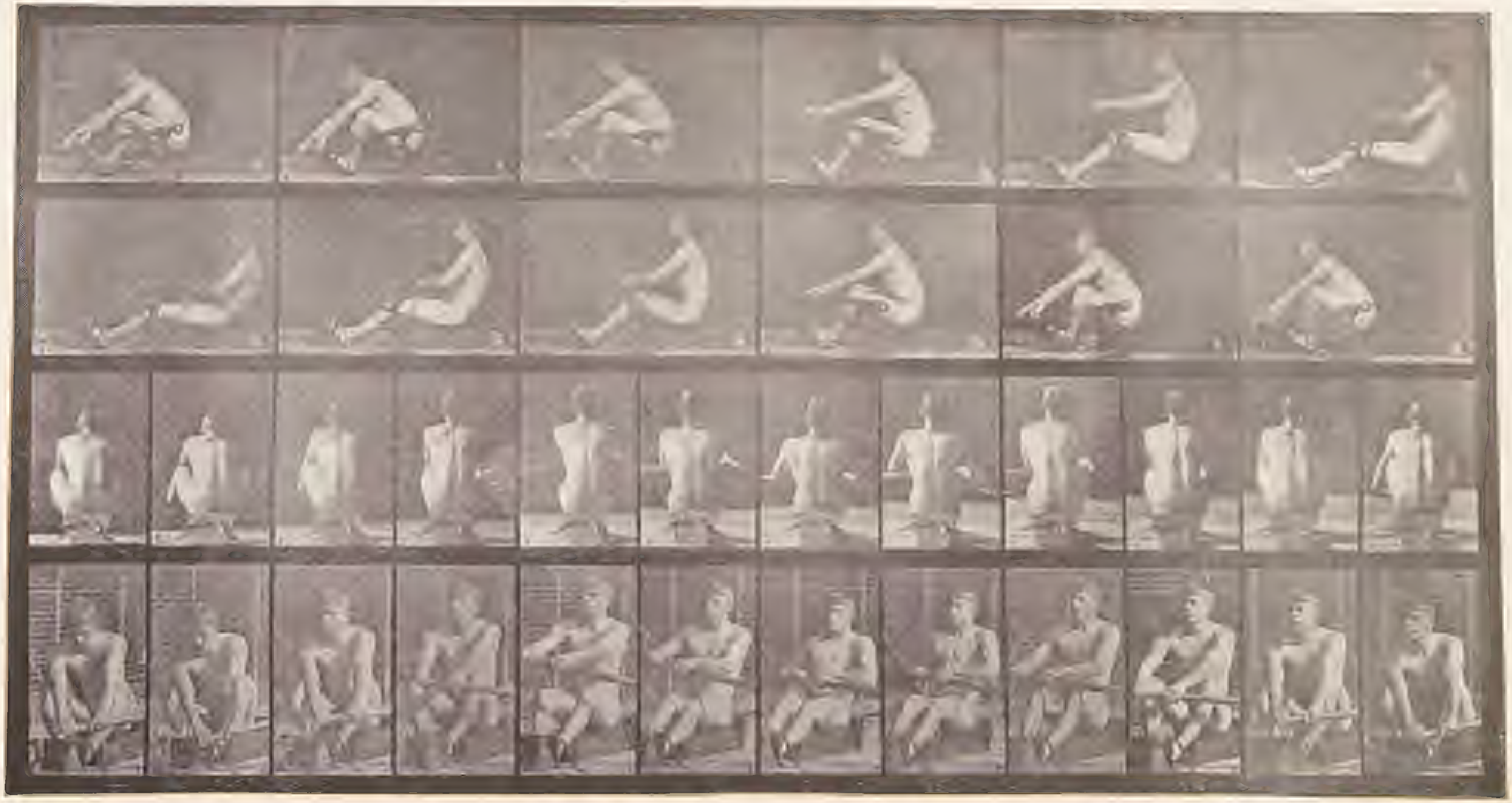


Rise and fall of hand in rowing.

The second trajectory of rowing (Fig. 5) represents the rise and fall of the first knuckle on the left hand.

This corresponds in some respects to the former, but the amplitude of the curve is greater, and in the second portion of the curve the same down-

PHOTO. IV.



ROWING MAN.

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ward curve is observed, due to the lowering of the body. This was at first thought to be due to the position of the hand in feathering; but this movement is accomplished during the last two and first two figures.

SWIMMING.—Swimming is an act unnatural to man, to learn which he must serve a longer or shorter apprenticeship to a new form of locomotion and acquire a new order of movements. The human body is lighter than water, and the difficulty first experienced in keeping the body afloat is usually referable to nervous agitation, and to spasmodic and ill-directed efforts of the extremities. Most quadrupeds can swim the first time they are immersed, not only because they are lighter than water, but also because the movements of locomotion employed by them in their horizontal position are precisely those required in swimming. Man, likewise, must assume the horizontal position upon the water, either upon the dorsal, vertical, or lateral surfaces of the body. In this position the extremities can be employed to the best advantage, and the body may be propelled in any direction.

The position most commonly employed in swimming is the prone one, with the vertical surface directed towards the water, and the head and shoulders and upper part of the back kept up by the trapezius, rhomboidei, levator scapulæ, serratus posticus superior et inferior, multifidus spinæ, etc.

The upper and lower extremities are simultaneously and slowly flexed and drawn towards the body, after which they are simultaneously and rapidly extended and directed away from the body, these movements closely resembling those employed by the frog in swimming. These simultaneous movements of the extremities describe a series of ellipses, an arrangement which, as pointed out by Pettigrew,[1] increases markedly the area of support furnished by the moving parts.

In the ellipses the continuous lines represent extension, the dotted lines flexion, describing as the extremities are flexed and extended a series of ellipses, which as the body advances are opened out and formed into loops, and, if a sufficiently high rate of speed be attained, these loops are converted into waved lines, as in flying or walking.

The alternate flexion and extension of the limbs decrease and increase the angles made by their several parts with each other, thus diminishing and augmenting the degree of resistance experienced by the swimming surfaces, and enable the extremities to elude and seize the water by turns.

Flexion is more slowly performed than extension, and the limbs are made to rotate in the direction of their length in such a manner as to diminish the resistance during flexion and increase it during extension, thus contributing force to the propulsive effort.

During the extension of the arms the palms of the hands and the inner sides of the arms, directed downward, assist in buoying up the anterior portion of the body. Towards the end of extension the hands are screwed slightly round, and the palms are pronated and directed outward and backward, assisting the posterior portions of the arms in the propulsion of the body. During flexion the palms of the hands are directed downward, and at the close of the movement they are slightly depressed, forcing the body upward, and giving to the head the bobbing or vertical wave-movement so often observed.

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[1] Pettigrew, Animal Locomotion, etc., p. 81.

When the lower extremities are extended, the soles of the feet, the anterior surfaces of the legs, and the posterior surfaces of the thigh, directed outward and backward, propel the body forward. This propulsive effort is increased by their becoming more or less straight, and by the greater rapidity with which the extension is performed. The inner surfaces of the lower extremities act upon the water in sustaining the posterior part of the body, assisted also by the slightly-inclined position of the body in the water in conjunction with the forward movement.

The flexion of the inferior extremities likewise is performed more slowly than the opposite movement.

Several grave objections urged against the ordinary or old method have led to the adoption of new methods. The prone position of the body upon the water exposes a large resisting surface; the extremities in the lateral position are applied at a disadvantage as propelling agents; but one-fourth of the ellipse is available during the propulsive effort, three-fourths being lost, with increasing friction; and the simultaneous action of both extremities leads to dead-points. To overcome these objections scientific swimmers have adopted the *overhand movement,* the method employed by the Indians, in which first the arm and leg on one side of the body are moved, and then the arm and leg on the opposite side. The direct advantage of this mode of swimming consists in the body being thrown more or less on its side at each stroke, the body twisting and rolling in the direction of its length, thereby reducing the amount of friction experienced in forward motion. In the overhand movement the swimmer is enabled to throw his body forward on the water and employ his extremities in a nearly vertical instead of a horizontal plane, a position best calculated for developing their power and reducing friction. The length of the effective stroke is doubled, being equal to nearly half an ellipse; the alternate operation of the sides not only contributes to continuity of motion, but also prevents dead-points or halts, and reduces friction. For these reasons it is the most expeditious method of swimming yet discovered; but it is so fatiguing that it can be indulged in only for short distances. The speed attained by man even by this method contrasts very unfavorably with that of seals, and still more unfavorably with that of fishes, owing to the small hands and feet possessed by him, and to the awkward manner in which they must be applied as propellers.

For long distances an improvement on the overhand movement is what is known as the *side-stroke.* This consists in swimming upon the side of the body; one arm, say the left, advanced in a curve, describes the upper side of an ellipse, while the right arm and both legs by a powerful backward stroke propel the body forward. The extension of the lower extremities accomplishes a double movement. When extended or pushed away from the body, they include within them a fluid wedge with the apex directed forward, and when fully extended they are converged in such a manner as to force the body away from the wedge and so contribute to the propulsive movement. The upper arm may also be extended in such a manner as to act as a cut-water, being advanced as the other three extremities are flexed, and *vice versa.* Either side may be employed, or they may be alternated. This plan reduces the amount of resistance to the forward movement, secures in great measure continuity of movement, and conserves the energy and resources of the swimmer to a pre-eminent degree.

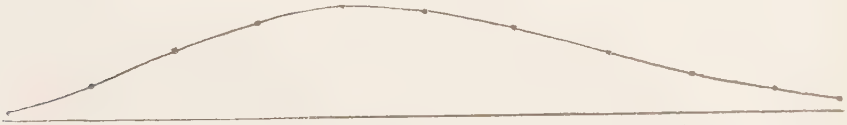
It will be seen from the foregoing account that all the muscles of the body are more or less exercised in swimming, but the forward motion is accomplished by the extreme force of all the extensors alternately or synchronously of the upper and lower extremities.

HIGH JUMPING.—The interval described in running when both feet are off the ground at the same time is in leaping much prolonged, and during this period the body is propelled both forward and upward by a violent effort of the extensor muscles (extensor quadriceps) of the thigh, the heels being raised by the contraction of the powerful calf muscles (gastrocnemius, soleus, plantaris, flexor longus, etc.).

The leap is usually preceded by a short run, to give momentum to the body, but the upward movement is accomplished entirely by the lower extremities, as above described. In some forms of leaping, particularly the standing high jump, much greater heights have been attained by employing a peculiar form of jumping copied after that of the cat. In this the jump is made sideways, the full force of both lower extremities is secured, the body is partially rotated during its flight, and the limbs drawn up are carried across the bar together. (See Photo. V.)

Fig. 6 represents the rise and fall and onward movement of the external malleolus of the right foot in high jumping. Its general outline resembles line *c* in walking, but the amplitude is much greater even than this, and it will be especially observed that in the first portion of the curve the rise is more quickly accomplished than the descent. The lower curve terminates abruptly when the feet reach the ground together.

FIG. 6.



Rise and fall and onward movement of right foot in high jumping.

By continuing this exercise great skill will be attained, and the extensors will be wonderfully developed, so that a person can jump easily not only to an increased height but also a considerable length, and land in any position desired. As an exhibition of skill the length of the run may be diminished, or the jump be repeated immediately. A good jumper is a practical example of what may be accomplished by continued exercise of certain muscles; but the most important factor—the development of the chest and its contents—has been neglected, and very often such persons suffer from consumption or heart-disease because the chest is constantly contracted to fix the ribs. True gymnastics are founded upon an anatomical and physiological law,—that in every position or exercise the full expansion of the thorax must be first considered, and the heart must not be over-stimulated. Harthelius says that in the Swedish system of gymnastics every movement, from the above law, may be looked upon as a respiration movement.

VARIETIES OF EXERCISE.—Among the early Greeks the five favorite exercises constituting the *pentathlon*—running, leaping, wrestling, hurling the lance, and casting the discus—were admirably adapted to develop the body with strength and vigor, and to confer the grace, celerity, and accuracy of movement which were so much coveted. These exercises were supplemented by games of ball, lifting and carrying weights, swimming, pugilism, and other manly and athletic sports, and revealed the recognition of the great principle of variety, there being no such potent cause of fatigue as monotonous repetition of the same act, whether physical or mental.

The tendency of all forms of exercise is to develop some portion of the body at the expense of the rest. In walking, running, and rowing, undue employment is given to the muscles of the trunk and the lower extremities. Indeed, it is difficult to find a variety of exercise that can be sufficiently repeated to exercise the muscles of the upper extremity so as to counterbalance the excessive development of the lower, or that calls equally into action all the muscles of the body. No system of physical education can be complete unless it aims at the symmetrical development of the whole body.

To the natural varieties of games and exercises employed by boys—base-ball, foot-ball, cricket, rowing, swimming—should be added systematic physical instruction in schools and gymnasiums, directed particularly with a view to develop the neglected and weak parts and to add to the symmetry of the whole.

The different varieties of exercises may be enumerated as follows: those exercising nearly all the muscles of the body,—climbing, sparring, fencing, and swimming; those exercising both the upper and the lower extremities,—foot-ball, rowing, shooting, tennis, rackets, lacrosse, and cricket; and those exercising chiefly the lower extremities,—running, walking, riding, dancing, and leaping.

EFFECTS OF EXERCISE.—The direct effect of exercise—which has already been described as a death or burning up of the cellular elements of the body—is an increased demand for oxygen to produce this combustion, which must be supplied through the lungs, thereby producing a quickened breathing. The respirations quicken, the pulmonary circulation is accelerated, the quantity of air inspired and of carbonic acid expired is marvellously increased. The increased quantity of air inspired under a variety of movements has been carefully investigated by Dr. Edward

Smith,[1] with the following result: taking the amount of air inspired in the recumbent position as 1, it increases on standing to 1.33, on walking one mile per hour to 1.9, on walking six miles per hour to 7, on riding or trotting to 4.05, and on swimming to 4.33.

Not only are the respiratory efforts deepened with exercise, but the frequency of the alternate acts of expansion and contraction of the chest is directly increased. For example, the adult healthy average, being 14–18 per minute, would be increased in walking rapidly to 25, in running to 36, and in rowing at racing speed to over 40 respirations per minute. At the same time the amount of carbonic acid eliminated is relatively lessened; thus, with twelve respirations per minute it is 4.2; with twenty-four, 3.3; with forty-eight, 2.9,—the absolute quantity, however, being increased.

To supply this increased demand for oxygen, the speed with which the blood circulates through the body must be accelerated. By a more rapid and forcible impulse of the heart, a larger quantity of blood is sent through the lungs and larger supplies of oxygen are taken in and carried to the various tissues of the body. In other words, exercise not only quickens the breathing, but also increases the rapidity and force of the heart's action. To make a similar comparison, Bryan Robinson has shown that the pulse of a man in the recumbent position, being 64 to the minute, was increased to 78 during a slow walk, and still further increased to 100 by walking a league and a half in an hour, and rose as high as 140 to 150 after running as rapidly as possible. It has also been shown by the experiments of Guy[2] that the increase from 66 in a man lying down to 71 on sitting up, and to 81 on standing, was entirely dependent upon the quantity of muscular force put forth in maintaining the equilibrium in each of these positions. When men have gone through a good deal of exertion and then are called upon to make a sudden effort, Park[3] has known the pulse to become very small and quick (160–170), but still retain its equability, and without any harm resulting. Something similar may be experienced, even by men in excellent condition, at the beginning of sudden or violent efforts, as in a foot- or boat-race, but as soon as the reciprocal action between the heart and the lungs is re-established the individual is said to have acquired his "second wind," and may continue the effort to the point of great fatigue. As long as this reciprocal action between the heart and the lungs is maintained, the interchange of oxygen and carbonic acid takes place with sufficient rapidity; but when this fails, the absorption of oxygen diminishes, carbonic acid accumulates in the blood, producing "breathlessness," or loss of wind," or, in other words, "blockage" has occurred. This condition is produced by three agencies,—interference with the passage of the blood through the lungs, its accumulation in the right auricle and ventricle, and the circulation of carbonic-acid-laden blood through the system. When exercise is regularly taken, the arteries accommodate themselves to the strong action of the heart, and a gradual improvement in the breathing-power occurs, with the establishment of the concordant action between the heart and the blood-vessels.

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[1] A Treatise on the Animal Economy, p. 177, Dublin, 1732.

[2] Cyclopædia of Anatomy and Physiology, vol. iv. p. 188.

[3] Op. cit., p. 376.

The phenomena of increased breathing-power and increased heart-action are not without their beneficial influence upon the other parts of the body, for exercise includes all the conditions requisite to increased health and strength. At the commencement of an exercise the contraction of the voluntary muscles put into action compresses the blood-vessels and impels the venous blood actively towards the heart, which, thus stimulated, contracts vigorously and propels the blood in increased quantity to the lungs. Stimulated by the presence of a large amount of venous blood, the inspiratory muscles contract, and elevate the osseous structure of the chest, the diaphragm pushes down the abdominal contents, and air rushes in to fill the cavity thus produced and supply the oxygen demanded for the purification of the blood. Laden with this life-giving element, it is returned to the heart, to be distributed again throughout the system to restore the loss incurred through the original muscular movement. In this manner not only are the voluntary muscles enlarged and strengthened, but the involuntary muscles, particularly the heart and diaphragm, improve in power and function. The increased activity of the circulation carries the blood in increased quantity and with greater rapidity not only to the muscles but also to all the other tissues and organs of the body, stimulating them to increased activity. The skin becomes red from turgescence of the vessels, the amount of perspiration is more than doubled, water, chloride of sodium, and alkaline sudorates pass off in great abundance, and fatty acids, urea, and other salts in smaller quantities. The appetite is largely increased, digestion is more perfect, absorption is more rapid, hepatic circulation increases, the abdominal circulation is carried on more vigorously, and the fæces are lessened in amount, probably from the lessened passage of water into the intestines. Owing to the increased elimination of water and sodium chloride by the perspiration, the amount of these two elements in the urine often lessens; the uric acid, pigment, and free carbonic acid are increased; the sulphuric acid is moderately increased, and the urea and phosphoric acid are but little changed.

It has been supposed that puberty is delayed by physical exertion; but, be this as it may, it is established that very strong exercise lessens sexual desire, possibly because nervous energy is turned in a special direction, but doubtless also through augmented moral power, the association between physical, intellectual, and moral strength being a natural one, unchangeable in its essential principles, though subject to individual exception. The beneficial effect upon the nervous system is equally striking. It has been supposed that excessive exercise renders the intellect less active, owing to the greater expenditure of nervous force in that direction; but not only is great bodily exercise quite consistent with extreme mental activity, but, considering the principle inculcated in the oft-repeated line from Juvenal of *mens sana in corpore sano*, we must infer that sufficient exercise is necessary for the perfect performance of mental work. From the stand-point of a comparative physiologist, Du Bois-Reymond demonstrated that the most marked influence of physical exercise is upon the nerve-centres. In every bodily movement of a composite nature, as fencing, swimming, sparring, or high jumping, the gray centres of the brain and cord are at work equally in securing the result, and are exercised at the same time.

So closely are the mind and the body correlated that it is hard ofttimes to distinguish what is due to the mind and what to the body. In very early childhood, with the dawn of mental and physical development, this is particularly noticeable, and Prof. Richards, of Yale University, under the title of "Body Brain Work," has described it as antedating the advent of brain-growth. Every time a child co-ordinates a well-directed movement, that movement exercises and develops its brain, and the movement of the muscles is as necessary to the mental development as the health and integrity of the brain are to the physical development of its parts.

The muscles and the brain are developed by reciprocal action, and in later life a direct relation is found to exist between great physical strength and the possession of those intellectual powers which naturally lead men to commanding positions of authority. Ancient, mediæval, and modern history alike contain numerous instances to prove the accuracy of this statement. "Samson, though he seems to have lacked discretion, was a judge in Israel. Pompey was the equal of any soldier in his command in feats of strength. Sallust says of him, 'Cum alacribus saltu, cum velocibus cursu, cum validis vecte certabat.' Cæsar was naturally of a delicate constitution, suffering from severe headaches, and probably epileptic, but by continual exercise became an athlete, 'admirable in all manly sports,' and surpassed by none in enduring the fatigues and hardships of a military life. Lycurgus not only laid down the laws which for five hundred years made Lacedæmon the chief city of Greece, but was able to outrun all the mob who persecuted him and forced him to seek refuge in a sanctuary. Cicero is described by Plutarch as at one time thin, weak, and dyspeptic, but as having been so strengthened by gymnastic exercises at Athens as to become robust and vigorous. Coriolanus's successes were attributed by his enemies to his strength of body, he having so exercised and inured himself to all sorts of activity that he 'combined the lightness of a racer with an extraordinary weight in close seizures and wrestlings.' Alcibiades, according to Herodotus, became master of the Athenians, in spite of his excesses, by reason of his 'force of eloquence, grace of person, and strength of body,' and from the same authority we learn that Alexander had unusual endurance. Themistocles, Socrates, and Plato excelled in gymnastic exercises; Sertorius swam the Rhone in full armor; Marcellus was 'of a strong body;' Pelopidas 'delighted in exercise;' Marius never missed a day on the Campus Martius; Cato 'maintained his character and persisted in his exercise to the very last;' and even the more mythological characters of

Theseus, Romulus, and Remus are accredited with 'strength of body and bravery equal to the quickness and force of their understanding.' "[1]

In the "University Oars" Dr. Morgan calls attention to the fact that of the one hundred and forty-seven Cambridge men who constituted the crews from 1829 to 1869, twenty-eight per cent. won honors in more important contests than those upon the river, securing in some instances the very highest academical distinction, and proving that mind and muscle, judiciously guided, are well able to work together with reciprocal advantage.

Sargent has pointed out that college men take about the same grade in their general studies as in required athletics,[2] and Dr. Morgan has shown that, while the general average of class men at Oxford was about thirty per cent., among cricketers it rose to forty-two per cent. and among rowing men to forty-five per cent.

In a recent investigation by a prominent American physician to show the comparative longevity of brain-workers as compared with muscle-workers, the advantage was clearly shown to be in favor of the former. This might have been expected *a priori* in view of the classes from which these so-called "muscle-workers" are naturally derived, who through poverty are forced all their lives to live under the most unsanitary conditions, who neglect from ignorance and powerlessness the most obvious laws of health, and who in all respects labor under a stupendous physical disadvantage when brought into comparison with the so-called "brain-workers." Their work is not exercise; it is fatiguing labor, performed ofttimes under the most depressing intellectual conditions.

On the contrary, it is not difficult to prove that a sound constitution usually accompanies a healthy brain, and that the cerebral and the muscular forces are directly correlated. In this connection, Dr. Beard declared that in all the animal realm there is a general, though not unvarying, relationship existing between the brain and the body, and that no one who has ever walked observingly through an asylum for the insane or feeble-minded and beheld the dwarfed, misshapen, immature, or stunted forms which surround him can doubt the force of the truth embodied in this statement. He points out the rapidity with which such poor creatures grow physiologically old, the evidences of senility noticeable in every organ and function,—in the gray hair and premature baldness, in the dimness of vision and dulness of hearing, in the wrinkled skin, the tottering step, the wasted limbs. Conversely, he maintains that one hundred great geniuses chosen by chance will be taller, broader, and weightier than a hundred dunces anywhere, and declares that in all lands, savage, semi-civilized, and enlightened, the ruling orders, chiefs, sheiks, princes by might and mind, authors, scientists, orators, great merchants, weigh more on the average than the persons whom they rule or employ, and that even among a band of workmen on a railway one can four times out of five select the "boss" by his stature alone; and Bates tells us that among the people of the Tapajos the footmarks of the chief could be distinguished from the rest by their great size and by the length of the stride. On the same principle Herbert Spencer has shown how in early times among rude tribes political leadership was associated, as in the present times, with physical strength.

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[1] White, Lippincott's Monthly Magazine, June, 1887, p. 1013.

[2] "In this connection it is worthy of mention that the most athletic seniors in the classes of 1885–86 and 1886–87 (Harvard) included one honor man who received honors, nine who received honorable mention, and twelve who were entitled to write commencement parts." —*Report upon Athletics, etc.*, Cambridge, 1888, p. 22.

In early Greece the veneration of age did not recompense for loss of strength, and an old chief, like Laertes or Peleus, had to relinquish his position. Throughout mediæval Europe the maintenance of political leadership depended largely on bodily vigor. Supremacy among the Australians and Tasmanians depended upon physical size and strength. The Esquimaux exhibit deference to "seniors and strong men;" among the Bushmen "bodily strength alone procures distinction;" and "the fiercest, the strongest, and the craftiest" among the Bedouins "obtains a complete mastery over his fellows."

The direct influence of exercise upon longevity has been curiously brought out in a list prepared not to show the average longevity, but to determine the age when great men have performed their best work: the average age at death was sixty-four. Likewise Madden, in his odd book upon "The Infirmities of Genius," in a list of two hundred and forty illustrious men, found the average age at death to be sixty-six, proving that many of the great men of the past have been noted for physical strength as well as for intellectual greatness, and that the attention given by them to physical development had been productive of both great mental ability and increased tenure of life. Personal examples from among the distinguished men of the present century, justifying this statement, might be produced indefinitely.

These, then, are the physiological effects of exercise,—an increase of the voluntary muscles in volume and power, an enlarged respiration and quickened circulation, through a strengthening of the involuntary muscles concerned in these processes, and an improved action of all the functions and faculties concerned in the growth and development of the whole body, the force and activity of the intellectual and moral processes, and the longevity of the individual.

While the beneficial effects of exercise are so great and so important, the fact must not be overlooked that it is also capable of great abuse. When unaccustomed exercise is engaged in after a long interval of rest, "blockage" occurs not only of the arteries going to the lungs, but also of those supplying the whole body, particularly when violent exercise is undertaken without due preparation, as when a tired, delicate, or overworked man, for months engaged in a sedentary, literary, professional, or mercantile pursuit, or a tender untrained student, suddenly indulges in some active or violent form of gymnastics, or engages in one of the recreative sports which make the greatest demand upon the heart and lungs, as boating or running: instead of a beneficial result accruing, serious and often permanent injury frequently follows. Such important involuntary organs as the heart and lungs cannot instantly pass from the quiescent state of ordinary life movements to great rapidity of action without strain being thrown upon them, and, instead of the vital action being quickened and the processes of repair and removal stimulated, the blood is blocked in the arteries, the cavities of the heart are over-distended, and dilatation or hypertrophy speedily results. To avoid this should be regarded as of vital importance in all forms of exercise, but especially in training should it be borne in mind that the chief object is "to establish a reciprocal action between the heart and the lungs, so that the increased supply of blood sent to the lungs by the heart may pass through them freely, so that there may be no blockage and consequently no strain."

It must also be recollected that the development of the voluntary muscles gives no indication whatever of the condition of the involuntary muscles involved in maintaining the respiration and circulation, and that an enormous muscular development may be absolutely useless without the cultivation of the heart and lungs, a circumstance pointed out by Maclaren to explain the result of the celebrated Heenan-King prize-fight.

Exhaustion of muscles from overwork is chiefly owing to the exhaustion of the supply of oxygen, and to the accumulation in them of the products of their own metamorphosis, especially paralactic acid. This is the result of the effect of general fatigue upon the nervous system, the heart, and the circulation of the blood. Hence rest is demanded in order to allow the removal of waste products from the tissues, to restore their alkalinity, and to acquire a sufficient quantity of oxygen. Thus, in the heart the interval between the contractions (about two-thirds of the time) is sufficient to allow the muscular structure perfectly to recover itself. Muscles that have undergone exhaustion have nutrition seriously impaired, as manifested by the so-called "acute local fatigue," not simply in loss of power, but often in irregular, painful muscular contractions, cramp, tremor, and peculiar, distinguishable muscular pain.

To avoid the results of these spasmodic efforts, children and youth should not be allowed to undertake any form of exercise calling for sudden or violent exertion until a certain age is passed, and certainly not without having been examined by the regular medical attendant before, or by the medical director upon, their entrance to the school or college gymnasium, to ascertain the condition of the heart and lungs, the general muscular development, and the existence of any hereditary taint. This would not conflict with the physical examination, to which full reference will be made under the subject of physical education, but would be preliminary and supplemental to the more thorough examination of this kind made to ascertain the development.

The evil effects resulting from the lack of perfect exercise are not so evident in the period of life under consideration as in later life, when the more active sports of youth are replaced by the sedentary pursuits and the enervating influences of a business life.

EXERCISE IN INFANCY AND CHILDHOOD.—The natural movements of an infant are the apparently purposeless muscular efforts which it makes as soon as it appreciates that it has extremities. "A child in health delights in movements of all kinds," says Sir William Jenner. "It joys to exercise every muscle. Strip a child a few months old, and see how it throws its limbs in every direction; it will raise its head from the place on which it lies, coil itself round, and grasping a foot with both hands thrust it into its mouth as far as possible, as though the great object of its existence at that moment was to turn itself inside out." These movements are its natural exercise, developing the muscles and brain correlatively, as already pointed out. For this reason its limbs should not be too much compressed or weighted down with heavy clothing, and it should be encouraged to roll about and exercise itself as much as possible. Its first attempts at combined movements, to crawl on all-fours, quadruped-like, are soon followed by attempts to assume the upright position by the support of some object, and endeavor to balance the upper part of its body, which at this time is developed far beyond the lower extremities. Sunlight and fresh air are at this time very necessary to add firmness and strength to the whole structure, and with the exercise of its limbs the upright position is soon acquired, and the child has gradually *taught itself* to walk.

These first efforts should be encouraged, but should not be too prolonged, lest injury result. As time advances, and the child becomes more and more independent of support, the constant activity and impatience exhibit plainly the intentions of nature, and after the child is three years old a degree of activity may be imparted by encouraging it to run with a ball, play with a small dog, or, later, exercise with a hoop. In this manner from a very early age exercise may be begun and be encouraged, a rocking-horse may be added to the nursery, later the careful use of a velocipede or tricycle may be employed in the open air, to be still further substituted, as the child advances, by the use of roller-skates and the exchange of the rocking-horse for the pony. In all these forms of exercise great care should be observed that the saddles be not too wide, lest deformity result. It is also to be distinctly understood that these are to be employed with the greatest caution; and if the child have hernia they should not be employed under any circumstances. At the end of the fifth year children may spend an hour or two in the kindergarten, but regular schooling should not be commenced till the end of the seventh year, and during the later period of childhood sufficient time should always be allowed for plenty of play and exercise in the open air, regard being had to the state of the weather. Thus, many children have fallen victims to pneumonia, bronchitis, and croup from having run or walked against a piercing easterly wind, long walks and violent play through overfatigue and cold have led to hip-joint and knee disease, and drinking cold water when overheated, or sitting upon damp ground or stones, has produced irretrievable injury in many others. The younger and more delicate the child, the more care has to be exercised in regard to the state of the weather. If born in the latter part of autumn, infants cannot with safety be taken out before the return of spring; but if the apartments are large and airy, little evil will result from this prolonged confinement. With the return of mild, dry, and serene weather, they must be carried out into the open air, and, should the weather permit, this salutary practice must be daily repeated. In fine weather children and youth may spend the greater part of the day out of doors, and as they grow in years they become gradually accustomed to the vicissitudes of a changeable climate. In large cities, large open spaces, parks, and gardens should be frequented, and in the summer months the overheated and overcrowded cities should, if possible, be exchanged for the country and sea-coast.

From five to twelve, boys and girls alike may engage in any of the light active games which do not throw weight or strain upon the growing joints, avoiding wrestling, foot-ball, and premature attempts at rowing.

In early boyhood and youth nothing can replace the active sports so much enjoyed at this period, and, while no needless restriction should be placed upon them, consideration should be paid to the amount and especially to the character of games pursued by delicate youth. For these it would be better to develop the weakened parts by means of systematic gymnastic exercises, by short excursions into the country, and by the lighter sports.

It should be borne in mind that in order to obtain the greatest advantage from those exercises which are calculated to improve the physical and nervous strength the child should be interested and made to feel that these exercises are a play instead of a task.

Children who are taught at an early age to be obedient seem to enjoy more thoroughly such exercises as combine discipline with rhythmic movements; and, consequently, the older the child the more important it is to adopt a system of calisthenics, or light drill, or games that combine gymnastics with rhythmic sounds and periods of rest.

The more permanent benefits of play (games) are promptness, attention, fast and easy running, climbing, balancing, strength, endurance, marksmanship, elasticity, etc. These games may be classified as follows:

1st. Exercises which in some respects stand in the line between free games and gymnastics, since they retain some school discipline and are played systematically under the direction of the teacher. These are of varied character, some more suitable for older, others for younger children (racing, "prisoner's base," etc.), the difference between these games and gymnastics being often not easily distinguishable. For young men the best of these is fencing.

2d. Exercises which also have some value in a gymnastic sense, but which are not to be played in classes or under the command of the teacher. These are called free gymnastic games, from the fact that the teacher can take part, if so disposed, but need not do so. To a great many free games belong certain words, declamations, or the singing of different melodies. If the instructor in gymnastics for children take up dancing, the principal object should be to give the children the greatest possible liberty, and not to attempt to obtain perfect grace and form-beauty.

3d. The third and last class of plays (games) includes games of no gymnastic value. To this class belong all sitting, forfeit games. These are as useless as the great number of meaningless movements which have been introduced into gymnastics, many of which are only methods without gymnastics, which is as incorrect as gymnastics without method.

THE DEVELOPMENT OF BOYS[1] has attracted considerable attention of late years, but the systematic records are as yet insufficient to admit of any dogmatic deductions. For older children the records of college gymnasiums are available to show the rapid increase which systematic exercise causes in the physical development. We have, however, the very valuable papers of Mr. Street, F.R.C.S., Mr. Roberts, F.R.C.S., and Dr. Bowditch.

Mr. Street's observations are based upon the examination of three thousand six hundred and ninety-five boys, drawn chiefly from the artisan class, varying from thirteen to nineteen years, and are interesting as exhibiting the height, weight, etc., for the five years inclusive.

TABLE

Showing the Relation of Weight to Height of Boys between the Ages of Thirteen and Nineteen Years.[2]

HEIGHT WITHOUT SHOES, IN INCHES.	WEIGHT IN POUNDS, WITHOUT COAT, HAT, AND SHOES.							AVERAGE WEIGHT IN POUNDS.
	AGE LAST BIRTHDAY.							
	Thirteen.	Fourteen.	Fifteen.	Sixteen.	Seventeen.	Eighteen.	Nineteen.	
71	. .	. .	. .	. .	149	129	150	142.6
70	. .	. .	136	144	147	151	137	143.0
69	. .	129	142	132	138	144	140	139.4
68	. .	. .	131	126	131	142	144	134.8
67	. .	123	120	125	132	129	138	128.0
66	. .	119	117	122	126	126	130	123.3
65	. .	107	117	115	120	127	121	118.0
64	111	112	115	115	116	115	120	115.0
63	103	108	108	110	115	117	117	111.1
62	96	101	104	106	109	111	113	105.7
61	96	98	99	101	106	102	109	103.0
60	90	93	92	96	101	109	. .	97.0
59	87	89	91	93	93	91	. .	90.7
58	86	86	87	88	94	. .	. .	82.2
57	82	83	83	86	. .	. .	. .	83.5
56	78	80	78	87	81	. .	. .	80.8
55	75	76	77	76	. .	76	. .	76.0
54	74	74	74	67	. .	. .	. .	72.2
53	72	69	. .	. .	. .	. .	. .	70.5
52	73	70	. .	67	. .	. .	. .	70.0
51	62	. .	. .	. .	. .	. .	. .	62.0

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[1] Roberts, The Physical Development and Proportions of the Human Body. [2] Street.

Starting with an average height of fifty-five inches at thirteen years, these figures show an increase of one inch during the first year, of three inches during the second year, of four inches during the third year, and of one inch each during the fourth and the fifth year, whilst from nineteen to twenty there is scarcely any increase in stature. From Mr. Roberts's table the increase for each year from thirteen to sixteen is over two inches, being greatest during the sixteenth year; during the seventeenth year it is about one and one-half inches (1.53); from seventeen to eighteen it is about one and three-fourths inches, dropping during the next year to less than one inch (0.68), and during the year from nineteen to twenty to less than one-half inch (0.43).

TABLE

Showing the Average Proportion and Growth of the Human Body from Birth to Maturity.[1]

AGE LAST BIRTHDAY.	AVERAGE PROPORTIONS OF THE BODY (MALES).			ANNUAL RATE OF GROWTH.			RATIO OF INCREASE. HEIGHT = UNITY.		
	Height, Inches.	Chest-Girth, Inches.	Weight, Pounds.	Height, Inches.	Chest-Girth, Inches.	Weight, Pounds.	Height, Inches.	Chest-Girth, Inches.	Weight, Pounds.
Birth	19.34	13.25	7.55	. .	. .	. . .	.	. .	. .
1 year	28.50	. . .	. . .	9.16	. .	. . .	. .	. .	. .
2 years	31.60	. . .	. . .	3.10	. .	. . .	. .	. .	. .
3 "	35.00	. . .	. . .	3.40	. .	. . .	. .	. .	. .
4 "	38.45	. . .	31.10	3.45	. .	. . .	. .	. .	. .
5 "	41.15	21.26	37.71	2.70	. .	. . .	. .	. .	. .
6 "	43 18	21.68	40.67	2.03	0.42	2.96	1	0.20	1.45
7 "	45.15	22.25	44.00	1.97	0.57	3.33	1	0.28	1.70
8 "	46.92	22.66	47.15	1.77	0.41	3.15	1	0.31	1.80
9 "	49.52	23.27	51.20	2.60	0.61	4.14	1	6.24	1.60
10 "	51.52	23.77	55.50	2.00	0.50	4.21	1	0.25	2.10
11 "	52.87	24.33	60.15	1.35	0.56	4.65	1	0.41	3.44
12 "	54.45	24.81	64.52	1.58	0.48	4.37	1	0.30	2.76
13 "	56.56	26.30	71.00	2.11	1.49	5.48	1	0.70	2 60
14 "	58.55	28.18	79.57	2.00	1.88	8.57	1	0.94	4.28
15 "	60.77	29.70	91.43	2.21	1.52	11.86	1	0.68	5.36
16 "	63.42	31.19	107.86	2.65	1.49	16.43	1	0.56	6.20
17 "	64.95	32.80	118.08	1.53	1.71	10.22	1	1.10	6.67
18 "	65.69	34.03	127.25	1.74	1.23	9.17	1	0.70	5.27
19 "	66.37	34.76	131.48	0.68	0.73	4.23	1	0.10	6.22
20 "	66.80	35.13	135.28	0.43	0.37	3.80	1	0.08	0.90
21 "	66.80	35.42	135.03	. .	0.29	. . .	. .	. .	. .

According to Dr. Bowditch's tables there is an increase in height from the thirteenth to the fourteenth year of over two inches, from the fourteenth to the fifteenth of over two inches, from the fifteenth to the sixteenth of over two and a half inches, from the sixteenth to the seventeenth of nearly one inch, and from the seventeenth to the eighteenth of a little over one-half inch, among the non-laboring classes, these figures being slightly less among the laboring class.

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[1] Roberts, op. cit.

TABLE

Showing Average Heights and Weights of Boston School-Boys, irrespective of Nationality.[1]

AGE LAST BIRTHDAY,	OCCUPATION OF PARENTS.					
	NON-LABORING.			LABORING.		
	No. of Observations.	Height, Inches.	Weight, Pounds.	No. of Observations.	Height, Inches.	Weight, Pounds.
5 years	135	41.64	41.21	694	41.57	41.00
6 "	243	44.11	45.50	1007	43.74	45.06
7 "	294	46.23	49.77	1133	45.61	48.93
8 "	295	48.08	54.64	1161	47.67	53.67
9 "	272	50.03	59.89	1097	49.73	59.22
10 "	262	52 12	66.31	1023	51.55	64.89
11 "	284	53.84	71.81	956	53.17	69.67
12 "	277	55.92	80.38	899	54.84	75.88
13 "	277	58.13	88.59	800	56.89	83.40
14 "	265	60.52	96.54	582	59.31	93.67
15 "	231	62.68	108.81	365	61.90	104.88
16 "	169	65.23	122.48	162	64.65	119.03
17 "	97	66.17	128.23	77	65.75	125.28
18 "	46	66.69	132.00	28	66.35	131.60

We have had some interesting studies made by Dr. A. A. Eshner at the Philadelphia Hospital, which are shown in the following table:

NAME.	AGE.	HEIGHT.	WEIGHT.	EXPANSION.
C. M.	19 days.	$22\frac{1}{4}$ inches.	$8\frac{1}{2}$ pounds.	$\frac{3}{4}$ inch.
L. H.	101 "	$25\frac{3}{4}$ "	$14\frac{1}{4}$ "	$1\frac{1}{4}$ inches.
C. H.	61 "	$23\frac{1}{4}$ "	10 " 1 ounce.	$\frac{7}{8}$ inch.
M. L.	126 "	$25\frac{1}{4}$ "	15 " 11 ounces.	$\frac{7}{8}$ "
M. B.	183 "	$25\frac{1}{2}$ "	14 " 1 ounce.	$\frac{5}{8}$ "
A. O'B.	308 "	25 "	15 " 2 ounces.	$\frac{3}{4}$ "
L. G.	199 "	$27\frac{1}{4}$ "	17 " 6 "	1 "
H. P.	162 "	$26\frac{1}{4}$ "	17 " 3 "	$\frac{3}{4}$ "
O'B.	11 "	$18\frac{3}{4}$ "	4 " 12 "	$\frac{5}{16}$ "
J.	22 "	$21\frac{1}{4}$ "	9 "	$\frac{1}{2}$ "
A. C.	9 "	21 "	6 " 13 "	$\frac{3}{8}$ "
G. N.	8 "	$20\frac{3}{4}$ "	7 " $4\frac{1}{2}$ "	$\frac{3}{8}$ "
A. R.	34 "	20 "	7 " 12 "	$\frac{7}{16}$ "
O'H.	16 "	$21\frac{1}{4}$ "	8 " $8\frac{1}{2}$ "	$\frac{3}{18}$ "
J C.	33 "	$21\frac{1}{4}$ "	6 "	$\frac{5}{16}$ "
W.	35 "	21 "	7 " $8\frac{1}{2}$ "	$\frac{5}{16}$ "
W. M.	51 "	26 "	16 " 4 "	$\frac{5}{8}$ "
M. C.	319 "	$29\frac{1}{4}$ "	19 " 4 "	$\frac{5}{8}$ "
F.	238 "	$26\frac{1}{4}$ "	17 " 6 "	$\frac{3}{4}$ "
G. M.	205 "	$25\frac{1}{2}$ "	11 " 3 "	$\frac{5}{8}$ "
E.	242 "	28 "	14 " 3 "	$\frac{5}{8}$ "
M. R.	65 "	$23\frac{1}{4}$ "	$10\frac{1}{2}$ "	$\frac{7}{8}$ "
K.	270 "	28 "	$18\frac{1}{2}$ "	$\frac{3}{4}$ "
V. B.	175 "	$24\frac{1}{4}$ "	11 " 7 "	$\frac{3}{4}$ "
C. E.	585 "	27 "	10 " 4 "	$\frac{5}{8}$ "
L. C.	90 "	$20\frac{1}{2}$ "	5 " $13\frac{1}{2}$ "	$\frac{3}{8}$ "

These measurements represent but a small number of children, and they should be carried out to a greater extent. The expansion noted is, of course, the ordinary expansion of breathing, and not force-respiration.

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[1] Bowditch.

From these tables it is evident that the period in a boy's life between the sixteenth and the seventeenth year is one of great importance, during which no great strain should be thrown upon his developing constitution, and that feats of strength or physical endurance should not be undertaken until this period is safely passed.

With his entrance to college the youth has an opportunity of laying the foundation of his physical development in a use which will serve him, with proper care, through his future life. As a rule, this is what the average American student requires, for Prof. Elliott, of Harvard, said of the majority entering that institution that they had "undeveloped muscles, a bad carriage, an impaired digestion, without skill in out-door games, and unable to ride, row, swim, or shoot;" and what is true of Harvard applies with greater force to many other American colleges. With the present systems of physical culture in operation in all the important Eastern colleges, the freshman on entrance is examined as to his physical condition and advised what forms of exercise he requires, and is re-examined from time to time to ascertain his progress and advised as to his subsequent course.

The systems of physical education at present employed in Harvard, the University of Pennsylvania, and other American colleges, and copied after those long in use in Oxford, are approximately as follows. Each student upon his entrance to college is stripped, and measurements are taken of his height, weight, circumference and diameter of chest, legs, thighs, arms, and forearms, which are recorded in some convenient form (centimetres or inches). These together are taken approximately as an indication of his development: it shows the amount of working material, but not its actual working value. These are followed by a series of tests to ascertain the total available strength; by means of the spirometer, the horizontal bar, and the lifting machine, the strength and capacity of the lungs, and the relative strength of the arms and chest, back, legs, and thighs, abdominal muscles, and forearms, are all estimated. These are recorded in the same form as the first, and, representing the development and available strength, readily admit of comparison. To these are finally added the personal and the family history of the individual.

The chart employed to record the measurements, etc., is as follows:

PERSONAL AND FAMILY HISTORY.

Be sure to answer every question; say "yes," "no," or "I don't know," whenever possible.

Name,
Class and department, or occupation,
Age, Birthplace,
Nationality of——
Father,
Mother,
Paternal grandfather,
Paternal grandmother,
Maternal grandfather,
Maternal grandmother,

Occupation of father,
If parents are dead, of what did they die?
Which of your parents do you most resemble?
Is there any hereditary disease in your family?
Is your general health good?
Have you always had good health?
Have you ever had any of the following diseases?

- Asthma,
- Shortness of Breath,
- Bronchitis,
- Spitting of Blood,
- Pleurisy,
- Pneumonia,
- Palpitation of the Heart,
- Bright's Disease,
- Rheumatism,
- Neuralgia,
- Sleeplessness,
- Headaches,
- Paralysis,
- Dizziness,
- Fits,
- Rupture,
- Dyspepsia,
- Habitual Constipation,
- Varicose Veins,
- Piles,
- Liver Complaint,
- Jaundice,
- Chronic Diarrhœa,
- Dysentery.

Have you ever had any injury or undergone any surgical operation?
What have been your favorite exercises?
How much time have you devoted to them daily?
How much time do you spend in the open air daily?
How many hours do you sleep daily?
How much time do you spend in study outside of the college?
To what extent do you use tobacco?
To what extent do you use alcoholic or malt beverages?

EXAMINATION OF MR.
CLASS.

	1.	2.	3.	4.
Date				
Age				
Height				
Weight				
Girth, Head*				
“ Neck				
“ Chest, full*				
“ “ repose*				
“ Belly*				
“ R. Thigh*				
“ L. “ *				
“ R. Knee				
“ L. “				
“ R. Calf				
“ L. “				
“ R. Arm*				
“ L. “ *				
“ R. Forearm*				
“ L. “				
Cap. Lungs				
Expirat. Strength†				
Strength R. Grip†				
“ L. “ †				
“ Back†				
“ Thighs†				
Dips†				
Chins†				

a means above and *b* means below the average according to age.	CONDITION.	DEVELOPMENT.	STRENGTH.
First Examination.			
Average.			
Second Examination.			
Average.			
Third Examination.			
Average.			
Fourth Examination.			
Average.			
Improvement.			

NOTE.—Development—The sum of the nine girths marked *.
Strength—The sum of the seven tests marked †.
Condition—The difference between development and strength; if in favor of the former it is minus (—), and if in favor of the latter it is plus (+).

With such data before him, the medical examiner is in a position to advise the student upon several matters of the greatest importance. He can limit or altogether interdict exercise, he can prescribe parallel and upright bars for a rachitic chest, he can suggest the rowing-machine or exercise upon the river for the student with weak legs, he can advise the corpulent individual with flabby muscles to become more active, and he can diagnose and prescribe for inherited tendencies and latent diatheses with a scientific thoroughness based upon a careful examination and an exact knowledge of the individual which few practitioners enjoy. These physical examinations are repeated once or twice during the year, and the results carefully noted and compared.

Dr. Sargent recently, in describing the system of physical education in use at the Hemenway Gymnasia at Harvard, said,[1]—

"Every student who enters the University is entitled to an examination, and eighty-seven per cent. of the whole number avail themselves of this privilege.

"As soon as the student presents himself at the director's office (which is done by application and appointment), he is given a history blank, which he fills out, giving his birthplace, nativity of parents, occupation of father, resemblance to parents, natural heritage, general state of health, and a list of the diseases he has had, all of which information is absolutely necessary in order for the examiner to put a correct interpretation upon the observations to follow. The student is then asked to make certain tests of the muscular strength of the different parts of his body, and to try the capacity of his lungs.

"He then passes into the measuring-room, and has his weight, height, chest-girth, and fifty other items taken. His heart and lungs are then examined before and after exercise, and a careful record made of the condition of the skin, muscles, spine, etc., which the tape-measure fails to give.

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[1] Physical Training Conference, 1889, p. 65.

"All the items taken are then plotted on a chart, made from several thousand measurements, and the examiner is thus able to know the relative standing of this individual as compared with others for every dimension taken, also his deviation from symmetry, and the parts which are in special need of development.

"To confirm the plotting of the chart, and to awaken in the young man a genuine interest in his physique, a photograph of each student desiring it is taken in three positions, and preserved for comparison with those to be taken of him later.

"From the data thus procured a special order of appropriate exercises is made out for this student, with specifications as to the movements and apparatus he may best use. At the present time this special order consists for most students of an illustrated hand-book, in which the apparatus, the weights for it, and the times to use it are carefully prescribed, together with such suggestions as to exercise, diet, sleep, bathing, clothing, etc., as will best meet the needs of the individual under consideration.

"Now, I think it will be admitted by all thoughtful persons that one-half the battle for mental education has been won when you arouse in a boy a genuine love for learning. So one-half the struggle for physical training has been won when he can be induced to take a genuine interest in his bodily condition,—to want to remedy his defects, and to pride himself on the purity of his skin, the firmness of his muscles, and the uprightness of his figure.

"Whether the young man chooses afterwards to use the gymnasium, to run, to row, to play ball, or to saw wood, for the purpose of improving his physical condition, matters little, provided he accomplishes that object.

"The modern gymnasium, however, offers facilities for building up the body that are not excelled by any other system of exercise. The introduction of the new developing appliances has opened up the possibility of the gymnasium to thousands to whom it was formerly an institution of doubtful value. The student is no longer compelled to compete with others in the performance of feats that are distasteful to him. He can now compete with himself—that is, with his own physical condition—from week to week, and from month to month. If he is not strong enough to lift his own weight, the apparatus can be adjusted to a weight he can lift. If he is weak in the chest or the back, he can spend his time and energy in strengthening those parts without fear of strain or injury.

"In fact, he can work for an hour, going from one piece of apparatus to another, keeping always within the circuit of his capacity, and adding slowly and surely to his general strength and powers of endurance. If the heart is weak, the lung-capacity small, the liver sluggish, the circulation feeble, or the nervous system impaired, etc., special forms of exercise can be prescribed to meet these conditions.

"Gentle running is usually advised as a constitutional exercise for all those who can take it. This is usually severe enough to start the perspiration and make a bath of some kind desirable. A tepid sponge- or shower-bath is generally advised; and, in my opinion, the bath which regularly follows the exercise at the gymnasium, and the habit of bathing established thereby, are almost as valuable as the exercise itself.

"After a period of six months or more, the student returns again to the director's office, and has another examination, in order to ascertain what improvement he has made, and to receive any new suggestions.

"This, in brief, is the educational part of the system of physical training carried on at the Hemenway Gymnasium."

From the accumulated results thus obtained a greater symmetrical development of all the parts is shown, and under this system not only are greater feats accomplished, but they are performed with greater ease and safety.

This is well exhibited in the tables[1] made out in Harvard and representing the increase in eight years. Whereas 675.2 was the highest number of points, according to the standard of the director of the Hemenway Gymnasium, gained by any man in college in 1879–80, and 632.2 was the average of the ten strongest men in the college in that year, in 1887 there were out of 1077 students attending college, 824 of whom were examined, 94 men with a strength above 675.2, 145 with a strength above 632.2, and the men with a strength above mean (of 400 points) reached the high number of 609. In the same report there were on record the "names of two hundred and forty-five students whose test of general strength (of arms, chest, back, legs, lungs, etc.) surpasses the test of the strongest man in 1880."

TRAINING.

The general principles involved in training are often poorly understood, even by trainers themselves.

As Dr. Parkes has expressed it, "training is simply another word for healthy and vigorous living," a condition which can be attained only by the strictest adherence to a system of diet, the employment of regular and systematic exercise, and the most scrupulous attention to the minutiæ of general and personal hygiene. By attention to these the body is brought into a perfect condition of health, with enlarged and strengthened muscular action, improved circulation, and increased breathing-power.

The most important consideration is *time*. When we consider the increased force of the heart, acting rapidly upon a large volume of blood, the enlarged calibre of the vessels, the expansion of the chest and of the lungs themselves, and the increased bulk and tone of the muscular system, voluntary and involuntary, it becomes obvious that a *long time* is demanded to accomplish these results.

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[1] Report upon Athletics, Harvard College, 1888, p. 36.

It is needless here to dwell upon the necessity for general exercise of the whole body, or to point out that the exercises should not be limited to the particular forms of muscular movement to be finally performed : suffice it to say that the work should be alternated, and that long intervals of rest should succeed periods of activity.

In regard to the diet to be employed in training, many of the old ideas and rules have of late years been much modified.

From what has been already stated under calorification, the work performed by the body demands a constant renewal of fuel, and it remains to discuss what is best suited to supply the most effective force-value.

All foods may be conveniently divided into four classes,—albumens, fats, starches and sugars, and inorganic constituents,—the type, force-value, composition, and chief constituents of which are well exhibited in the following table, quoted from Ralfe :[1]

CLASS.	TYPE.	FORCE-VALUE OF FIFTEEN GRAINS IN THE DRY STATE.	COMPOSITION.	CHIEF CONSTITUENTS OF ARTICLES OF DIET.
i. Albumens.	White of egg.	13,851 foot-pounds.	$C_{72}H_{112}N_{18}O_{23}$.	Flesh (myosin), bread (gluten), cheese (casein).
ii. Fats.	Butter.	27,716 foot-pounds.	$C_{37}H_{104}O_6$.	Fat or meat, milk, butter.
iii. Starches, sugars.	Starch, grape-sugar.	11,720 foot-pounds.	$C_6H_{10}O_5$.	Potatoes (sago), honey, cane-sugar, grape-sugar in beer or wine.
iv. Inorganic constituents.	Water.	Not known.	H_2O.	Common salt (chloride of sodium), bone-earth (phosphate of lime) in milk, bread, and meat, alkaline salts in meat and vegetables.

Liebig's classification into tissue-making and heat-producing foods is excellent : there is, however, no distinct line of demarcation between them, for, although some foods are especially heat-producers, all tissue in its combustion produces heat, and heat-making substances, like inorganic substances and carbo-hydrates, make tissue as well as albuminous ones.

Of the most important chemical elements entering into the composition of the food, carbon and nitrogen, it is significant that nitrogen is contained only in albuminous foods, and further, as this element is present in every tissue which exhibits energy, the food which contains it must be considered as essential. Taken exclusively, however, albuminous foods are highly uneconomical. It is estimated that, in a state of health, one grain of nitrogen is excreted for every fifteen grains of carbon.

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[1] Op. cit., p. 55, Exercise and Training.

"If a man should confine himself to vegetable food,—say bread, for example,—for every grain of nitrogen that he ate he would take in not only fifteen grains of carbon that are necessary, but fifteen grains too much; in eating meat alone, while he obtains the one grain of nitrogen, he gets only about three grains of carbon,—twelve grains too little. He must eat, therefore, nearly five times as much meat as is necessary, so far as the amount of nitrogen is concerned, in order to get the fifteen grains of carbon, and in so doing he loads his system with five times too much nitrogen. In eating bread you get twice as much carbon as is needed, and in eating meat four times too little. In diminishing the amount of bread you get too little nitrogen, and in increasing the amount of meat too much. If, however, the bread and meat are taken together in proper proportions, we will get, according to the above calculation, the ratio of sixteen of carbon to one of nitrogen in the excretions, which differs but little from that actually found, one to fifteen, and which can be accounted for by remembering that the food of man consists not only of bread and meat, but of other substances containing carbon and nitrogen."[1] Or, as Prof. Huxley has aptly said,[2] "a man confined to a purely albuminous diet must eat a prodigious quantity of it; this not only involves a great amount of physiological labor in comminuting the food, and a great expenditure of power and time in dissolving and absorbing it, but throws a great quantity of wholly profitless labor on those organs which have to get rid of the nitrogenous matter, of which three-fourths is superfluous."

Carbon is the essential element of force, and the fatty principles of food yield in their combustion double the force-value of an equal quantity of albumen or starch, for the carbon is stored up in fat to the amount of eighty per cent., while in albuminous matter and starch there are but fifty-three per cent. and forty per cent. respectively. The carbon thus stored up in the fat of the body also possesses this advantage, that it is always ready for immediate use. On the one hand, man cannot live in good health without fat, and, on the other hand, he can live but a short time upon fat alone. The physiological effect of a carbo-hydrate diet—converted starch and sugar—does not differ essentially from that of a fatty diet, except that the components of the former are more readily oxidized, seventeen parts of sugar being equivalent to ten parts of fat, and that in some occult manner they play an important part in promoting nutrition.

The importance of the inorganic constituents—water, sodium chloride, phosphate of lime, and the alkaloid salts of sodium and potassium—is so well understood that it is only necessary here to refer to them.

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[1] Chapman, Treatise on Human Physiology, p. 39.

[2] Lessons in Elementary Physiology.

VARIETY AND QUANTITY OF FOOD.—The anatomical construction of the alimentary tract (especially the diminished posterior molars and the modified canal midway between that of the herbivora and that of the carnivora) and the analysis of the excreta indicate, what experience proves, that a mixed diet best meets the requirements of man.

The quantity of food that a man *should* eat to live is very different from what he *can* eat and live. In this connection it may be mentioned that, according to Sir Edward Parry, a young Esquimau is said to have eaten thirty-five pounds of food in twenty-four hours; and "Captain Cochrane, on the authority of the Russian admiral Saritcheff, tells how one of the Yakuts consumed the hind quarter of a large ox in twenty-four hours, together with twenty pounds of fat and a proportionate quantity of melted butter. As the man had already gorged himself in this disgusting fashion, it hardly seemed possible that he would be able to consume any more; but the worthy Russian admiral, to test him, gave the savage a thick porridge of rice boiled with three pounds of butter, weighing together twenty-eight pounds. The glutton sat down to this abundant banquet, although he had just partaken of breakfast, and, without stirring from the spot, or showing any sign of inconvenience, got through the whole."

On the other hand, Cornaro is reported to have lived for forty-eight years on only twelve ounces of vegetable matter and fourteen ounces of light wine daily; and Thomas Wood for eighteen years lived on a daily allowance of sixteen ounces of flour made into a pudding with water.

These represent the extremes, and it remains to estimate the amount of food required by the average man in twenty-four hours.

From the estimates of Prof. Dalton, "the entire quantity of food required during twenty-four hours by a man in full health and taking free exercise in the open air is as follows:

Meat	453	grammes	(16 ounces)
Bread	540	"	(19 ")
Butter or fat	100	"	(3½ ")
Water	1530	"	(52 ")

"That is to say, rather less than two and a half pounds of solid food and rather more than three pints of liquid." So there must be added from time to time fresh vegetables, fruit, milk, tea, coffee, and sugar, to contribute variety and maintain health.

From the exact data furnished by modern physiological research elaborate dietaries have been constructed, exhibiting the amount and variety of food required for youths in training; but such tables for physical use are unnecessary, and Dr. J. William White has expressed the whole subject "in a nutshell" in his admirable article on "Exercise and Athletics."[1]

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[1] A Physician's View of Exercise and Athletics, Lippincott's Magazine, June, 1887, p. 1024.

"The diet," he says, "should be plain and sensible, and should not contain an excess of either animal or vegetable food. An ordinary farm-house table, with its mid-day dinner and early tea, will rarely (with the exception of coffee, hot cakes, pastry, and fried meats) offer anything which should be excluded from rational training diet as it is at present understood."

The amount of fluid required in twenty-four hours in training is about five pints in winter and six pints in summer, a considerable portion of which (about one and three-fourths pints) will be contained in the food taken. Water should not be drunk shortly before taking exercise, or large quantities during or immediately after meals; nor should a dry, parched condition of the mouth and throat be mistaken for genuine thirst. This should be first relieved by rinsing the mouth and holding water in it for a short time, and then the actual need for fluid in the system may be supplied by frequent small draughts at short intervals. During training, tobacco, and especially cigarettes, being a depressant upon the heart, should be positively forbidden.

Alcohol, if allowed at all, should be used in the greatest moderation, and be limited to light table ales, light beer, or red wine. The reaction from the great restrictions of the past has led to too great laxity in the use of alcoholic beverages during training.

As pointed out by Dr. Parkes, "a small quantity of alcohol does not seem to produce much effect, but more than two fluidounces manifestly lessens the power of sustained and strong muscular work. In the case of a man on whom I experimented, four fluidounces of brandy (= 1.8 fluidounces of absolute alcohol) did not apparently affect labor, though I cannot affirm it did not do so; but four ounces more, given after four hours, when there must have been some elimination, lessened muscular force; and a third four ounces, given four hours afterwards, entirely destroyed the power of work. The reason was twofold. There was, in the first place, *narcosis*, blunting of the nervous system,—the will did not properly send its commands to the muscles, or the muscles did not respond to the will; and, secondly, the action of the heart was too much increased, and induced palpitation and breathlessness, which put a stop to labor. The inferences were that *any amount* of alcohol, though it did not produce narcosis, would act injuriously by increasing unnecessarily the action of the heart, which labor alone had sufficiently augmented. I believe these experiments are in accord with common experience, which shows that men engaged in any hard labor, as iron-puddlers, glass-blowers, navvies on piece-work, and prize-fighters during training, do their work more easily without alcohol."

BATHING.—For its physiologically stimulating effect upon the nervous and circulatory systems the cold bath is now so generally adopted that it requires simply to be mentioned in this connection. If it be employed immediately after the exercise of the day, it may be omitted on rising or be substituted with advantage by the sponge-bath. In either case it should be followed by vigorous friction with a coarse towel. Occasionally in addition the warm bath with an abundance of pure soap should be employed, to cleanse the skin thoroughly, and this will best be taken before retiring at night. This will regulate the action of the skin, and diminish, if not entirely remove, the necessity for sweating as formerly employed.

The bowels should be regulated, and constipation, often a serious annoyance, carefully guarded against. For this purpose some of the milder laxatives and salines should be prescribed, preferably the natural mineral waters.

If during training nervousness, sleeplessness, inattention, loss of appetite, diminution in weight, exhibit a tendency to "training off," a little extra attention to the minutiæ of training will correct them; but, if the heart becomes intermittent or irritable, and breathlessness ensue, a physician should at once be consulted, to ascertain if any pathological cause exist.

The amount of work daily performed by a crew in training will depend much upon the ultimate exertion to be undertaken, for the crew that has but a half-mile or mile race will not need the severe training required for a three- or four-mile race. In the selection of the crew attention must be paid to the muscular development and the breathing-power, particularly to the latter, since the severest strain at the most important period of the contest will be thrown upon the heart and lungs.

The period of actual training includes from three to six weeks, and during this time the following schedule may be taken as an example of a single day's work.

Rising at six in summer and seven in winter, the cold bath is immediately taken. After dressing slowly, a brisk walk or run is taken about the track, to develop the "wind." Breakfast is served at eight, and consists of an egg, one or two chops or a piece of steak and greens, with bread and butter and a pint of milk. From breakfast till noon is occupied with the usual college duties, an hour being taken, if possible, for some light gymnasium work, the exercises at this time being particularly directed to those parts which are not employed in the more important exercise of the day, thus fulfilling the rule already quoted of exercising all the muscles.

A light lunch of cold meat, bread and butter, with milk, water, or tea, is taken, and an hour and a half or two hours later the principal exercise of the day begins. For the boat crew this will consist of from one and a half to two and a half hours at the machines or on the river, at one-half, three-fourths, or full speed, or alternating, at the pleasure of the trainer.

On returning to the house the men are immediately sponged down and rubbed down briskly, after which a short rest precedes the principal meal of the day. One cold bath a day is sufficient. This meal or dinner, as pointed out before, will exclude few articles found upon the ordinary farmhouse dinner-table (except hot cakes, coffee, fried meats, pastry, etc.), and should be heartily partaken of. At ten o'clock a light tea of crackers and milk or thin oatmeal gruel may be taken, and at eleven o'clock they retire for the night.

If the principal exercise of the day be taken in the morning, a mid-day dinner from twelve to half-past one should follow it, with the interval of rest between.

The results of systematic exercise engaged in for only a limited time are remarkable, as evidenced by the following table, showing the effect of four months and twelve days' exercise, under Maclaren's system, on fifteen youths ranging from sixteen to nineteen years of age.

No.	Age.	Measurements, etc.							Increase.				
		Height.		Weight.		Chest.	Forearm.	Upper Arm.	Height.	Weight.	Chest.	Forearm.	Upper Arm.
		Ft.	In.	St.	Lb.	In.	In.	In.	In.	Lbs.	In.	In.	In.
1	18	5	1¼	7	8	29½	9½	8¾	1	. .	½	. .	½
		5	2¼	7	8	30	9½	9½					
2	19	5	8½	9	5½	28	11	10¼	¼	5½	3½	. .	1⅛
		5	8¾	9	11	31½	11	11⅜					
3	17	5	5¾	9	1	26½	10⅜	8½	⅜	. .	3	. .	1½
		5	6⅛	9	1	29½		10					
4	18	5	8¼	10	0	33	10¾	10¼	¼	3	2	. .	1¼
		5	8½	10	3	35	10¾	11½					
5	18	6	0½	10	13	32	10½	9¼	¾	3	2	. .	1⅝
		6	1¼	11	2	34	10½	10⅞					
6	17	5	3½	8	1	31	10⅛	9⅞	1	6	2	. .	1⅛
		5	4½	8	7	33	10⅛	11					
7	18	5	5¼	7	13	26	9¼	7⅞	½	3	3	¼	1⅝
		5	5¾	8	2	29	9½	9½					
8	16	5	6¾	8	3	28½	9	8½	½	1	2½	⅛	1
		5	7¼	8	4	31	9⅛	9½					
9	17	5	8⅜	11	3	31	11¼	10¼	¾	1	2	⅛	⅞
		5	9⅛	11	3	33	11¼	11⅛					
10	18	5	11⅝	11	8	30	10¼	10½	¾	1	3	½	½
		5	11⅝	11	8	33	10¾	11					
11	19	5	7¾	10	2	33	10½	10¼	⅞	1	1½	½	⅝
		5	8⅜	10	2	34½	10½	10⅞					
12	18	5	10½	10	11	32	10½	10	1⅜	1	1½	½	1
		5	11⅞	10	11	33½	10½	11					
13	19	5	7⅞	11	13	33	11½	12	1¾	1	2½	½	½
		5	9⅝	11	13	35½	11½	12½					
14	17	5	6¾	9	13	29	10⅝	8¼	⅞	4	3	½	1¼
		5	7⅝	10	3	32	10⅝	9½					
15	19	5	10½	10	1	27½	10⅝	10⅞	1⅜	8	5¼	½	1¼
		5	11⅞	10	9	32¾	10⅝	10⅞					

EXERCISE FOR GIRLS.

At the present time, when women are striving to engage in so many of man's burdens and responsibilities, and are even desirous of competing with him in the cares and duties of professional life, the subject of exercise for girls assumes a new and greater importance, and the physical training of girls, the co-education of the sexes, and employment for young women appeal to every one interested in children and their development.

During early childhood boys and girls are very much the same. They walk, talk, romp and play, love and hate, with an innocent *abandon* ignorant of sex. But to the observing the difference even here is apparent. The infant Ulysses breaks the thin disguise of gown and sleeves, dropping the distaff to grasp the sword, while the baby Andromache, inspired with the divine instinct of motherhood, scarcely able to creep, caresses the diminutive image of herself. With the advent of puberty the sexes diverge. The unmistakable difference of face and feature, form and limb, denotes a change of mind and matter, and reveals the demand for a special training. From the time of Hippocrates woman has been described physiologically as enjoying a tripartite life, the divisions being marked by the advent and disappearance of the catamenial function. The lines of separation, diverging as childhood recedes, again unite as old age approaches; and Shakespeare's description of senility,—

> "Last scene of all,
> That ends this strange eventful history,
> Is second childishness and mere oblivion,
> Sans teeth, sans eyes, sans taste, sans everything,"—

applies alike to both sexes, and humanity as a sexless being passes the portal of death. With the first and a portion of the second tripartite state of women we are concerned.

Until the age of nine or ten is reached, girls, as a rule, are allowed to exercise and mix on equal terms with their brothers, whom they often excel both in spirit and in skill. From this time on, the decrees of fashion impose a bondage upon the movements of the female, and the decorous girl must abandon her romps and games and be content to confine herself in stays and tight boots and exercise the regulation walk. At this period systematic physical development should be commenced. There should be in operation in every girls' school, academy, and college a system of physical education similar to that employed in men's colleges, which should first eradicate any special defects and weaknesses, and then create, develop, and maintain the symmetry of parts, gradually increasing the strength and bodily vigor up to maturity. In this connection, as an example of what may be done, a rapid sketch of an hour's exercise, as given by Miss Mary E. Allen, director of the Allen Gymnasium, Boston, may be cited:[1] "Putting each pupil into an absolutely unfettered costume, we begin the hour with a series of free movements, without apparatus, which exercises certain sets of muscles from head to foot in regular succession, the object being gently to accelerate the circulation and to limber the joints. Following this will come, perhaps, an exercise on the breast-bars, upright bars about eighteen inches apart, which gives a little harder work, but which concerns only localized parts of the body. By this time the muscles are sufficiently warmed to bear more strain, and a hard pull at the chest-weights exercises from head to foot, though the trunk and arms get the main share. Following this, leg-development is in order, and a jump over a light rod which is displaced if a trip occurs, with a landing upon a very soft mat, fills the demand; by this time the respiratory muscles are ready for enforced work, and a set of deep breathing-exercises, gently and gradually increased in force, oxygenizes most fully the blood as it flows to the lungs, strengthens them, and furnishes strong action to various respiratory muscles, as the diaphragm, intercostal, and abdominal muscles, with stimulation to the organs situated in juxtaposition, as the stomach, liver, etc. These breathing-movements, of which I make large use, they being a prominent feature of our work, are taken by the class lying down, and thus relief is afforded to the spine and brain. In addition, various other exercises are given in the recumbent position, to strengthen the back and lateral muscles. Now a wooden-dumb-bell drill gives stronger all-over work than the free movements, and a run up the ladders and round the running track again carries the work into the leg muscles, while the thorax receives its share of gain in increased respiration. Indeed, some authors ascribe stronger development to the thorax by leg exercises than by those of the arms. A complete rest is now in order, and the whole class, donning their wraps to prevent any liability to chill, stretch for a few minutes upon the mats. The attraction of the vaulting bar is next presented, where the exercise gives courage, elasticity, and agility. Then, perhaps, an iron-dumb-bell drill, adding still stronger all-over training than the wooden-bell drill, is succeeded by a brisk, lively, competitive game of bean-bags, which induces profuse perspiration, after which the class is drawn into marching order and instruction in carriage and gait is given by a series of marching, hopping, and skipping movements, and the class is dismissed, exhilarated, buoyant, and hot, to its refreshing bath and fresh clothes.

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[1] American Association for the Advancement of Physical Education, 1890.

"Thus you notice that no one set of movements is continued for longer than six or eight minutes. So the mind is kept healthily occupied by diversity of work, and a large number of muscles are gently exercised, insuring symmetry of development, and much more exercise is accomplished than by the use of harder movements on a few machines. In class work, every exercise possible is performed to the accompaniment of music, in which I most heartily believe.

"As the years increase, the body demands harder work, and exercises above the floor on high parallel bars, where the weight is held by hands over the head; low parallels, where the weight is suspended or held by the shoulders; travelling rings, where the weight is held by one hand; climbing and shinning exercises,—are added, according to development."

In addition to this, out-door games should be a regular part of the curriculum, and swimming should be regularly taught, not alone for the protection it affords, but also for its excellence as an exercise bringing into play all the muscles of the body. Another useful exercise is rowing, which should be engaged in whenever the opportunity offers, strengthening and developing nearly all the muscles of the body. Equestrian exercise is also excellent if used in moderation, especially until the full growth is attained. The recognition of the importance of exercise for girls has of late years become so general that little need be added. The great difficulty in providing suitable places for ladies to exercise is the expense; but this is being solved, in the larger cities at least, by the establishment of ladies' clubs, and the unions formed in connection with men's athletic associations.

TABLE

Showing Average Heights and Weights of Boston School-Girls, irrespective of Nationality.[1]

AGE.	OCCUPATION OF PARENTS.					
	NON-LABORING.			LABORING.		
	No. of Observations.	Height, Inches.	Weight, Pounds.	No. of Observations.	Height, Inches.	Weight, Pounds.
5 years	120	41.66	40.55	491	41.26	39.48
6 "	172	44.12	44.14	809	43.24	43.13
7 "	247	45.71	48.02	921	45.41	47.16
8 "	297	47.92	52.79	982	47.47	51.81
9 "	224	50.16	58.78	913	49.27	56.74
10 "	232	51.66	63.76	854	51.25	61.98
11 "	210	53.66	70.49	719	53.41	68.01
12 "	237	56 16	80.18	671	55.70	77.52
13 "	191	58.67	90 68	593	58.01	87.88
14 "	226	60.28	99.40	419	59.84	97.92
15 "	168	61.19	107.70	258	61.00	105.11
16 "	147	61.46	111.22	169	61.55	112.59
17 "	98	61.88	115.15	89	61.92	115.72
18 "	77	62.26	115.83	46	61.70	112.94

According to the tables of Dr. Bowditch made from measurements of Boston school-girls from five to eighteen, irrespective of nationality, there is in children of the non-laboring class an increase in height from five to thirteen years from 41.66 inches to 58.67 inches, while the weight is nearly doubled. From thirteen to fourteen the increase is 1.61 inches, from fourteen to fifteen it is less than one inch (.91), from fifteen to sixteen a little over one-fourth of an inch (.27), from sixteen to seventeen less than half an inch (.42), and from seventeen to eighteen less still (.38). These figures are for the most part greater than in the children of the laboring class, and both tables show a steadier and more regular increase and one extending over a longer period than in boys of the same age. The importance of these deductions is twofold,—showing that girls require throughout a longer period the utmost care that they are not overtaxed, and also that during the later part of this period they should not be subjected to excessive study, but should be permitted to perfect their development.

If we compare the average girl of to-day with the ideals beheld in the pictures and statues of the past,—the well-developed and shapely arm and shoulder, the high chest, the vigorous body, and the firm and erect carriage of the Minervas and Niobes, Venuses and Junos, of mythology, the Helens, the Madonnas, and the mediæval beauties,—there is at once a consciousness that something radically wrong exists. Instead of this galaxy of beautiful parts, a vision of pipe-stem arms, scrawny necks, angular shoulders, flat chests, narrow backs, stooping carriage, and weak walk, recalls us to the realities of the present. If we further compare the brilliant eye, the damask cheek, and the luxuriant form of the robust English damsel, the sturdy German fräulein, or the hardy Provençal maiden, with the vacant gaze, pallid features, and attenuated figures of the fragile, easily-fatigued, languid girls, the products of modern American habits and customs, the contrast is equally striking, showing that there is a conservation of force on the part of the trans-Atlantic female, and a deterioration of force on the part of her cis-Atlantic civilized sister.

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[1] Bowditch.

As pointed out in the first section of this article, the statues of the goddesses of the Greeks were taken from models who from attention to physical culture were ideals of health and symmetry; and the moral is evident that the errors which exist in our present system of female education are the lack of proper physical exercise, and a disregard for the obligations which sex imposes upon the developing females.

But there is one fact that is constantly brought to the notice of the physician, and it is his duty to caution against it, and that is that girls who become enthusiastic in any form of exercise are apt to disregard totally the catamenial function. Cases have been known where champion matches have taken place during the menstruation of one of the players. The strain and over-exertion incident to the contest would certainly have a baneful influence on the sexual organs at this time. The same must be said of fatiguing horseback-rides, long drives with exposure, mountain-climbing, swimming, and the like. The injury done under such circumstances is illustrated in the note-books of gynæcologists.

Remarkable degenerative changes have occurred during the developmental period, the most important era in a woman's existence. There has been a disregard of the four conditions considered requisite by Clarke[1] for the proper education of the female: "first, a sufficient supply of appropriate nutriment; secondly, a normal management of the catamenial functions, including the building of the reproductive apparatus; thirdly, mental and physical work so apportioned that repair shall exceed waste, and a margin be left for general and sexual development; and, fourthly, sufficient sleep."

Then, again, dress has had a share in producing these changes. The gifted authoress of "The Gates Ajar" and her followers, in their explanation and advocacy of a new clothes-philosophy, have done much to emancipate women from "corsets that embrace the waist with a tighter and steadier grip than any lover's arm, and skirts that weight the hips with heavier than maternal burdens."

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[1] Clarke, Co-Education, p. 60.

GYMNASTICS.

The traditional history of the Chinese, the superstitious religious practices of the ancient Indians, the earliest mythological fables of the Egyptians and Greeks, all contain references to the employment of exercises for the restoration and preservation of the health and the improvement of all the faculties.

Pliny tells us that Asclepiades, who lived in the second century B.C., cured all diseases by physical exercises alone, and justified his declared willingness to forfeit all claims to the title of physician should he ever fall ill except from accident or senility, by living for more than a century and dying eventually from the effect of an accident. The relation which was thus early established, both in history and in tradition, between physical exercise and health and longevity became later, as among the early Chinese, the vital principle of civilization of all the more powerful nations of the world. In Greece, and especially in Sparta, the idea of personal hygiene, or physical culture as we now call it, overtopped every other, and resulted in a type of physical perfection which has never since been equalled. The Romans, eminently a warlike people, appreciated the value of exercises only so far as they promoted the physical force for military purposes.

With the increase of luxury and vice, the gymnasia declined in reputation and favor, and, when Greece and Rome began their uninterrupted descent in the scale of nations, gymnastic and athletic proficiency likewise declined, to become in the Middle Ages almost the exclusive property of the nobility and professional soldiery. During all this period there existed but a vague and empirical estimate of the true value of movements, exercise, gymnastics, etc., and it was not until the beginning of the present century that gymnastics became systematized and popularized. In 1811 Jahn established in Berlin his gymnasium or Turnplatz, introduced new apparatus, improved the defective system, published his celebrated essay on the principles of gymnastics, and established the *Turnvereine.* The success of these and the energetic aids of Guts-Muths and Spiess led to the establishment of similar societies in Switzerland, Sweden, and France, and later in England and in our own country. It remained, however, for Peter Henry Ling, a native of Sweden, in 1816, with a remarkable intuitive grasp to collect and arrange the scattered fragments and shifting facts, to separate the real and rational from the superstitious and empirical, and to create with the aid of anatomy and physiology, upon a philosophical and scientific basis, a system of movements and exercises for the development and perfection of the whole body. It is a little over fifty years since Ling departed, and still, as Dr. Richter, the great physician of Hanover, said, "his principles are incontestable." He had learned, in a life of vicissitudes and excitement as a fatherless boy, a theological student, a tutor, a naval volunteer, and a fencing-master, the value of a sound body; and, as a leading French authority has said, "if he was not the creator of the modern, scientific gymnasium, he was its regenerator."

What is implied by exercise has already been described. Movements include more: they are motions of specific kinds, having specific effects, employed for specific purposes, and practised to secure definite results.

As pointed out by Taylor, "movements are mechanical agencies, directed either upon the whole system or a part of it, for the purpose of inducing determinate effects upon its vital actions, and generally having reference to its pathological state."

Movements.—Movements are conveniently divided, according to the source from which the moving power is derived, into active and passive. They are also said to be single when but a single person is engaged in their execution, and duplicated when more than one is engaged. All the movements to be described in the first part of this section are single, but in the second part they are of the second variety. These second or duplicated medical movements are also of two varieties, of each of which examples are given. In one the patient, quite passive, receives the motion of some particular variety given by the physician or operator; in the other the patient is required to bring into action some particular part, the quality, amount, and duration being entirely controlled by the physician or gymnast.[1] The degree and kind of resistance employed by the operator in his or her manipulations require a variety and nicety in the different stages similar to the delicacy of touch employed by musicians for giving expression and effect in instrumental performances, constituting a *tactus eruditus* that cannot be acquired from books.

Movements are also described as *concentric* when the muscular contraction steadily increases, and *eccentric* when the muscle is stretched and its muscular contraction steadily decreases. From the explanation already given of the physiological action of muscular tissue, from the common experiences of every-day life, and from the fact that "fatigue is in proportion to the amount of mental and nervous rather than to the amount of muscular action employed," it is evident that all movements should be slowly performed, from a particular position or base, and the more prolonged the movement the greater the amount of muscular exercise in proportion to the time occupied, especially in the respiratory exercises and the trunk movements generally on account of the vital organs contained.

Swedish Movements.—According to Prof. Ling, gymnastics are divided into four great classes:

1. Pedagogic or school gymnastics (subjective active), in which the person through his own strength exercises and develops the power to control his own body by his own will.

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[1] The term gymnast designates a graduate from the Royal Gymnastic Central Institute, Stockholm, Sweden. We desire here to express our indebtedness to Miss Anna Jonsson, of Philadelphia, who is a graduate of this institution, for much assistance in the preparation of that portion of this article which relates to the Swedish system of gymnastics.

2. Military gymnastics (objective active), in which one person exercises or contends with another outside will, and by his own muscular power or a weapon of some character masters another's will.

3. Medical gymnastics (subjective passive), in which a person endeavors, through certain positions or with the resistance of other persons, to cure or relieve disease produced by a disturbance of the organism.

4. Æsthetic gymnastics (objective passive), in which the person by a motion or gesture endeavors to reflect his thoughts, emotions, feelings, etc.

The movements employed in the Swedish system may be described under the different regions included, as the head and neck, arms, trunk, etc., but are most conveniently classed under the following divisions:

Educational Gymnastics.—1. Fundamental positions. 2. Arch flexions. 3. Heaving movements. 4. Balance movements. 5. Shoulder-blade movements. 6. Abdominal exercises. 7. Lateral trunk movements. 8. Slow leg movements. 9. Jumping and vaulting. 10. Respiratory exercises.

Medico-Gymnastic Exercises.—Respiratory exercises. General health exercises. Exercises for lateral curvature.

The introductory exercises are intended to secure general attention and muscular control, and to correct the general equilibrium and base of support before the more difficult exercises are undertaken. These include the formation of the lines, numbers, changing position, easy feet and leg movements, head movements, certain arm and trunk movements, and marching, all of which require about five or ten minutes. The calisthenics which immediately follow should occupy only from twenty minutes to half an hour.

The arch flexions comprise various forms of backward flexions of the trunk, and are given for the purpose of strengthening the dorsal spine, expanding the lower portion of the chest, and stretching the upper portion of the abdomen.

The heaving movements comprise forms of self-suspension by means of the arms on a horizontal bar or other apparatus, and are given for the purpose of expanding the upper chest and strengthening the arms.

The balance movements are positions taken from a smaller area than that included within the feet in standing, the difficulty being increased by the altitude of the supporting surface and the diminution of the area of support. They are introduced to develop the equipoise of the body and to secure grace and beauty of action.

The shoulder-blade movements are various forms of arm movements intended to correct the position of the shoulder-blades.

The abdominal exercises bring into play chiefly the muscles of the abdomen, and are employed not alone to strengthen the abdominal walls, but also secondarily to affect the digestive organs.

The lateral trunk movements include various forms of sideways bending and twisting of the thorax, and are employed to strengthen the muscles about the waist, but also influence secondarily the organs in this region, especially the digestive.

The jumping and vaulting are employed to promote the general elasticity and grace of the body.

The respiratory exercises counteract the ill effect of the preceding exercises, and are employed to diminish the frequency of the heart-beat and render the breathing less labored.

The proper arrangement of the order of the movements is very important, to enable the pupil or patient to secure uniform and beneficial results. For this purpose Prof. Ling proposes the following order, subject to modification in particular cases:

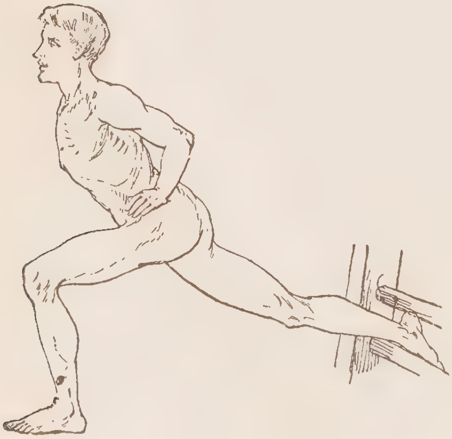
1. A respiratory movement.
2. A movement of the lower extremities.
3. A movement of the upper extremities.
4. A movement of the abdomen.
5. A movement of the trunk.
6. A movement of the lower extremities.
7. A respiratory movement.

The principal base position—standing—is as follows: Heels together, feet at a right angle (ninety degrees) with each other, knees extended, hips extended, rotated outward, and fixed, back muscles extended, scapulæ fixed backward, fingers, forearm, and arm extended, hanging in a position between pronation and supination, neck extended, chin retracted, and the eyes fixed forward and upward.

This position is so particular that its correctness would be destroyed by the lowering of an eyelid.

The Swedish system has divided the principal base positions into five, of which the one above described is the principal one, to which are added the knee standing, lying, half-lying, and hanging. The correct base position must always be assumed before any other position is taken and before any movement may be undertaken from these secondary positions.

FIG. 7.



Standing position.—Fig. 7. Foot grasp, wing forward, falling position.—In this position the left foot is fixed about one and one-half feet from the ground, the left leg being extended. The left leg extended, the back extended, and the head kept forward and upward, the weight of the body is thrown upon the right leg, the knee of which is bent at about a right angle. The person must alternately change the feet, so that the whole body shall be exercised to the same extent.

Fig. 8. Wing balance standing, knee bending and extending.—The difficulty in this movement is to maintain the balance of the trunk when the person rests only upon one foot, while the opposite thigh remains horizontal and the leg is alternately extended and flexed.

FIG. 8.

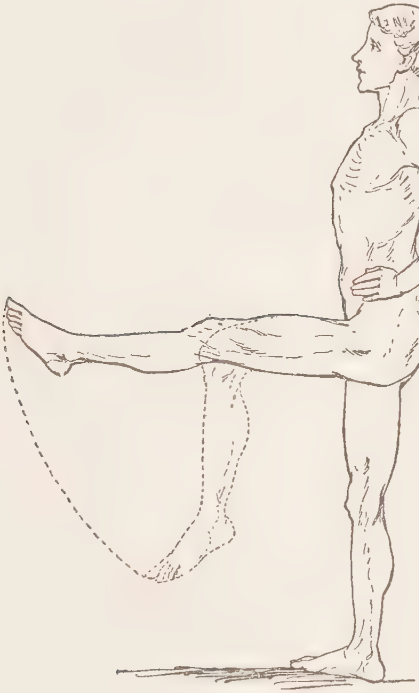


FIG. 9.

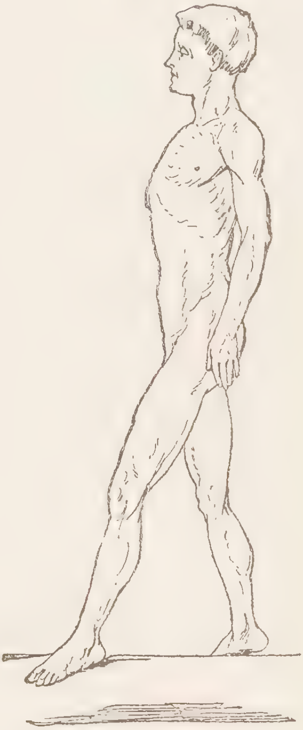


Fig. 9. Balance walking on horizontal bar.—In balance walking the eyes must be fixed forward and upward, the shoulders fixed backward, the chest expanded, the elbows slightly bent. The foot is advanced by slightly flexing the supporting knee, the leg describes a half-circle, and the toes are first applied to the bar, followed by the side of the foot and heel. The balance of the body is further assisted by the elevation and depression of the arms.

Fig. 10. Wing standing, legs lifting sideways.—In this position the weight of the body is thrown upon the resting lower extremity and the same side of the body. The alternate lifting and sinking of the working leg is accomplished by the concentric and eccentric action of the adductors and abductors. This movement may also be considered as a balance movement. The lower extremities are alternately exercised.

Fig. 11. Stretch stride standing, trunk backward bending with support.—By having the arms in stretch position and the support in this movement, the bending backward can be so far performed as highly to

influence the upper portion of the thorax, separate the lower ribs, and extend passively the abdominal muscles.

Fig. 10.

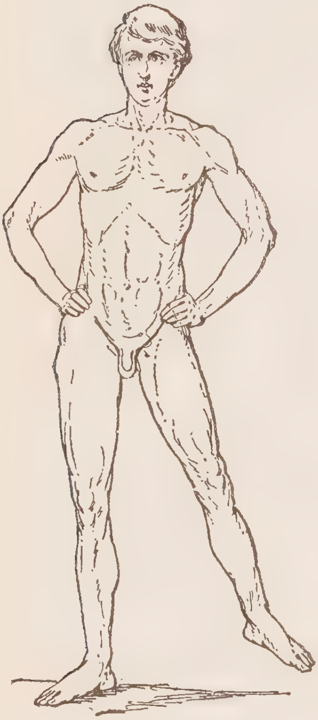


Fig. 11.

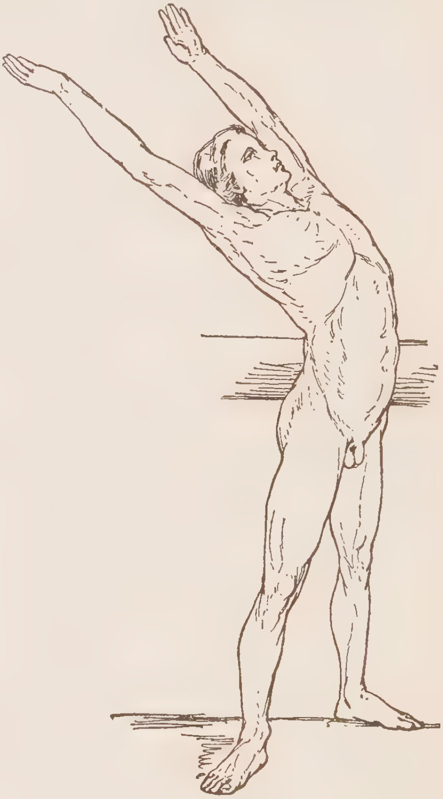


Fig. 12. Arch support standing, knee flexing and extending.—The hands are stretched and fixed to the bars, the body about two feet distant from the wall, so that the trunk and neck describe a curve in which the chest is well expanded. After assuming this position the knees are alternately flexed and extended. This position, as well as all arch standing positions, makes especial pressure upon the circulation in the vessels supplying the spinal column.

Fig. 13. Hanging position. A.—The hands are parted about one and one-half feet and fixed upon the bar, the head upward and backward, chest expanded, shoulder-blades held backward, heels together, feet rotated at ninety degrees, the body hanging on the flexors of the arms, the principal trunk muscles, and the ligaments.

Fig. 14. Underhanging, double arm flexion. B.—In this position the hands are parted as in the preceding position, but are passed beneath the bar and grasp the upper side. Only by gradual exercise is the person able to elevate the entire body by the double arm flexion.

Fig. 15. Bent hanging, double knee extending.—Bent hanging designates a position in which the body is hanging by the hands on the bar, with both knees flexed forward and upward at a right angle. The movement refers to the extension of the legs. The principal effect of this position is upon the abdominal, psoas major, and iliacus muscles, but the movement itself exercises strongly the extensor quadriceps femoris.

Fig. 12.

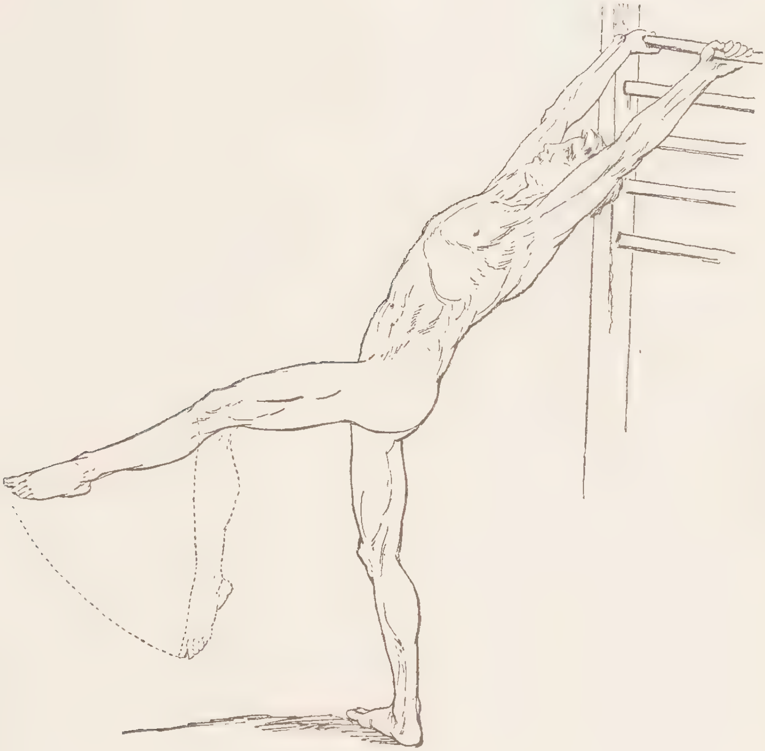


Fig. 16. Bent underhanging, hand walking.—For bent underhanging see Fig. 15. The legs remain in the extended position. The hands are on either side of the bar; the body is slightly elevated and slowly moved backward by the alternate grasping and relaxation of the hands. This movement would be rendered much more difficult by moving the body forward.

Fig. 17. Inclined rope climbing upward.—The hands grasp the rope, one over the other, the highest one being the opposite of the knee that crosses the rope. The walking upward is performed by alternately changing the grip of the hands and alternately swinging one leg and then the other across the rope, the hanging leg being always fully extended. This movement combines the lifting with the trunk change turning movements.

Fig. 18. Yard stride standing, trunk forward bending.—In the yard stride standing position the arms are extended and lifted horizontally sideways, the feet are apart. The motion is made by the flexors of the trunk forward. This movement by taking the yard standing position exercises the shoulder and back muscles (rhomboidei, trapezius, and latissimus dorsi), and it is therefore a valuable exercise in the treatment of lateral curvature.

Fig. 13.

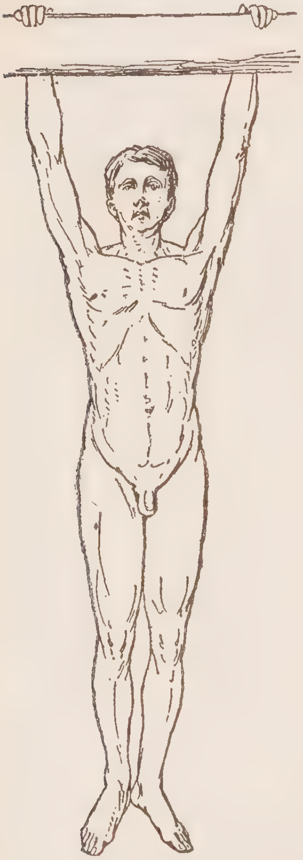


Fig. 14.

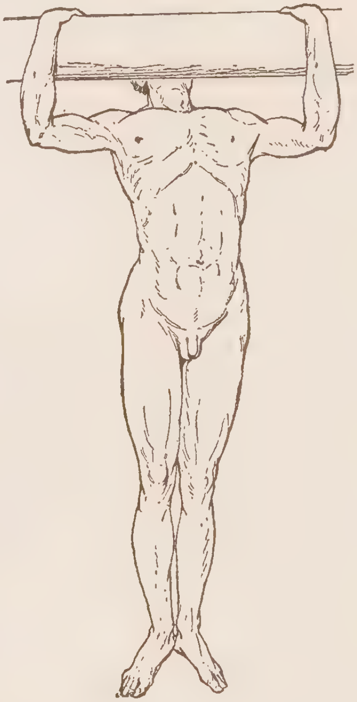


Fig. 19. Yard forward lying, arm bending and stretching sideways.—The heels are held down, and the hips only are supported across a bench, the upper part of the body and the neck being kept in an extended position forward and upward. The movement consists in horizontally flexing and extending the arms while in this position, the action upon the muscles being limited to the shoulder, back, and arms.

Fig. 20. Stretch forward lying, arm bending and stretching upward.—This position differs from the preceding only in the movement of the arms, which are alternately flexed and extended downward and upward, exercising principally the muscles of the upper dorsal region.

Fig. 21. Stretch lying, legs elevating.—The trunk is resting on a low couch, the arms stretched upward parallel and lying free upon the

FIG. 15.

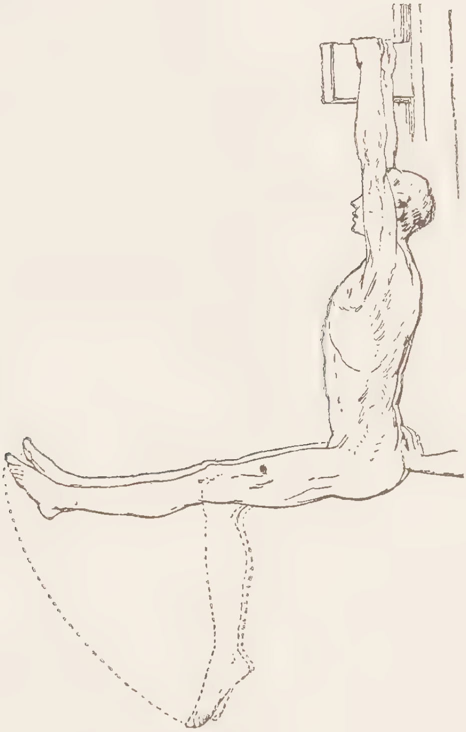


FIG. 16.

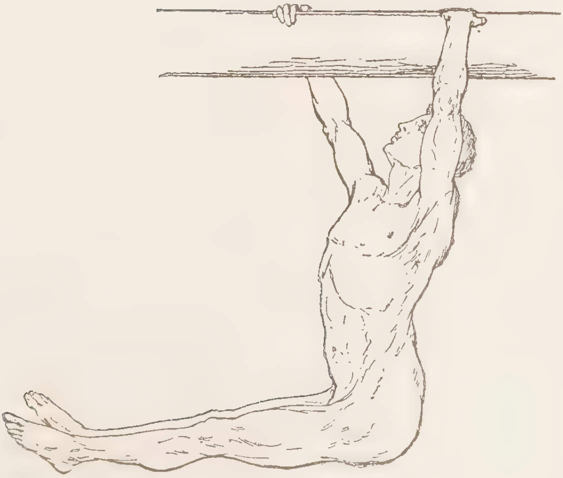


Fig. 17.

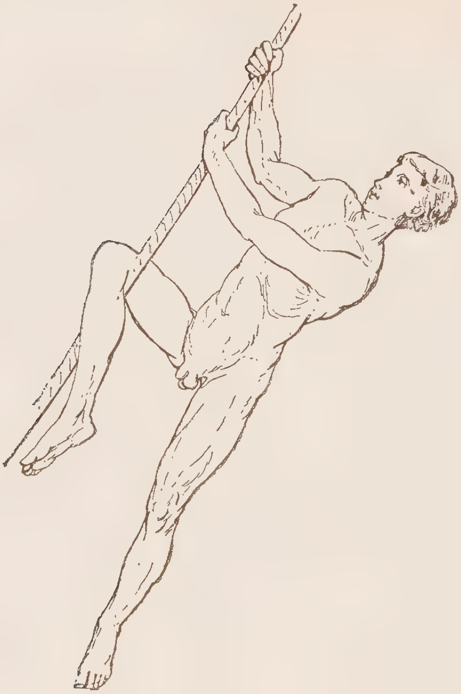


Fig. 18.

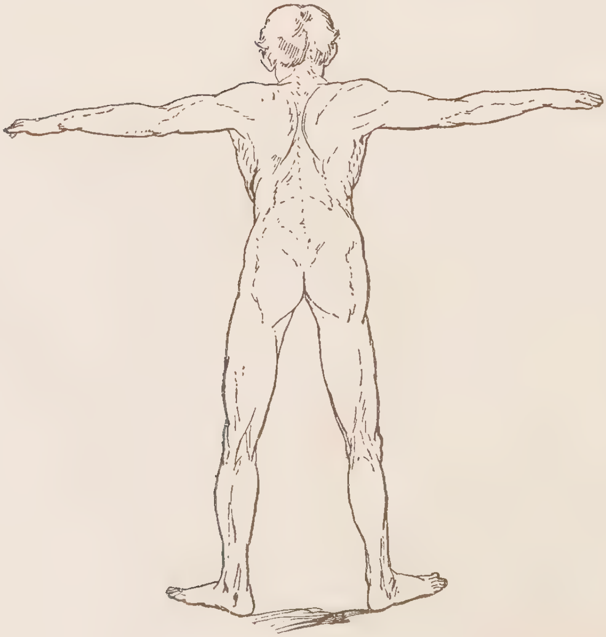


Fig. 19.

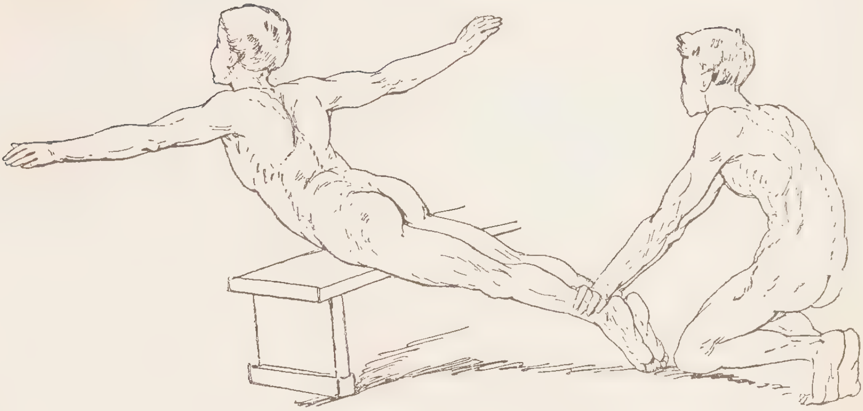


Fig. 20.

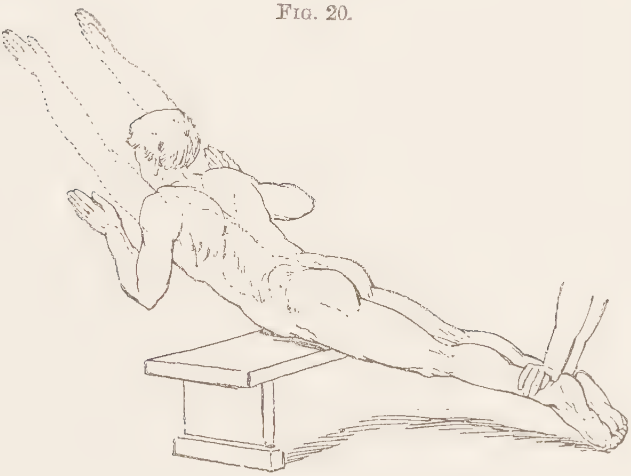
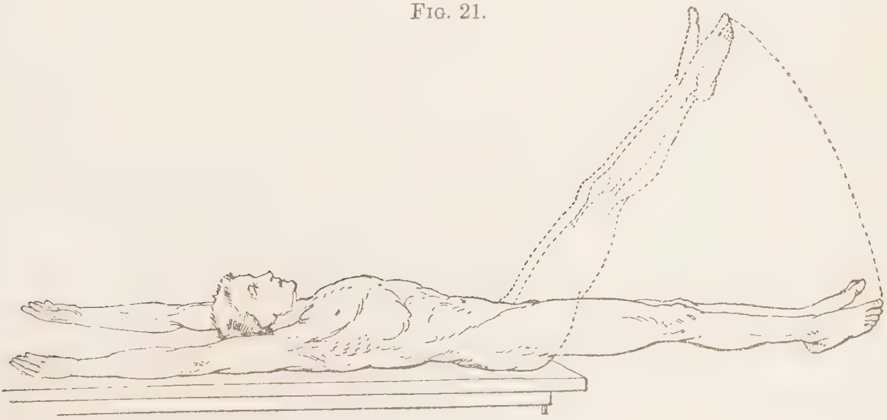


Fig. 21.



couch. The legs are kept in a horizontal position without any support, and from this they are elevated to a vertical position, strongly exercising the abdominal muscles.

Fig. 22. Stretch knee stride standing, trunk backward bending.—In the stretch knee stride standing position the heels are together, the knees separated from fourteen to eighteen inches, the thighs extended, and the arms extended upward and parallel, the hands never being approximated nearer than the distance between the shoulder-joints. In the bending backward the trunk is slowly flexed backward, while the thighs still remain in the extended position, so that, although the muscular action is greatest upon the psoas, iliacus, and abdominal muscles, the flexion is confined entirely to the lumbar region.

Fig. 23. Feet fixed, wing sitting, trunk backward bending.—The feet are fixed under the lowest bar, both entire lower extremities are extended and resting upon the floor. The arms are in the wing position, the back extended, the scapulæ fixed backward, the neck extended, the chin retracted, and the eyes fixed forward and upward. The body is slowly and alternately bent backward, and again elevated to the horizontal. This position is one of the most important abdominal movements, exercising, as it does, the abdominal muscles, and producing a reflex stimulating action upon the abdominal viscera.

Fig. 24. Feet fixed, arms stretch sitting, trunk backward falling.—The feet are fixed under the third bar of the "ribbed chair,"[1] heels together, the toes rotated outward, the lower extremities, trunk, and arms extended, and the arms and trunk maintained in a horizontal position. The effect of this exercise is the same as that of the preceding, except that it is more intense.

Fig. 25. Stoop falling position.—The body is supported by the hands and feet resting upon the floor. The whole body is held rigid, the heels together, and the hands separated the width of the shoulders and pronated and adducted. The body is alternately depressed and elevated by the flexion and extension of the arms. This movement expands and enlarges the thorax, develops the arm muscles, contracts the abdominal muscles, and strengthens the extensors of the legs and feet. It is a combined respiratory and abdominal exercise, and correctly taken it may be considered one of the most stimulating and strengthening movements for the development of the entire body.

Fig. 26. Horizontal stoop falling position.—This is identical with the preceding, except that the feet are elevated and supported in a position horizontal to the body.

Fig. 27. Reverse stoop falling position.—This is the same as the pre-

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[1] The name given to a Swedish apparatus consisting of a number of horizontal bars arranged about eight inches apart, one above the other, from the floor nearly to the ceiling.

Fig. 22.

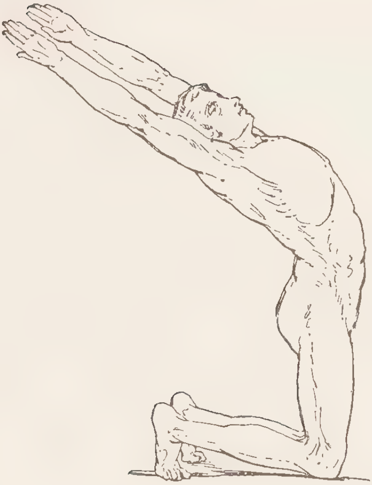


Fig. 23.

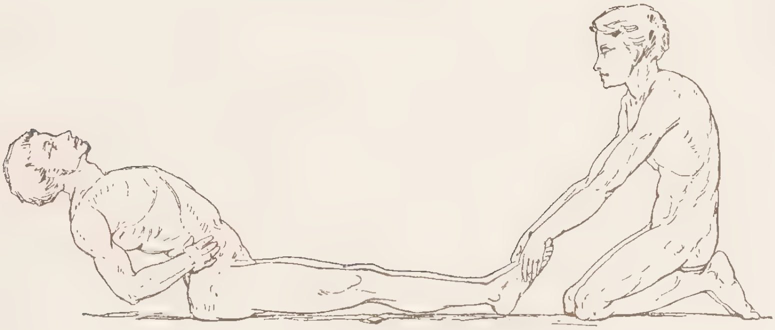


Fig. 24.

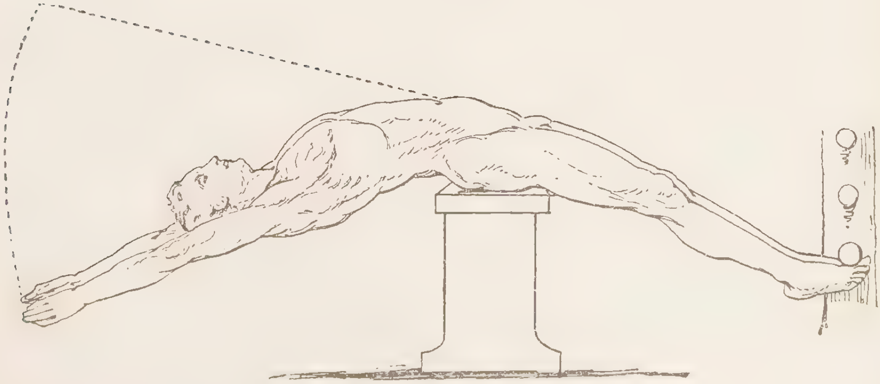


Fig. 25.

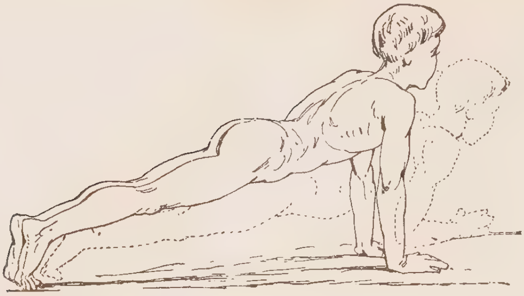


Fig. 26.

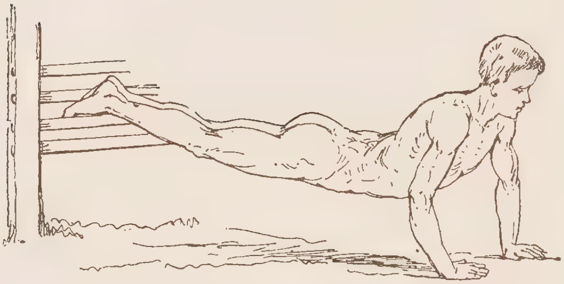
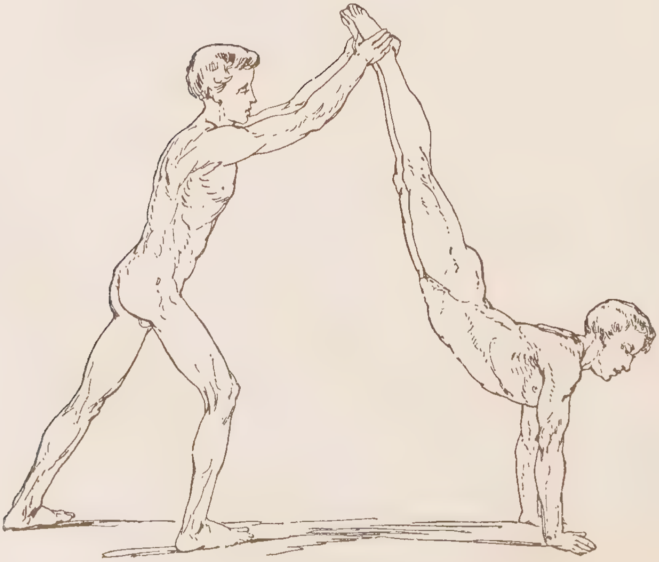


Fig. 27.



ceding exercise, but with the feet elevated above the horizontal. The effect, however, is intensified, because almost the entire weight of the body is supported upon the upper extremities.

Fig. 28. Arms bent, support falling, leg elevating.—The body is supported upon the flexed arms, the hands being fixed against a horizontal bar waist-high. The body is inclined downward and forward, with the heels resting upon the floor. The legs are alternately elevated and depressed.

Fig. 29. Foot fixed, stretch balance standing, trunk sideways bending.—The person standing upon one leg, the other foot is separated about half a yard and fixed at about twelve inches from the floor. The arms are extended parallel, and the trunk is flexed laterally to the side opposite the fixed foot. These flexions are then alternated by changing the feet.

Fig. 30. Arms bent, trunk turn, stride standing, arm extending.—In this position the fingers are close together, the hands slightly flexed, the arms flexed, and the hands fixed to the tip of the shoulder, the chest expanded and the scapulæ retracted, the feet separated about fourteen to eighteen inches to fix the hips, while the upper part of the trunk is rotated a quarter of a circle first to one side and then to the other. The motion is to extend the arms upward while in this rotated position. It may be repeated four to eight times each side.

Fig. 31. Half stretch, half support, side falling, legs elevating.—This position is taken by supporting the entire rigid body with one hand upon the floor, pronated and adducted, and the side of one foot. The other arm is extended upward. The movement consists in elevating the upper leg and then approximating it to the other. This movement is performed from both sides, as it strongly exercises the lateral muscles of the trunk.

Fig. 32. Sideways hanging position.—The hands are separated about three feet and fixed upon the bars so that the lowest one is one yard from the floor. By maintaining this grasp, and by exerting a strong contraction of the upper lateral trunk muscles, the legs are lifted sideways upward to a horizontal position. As soon as the exact position is secured, the feet are slowly lowered to the floor and the movement repeated upon the opposite side.

Ling designates this as the most difficult—the final position—of his system of school gymnastics. It is generally undertaken only by boys, because the relatively heavier lower extremities and weaker lateral trunk muscles of girls render it much more difficult for the latter to perform.

Fig. 33. Jumping in height on the place.—This exercise can be introduced by heels rising and knees bending, in which we have four counts, —heels rise, 1! knee bend, 2! knee extend, 3! heels sink, 4! In the jumping we add one more count,—5! in which the jumping is performed on the third count by a sudden effort of all the extensor muscles of the lower extremities. The body descends upon the tiptoes, heels together, and bent knees, the trunk in upright position. To render the position easier, the arms may be lifted momentarily from the side and depressed in the third count.

Fig. 28.

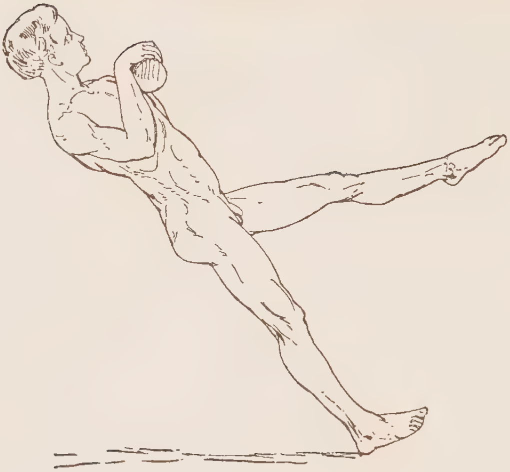


Fig. 29.

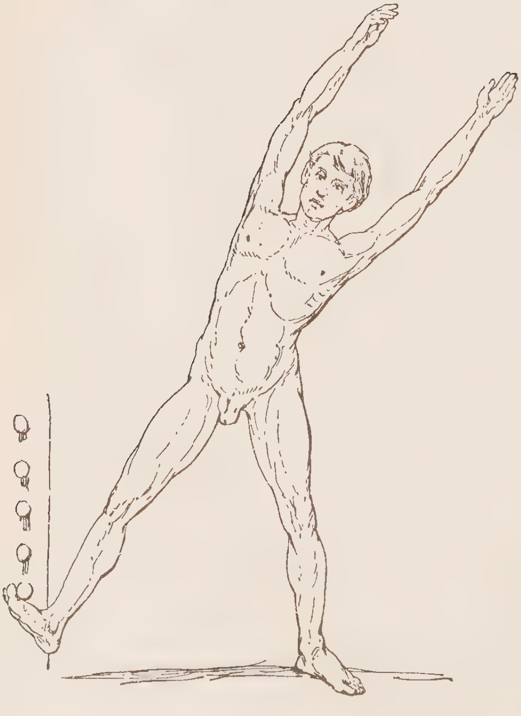


Fig. 30.

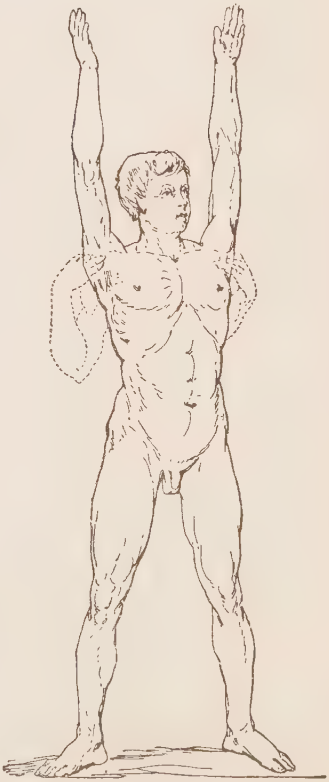


Fig. 34. Running jump.—The running is commenced by one step forward, 1 ! the other foot extended, and the jump accomplished and terminated by bringing the feet together, 2 ! The body will descend in heels rising, knees bending position, in which the trunk will remain upright. Knee extension, 3 ! lowering of the heels, 4 !

Fig. 31.

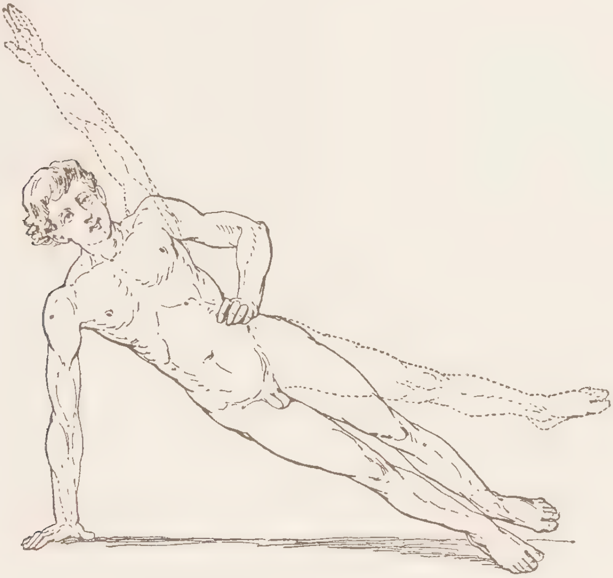
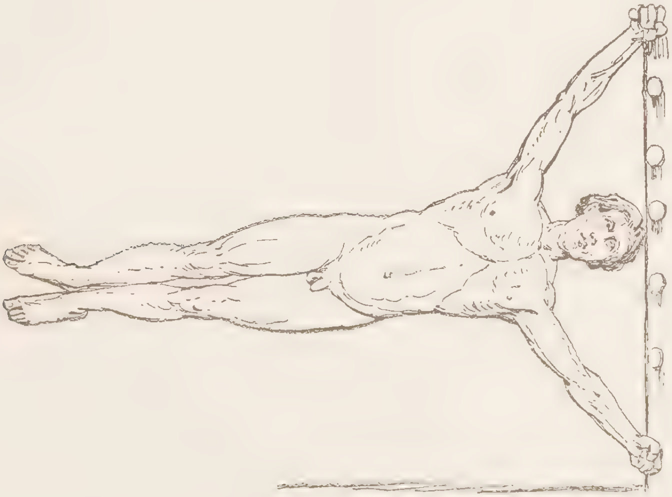


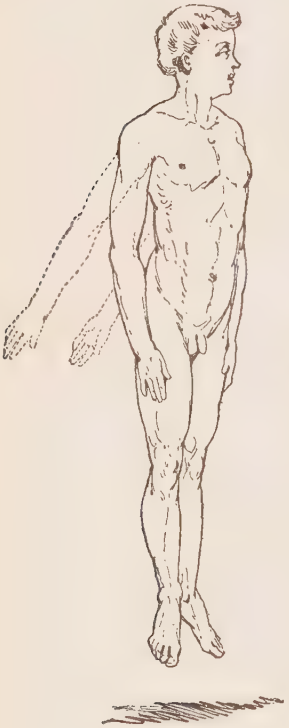
Fig. 32.



These movements may be rendered more complicated by adding more steps, and either allowing the children to count for themselves or omitting the counting altogether.

Fig. 35. High jumping.—The jump is preceded by a short run, to give momentum, and if the bar is high the children may be allowed to have the "spring-board." In jumping over the bar the eyes must be fixed upward, the neck extended, the shoulder-blades back, the arms and trunk extended, and the thighs slightly flexed; the knees must be very strongly flexed, and the heels kept together. The shock of the descent is received upon the extended toes, elevated heels, and flexed knees. This position is maintained for a moment, and the body is elevated by extending the knee, 3! and sinking the heels, 4!

FIG. 33.



This can be made more complicated not only by elevating the bar, but also by increasing the length of the jump. Another modification of high jumping is turning during the jump, so that the body rotates a quarter of a circle or more in its flight. If the high jump is made from a higher to a lower level, this also increases the difficulty.

Fig. 36. Vaulting.—The bar must be fixed about waist-high. The hands are placed upon the upper surface of the bar, 1! A short double spring is taken, and the body is elevated, and rested upon the rigid arms and bar, 2! The hands are changed forward under the bar, 3! The vaulting over the bar is performed by changing the balance of the body by flexing the knees and bending the body forward, so that it is brought to the other side of the bar in the fall hanging position, followed by elevating the body to stretch arch standing position, 4! This position is raised to stretch standing position, 5! and ground standing, arms down position, 6!

The exercise may be rendered more difficult by lowering the bar.

The following eight exercises are selected from among all those included in the systems of Ling, as being ideal positions exercising together all the muscles of the body. Each one taken to its full extent stimulates especially the circulation, respiration, and digestive organs, as well as strengthens the nervous system both directly and reflexly. Taken together they may be considered a system in themselves, since they include exercises for every muscle in the body.

Photo. VI. Neck rest standing, heels rising.—The wing standing, heels rising position is assumed from the first base (standing) position, but the hands are fixed at the hips, the thumbs backward, and the fingers firmly flexed about the waist. The elbows are pointed out sideways backward. In the heels rising position the heels are elevated as high as possible, so that the person stands on the tips of the toes.

Fig. 34.

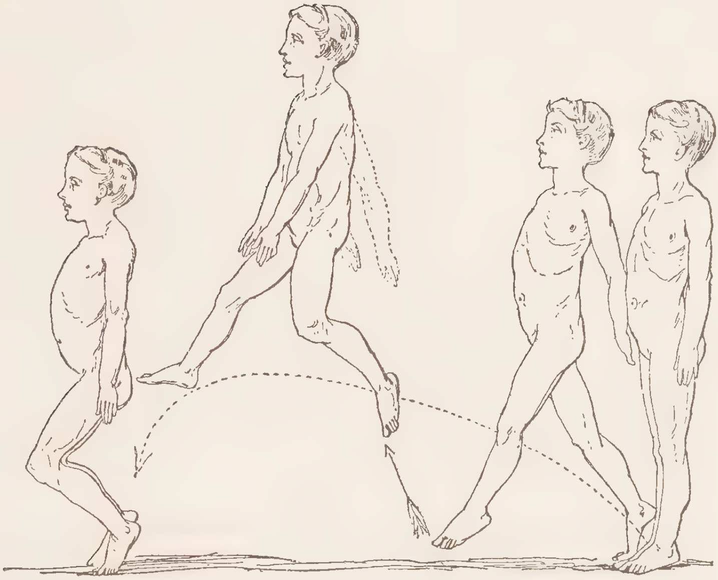
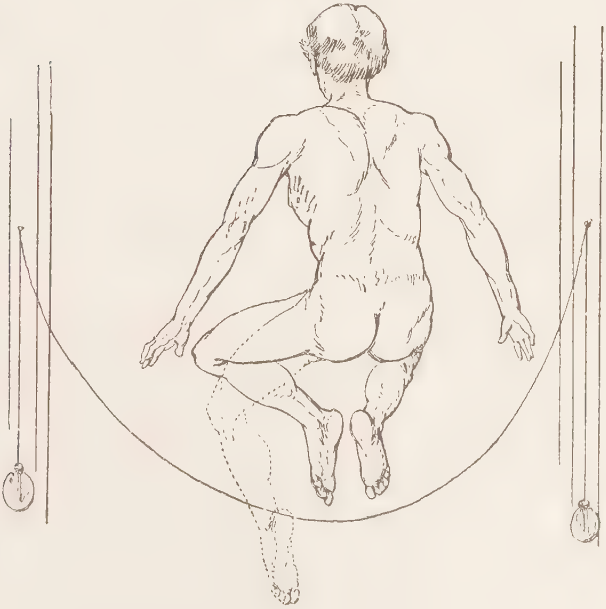
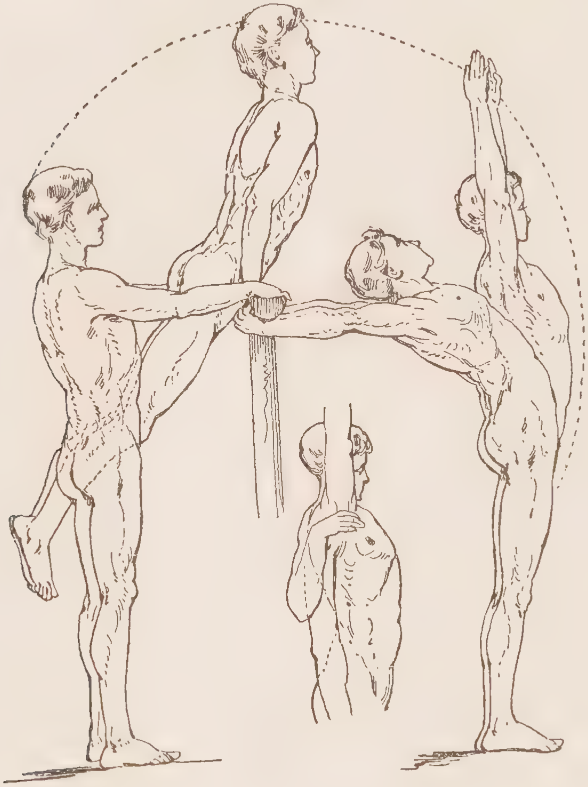


Fig. 35.



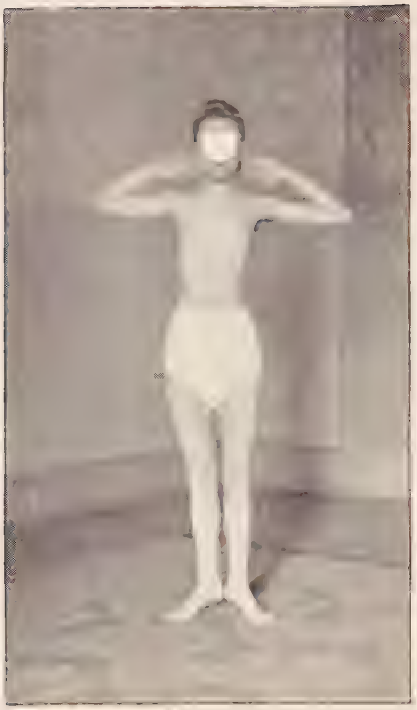
The muscles exercised are the following. Position: wing standing. Muscles: the deltoideus and supra-spinatus lift the arm (upper part) horizontally. Flexion of arm: biceps brachii; brachialis anticus; spinatus longus; pronator radii teres; flexor carpi radialis; flexor ulnaris; palmaris longus; flexor digitorum sublimis. Pronation of hand: pronator radii teres; pronator quadratus; flexor carpi radialis; palmaris longus; flexor digitorum sublimis. Fixation of arms and hands to the hips: pectoralis major; latissimus dorsi; teres major et minor; subscapularis. Heels raising, extension of feet: gastrocnemius; soleus; plantaris; flexor digitorum communis longus; flexor longus pollicis; tibialis posticus; peroneus longus et brevis. Extension of leg: rectus femoris; vastus externus et internus; cruralis. Extension and rotation outward of thigh.—Extensors: gluteus maximus, medius, et minimus (posterior part); obturator internus; gemelli; quadratus femoris; biceps femoris (the long head); semitendinosus; semimembranosus. Rotators outward: adductor longus, brevis, et magnus; gluteus maximus et medius; psoas major; iliacus; pectineus; pyriformis; obturator internus et externus; gemelli; quadratus femoris; biceps.

FIG. 35.



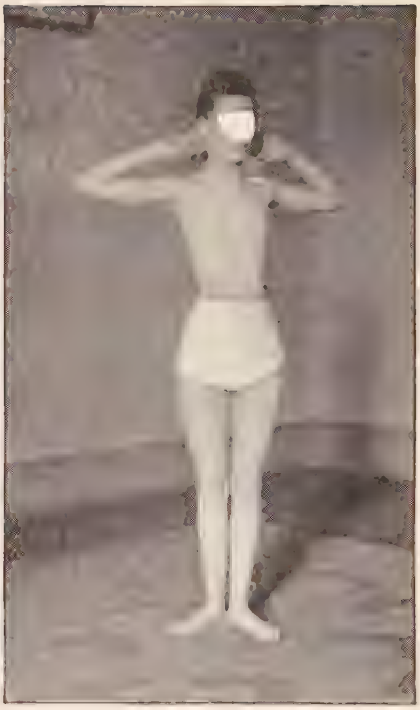
By taking the wing standing position the upper part of the thorax

PHOTO. VI.



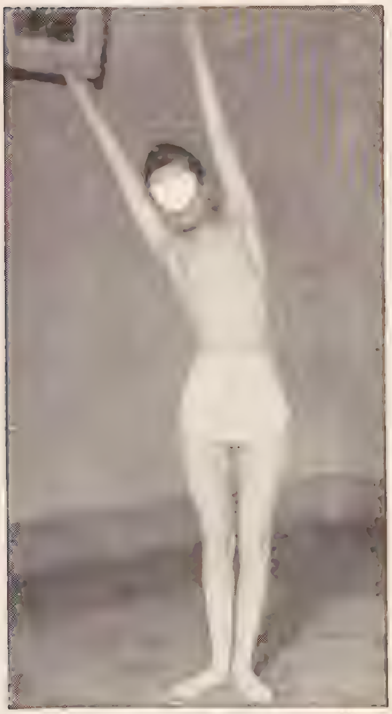
Neck Rest Standing, Heels Rising Position.

PHOTO. VII.



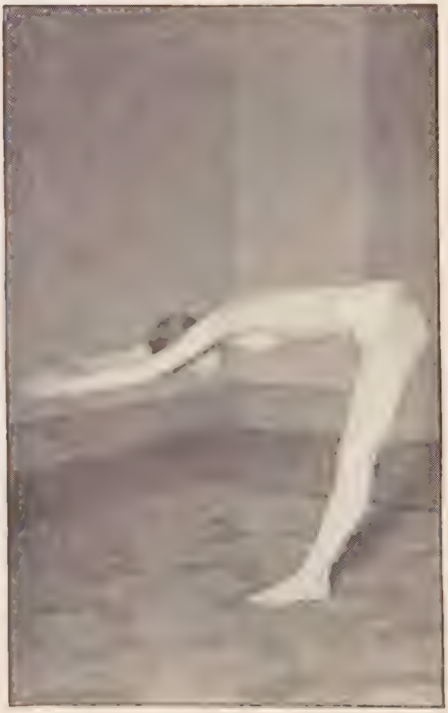
Neck Rest Standing, Trunk Change Turning Position.

PHOTO. VIII.



Stretch Standing, Trunk Sideways Bending Position.

PHOTO. IX.



Stretch Stride Standing, Trunk Forward Bending Position.

is elevated and fixed, the chest is expanded, the lungs are inflated, and the pressure on the heart and greater blood-vessels diminished, thereby producing increased oxygenation and increased circulation of the blood. The wing standing position also fixes the hips. The heels raising, alternately performed, stimulates the circulation in these parts,—flushes the parts, so to speak,—drawing the blood from the head, depleting the brain, and preventing "cold feet." This alternate movement also increases the secretion of the synovial fluid, preventing stiff ankle-joints. This movement may be repeated ten to twenty times.

Photo. VII. Neck rest standing, trunk change turning.—In the neck rest position the arms are lifted upward and the hands rest behind the neck, with the tips of the fingers touching. The wrist-joints are extended, the elbow is flexed, and the arms are abducted so that the elbows are directed outward. The scapulæ are drawn backward; the neck is extended and the chin retracted. In the trunk change turning the hips are fixed, so that the turning is confined to the upper part of the trunk only. In the lower extremities the knees are extended, and the heels are kept together, with the feet at a right angle.

Muscles exercised: in the position neck rest standing the scapulæ are rotated, the arms are lifted, the weight of the forearms bends them, and the hands are pronated and fixed to the head by the muscles of the scapulæ. Muscles: levator scapulæ; rhomboideus; trapezius (upper and middle part). In turning the trunk the whole "*muscle spirale*" is in contraction. Muscles: the pectoralis major and intercostales internus on the right side are contracted in the same spiral line as the pectoralis minor, serratus anticus major, and intercostales externus on the left side; the obliquus abdominis externus on the right side is contracted in the same line as the obliquus abdominis internus on the left. All these muscles work together or turn the side forward; on the back from the other shoulder. Muscles: the inferior part of the trapezius, latissimus dorsi, serratus posticus inferior in the same line as on the opposite side, multifidus spinæ and intercostales externus. The direct muscles of the trunk and abdomen are also more or less in action, some concentrically and others eccentrically.

The position neck rest standing has the same influence as the stretch standing, except that it affects all portions of the chest.

The trunk change turning exercises the *spiral muscles* of the trunk, affecting particularly the vena cava inferior, and thereby stimulating the passage of the venous blood to the lungs. This may be performed from six to ten times upon each side.

Photo. VIII. Stretch standing, trunk sideways bending.—The stretch standing, trunk sideways bending is taken from the stretch standing position, in which position the arms are extended parallel upward, with the fingers straightened. The head is extended and the chin retracted. The sideways bending is accomplished entirely by the lateral trunk muscles, the head and arms remaining in the stretch standing position.

Muscles: extensors of hands and arms, rotators of scapula, eccentric action of the muscles of the convex side of the bending body; the muscles of the bent side remain nearly passive, as the bending is mostly produced by the weight of the body after the first motions are started upon the same side. Extension of the fingers: the extensor digitorum communis, interossei interni, adductor pollicis, and adductors of the fingers. Extensors of the hand: extensor carpi radialis longior et brevior; extensor carpi ulnaris; extensor longus pollicis; extensor digitorum communis; extensor indices proprius; extensor minimi digiti proprius. Extensors of the arms: triceps; anconeus, also some of the extensor muscles of the hand; extensor digitorum communis. Rotation of the scapula: serratus anticus major and trapezius. Flexion of the trunk: multifidus spinæ and interspinales. Sideways bending: intertransversarii; transversus abdominis; quadratus lumborum; obliquus abdominis internus et externus.

The intention in assuming the stretch standing position is not only to stimulate the upper part of the thorax, but also to increase the effect of the trunk sideways bending, alternately performed, upon the liver, the portal system, the spleen, and the abdominal contents generally. This movement may be repeated from three to six times on each side.

Photo. IX. Stretch stride standing, trunk forward bending.—In the trunk bending forward the correct stretch stride standing position is first assumed. For the stretch standing position see Photo. VIII. In the stride the feet are separated about eighteen inches. In the bending forward the hands remain in a position parallel with the head. The bending is performed by the flexion of the entire spinal column. The knees must be extended, and the balance be maintained by a powerful action of the calf muscles. Bending the body forward sufficiently to touch the hands to the floor can be accomplished only after long practice.

Muscles exercised: in assuming the stride standing position the muscles exercised are the adductors of the supporting thigh and the abductors of the limb that is moved sideways. The first motion, bending the trunk, is performed by the flexors of the abdomen. After the bending is started the weight of the body increases the motion, and (the stretching muscles) the extensors for the back as well as the ligaments keep the body from falling forward.

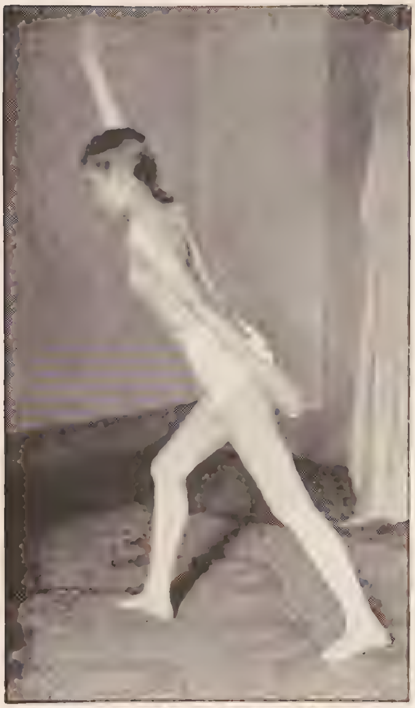
Flexors of the abdomen (or the trunk):

Direct, { rectus abdominis;
obliquus abdominis externus et internus;
psoas major et minor.

Indirect, { pectoralis major et minor;
serratus anticus major;
transversalis abdominis.

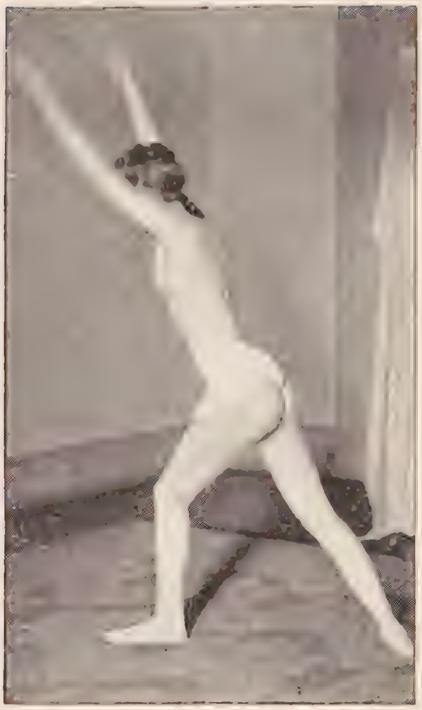
Extensors of the trunk: trapezius; rhomboideus; latissimus dorsi; serratus posticus superior et inferior; extensor dorsi communis; multifidus spinæ; quadratus lumborum; levatores costarum.

PHOTO. X.



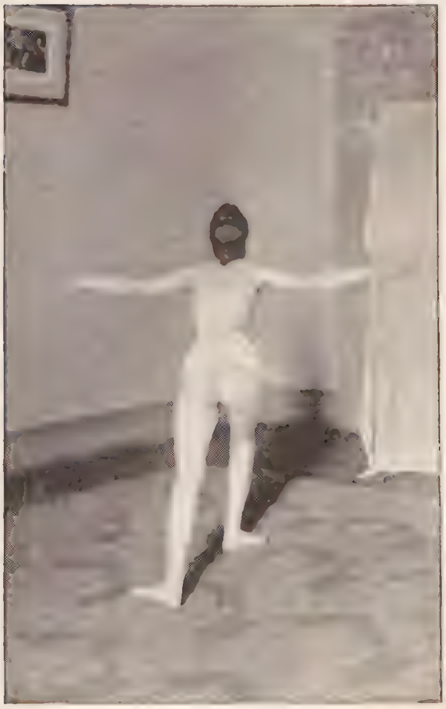
HALF STRETCH WALK, STANDING POSITION.

PHOTO. XI.



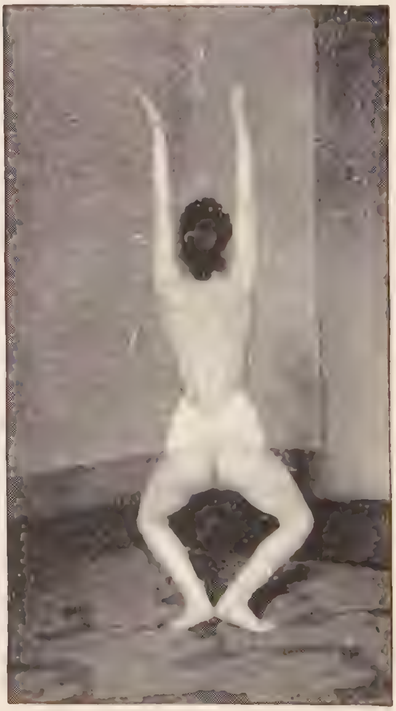
STRETCH WALK STANDING, TRUNK TURNING POSITION.

PHOTO. XII.



YARD WALK, FALL STANDING POSITION.

PHOTO. XIII.



STRETCH STANDING, HEELS RISING, KNEE BENDING POSITION.

Ligaments: fibrocartilagines intervertebrales; ligamentum longitudinale posterius; ligamenta subflava; ligamenta interspinalia et ligamentum nuchæ; ligamentum apicum; ligamenta transversaria.

The stretch standing position has been already described, but having this position in the trunk bending forward stretches the muscles and makes pressure upon the circulation of the blood-vessels of the spinal cord, and by the *slow* alternate flexion and extension the blood is forced into and out of the vessels of the brain, thereby removing the venous blood and replacing it with fresh arterial blood. This has a strengthening and stimulating effect upon the brain-substance and the spinal cord, as well as upon the entire nervous system to its finest ramifications.

Photo. X. Half stretch walk, standing position.—In the half stretch walk, standing position, the left arm is extended forward and supinated, and the right arm extended and pronated downward and backward. The left knee is flexed, and a great portion of the weight of the body is thrown upon it, by which action the extensors of the left leg are put into very strong concentric exercise. The right leg is extended backward and abducted, and is assisted in maintaining this position by the ileo-femoral ligament. This position may be retained from one to two minutes.

Muscles exercised: extensors of back, legs, and arms. Right arm pronated and moved backward by latissimus dorsi, rhomboideus, trapezius (middle and inferior part).

By taking this position any undue strain upon the abdominal organs is prevented. The intention of the exercise is to stimulate the back muscles as well as the circulation to the spinal cord.

Photo. XI. Stretch walk, standing, trunk turning position.—This position differs from the half stretch walk (Photo. X.) in both arms being extended instead of one. The lower extremities are both in the same position as in Photo. X. In the turning the arms must remain parallel to each other, and, as the weight is supported entirely upon the flexed knee, the turning is confined to the upper part of the trunk.

Muscles exercised: by the position, see Photo. X.; by the turning, see Photo. VII.

This position taken to its fullest extent has at the same time the strongest influence upon the respiration, circulation, and digestion of any single exercise in gymnastics, and has a beneficial reaction upon the entire nervous system.

Photo. XII. Yard walk, fall standing position.—This position differs from the half stretch walk (Photo. X.) in the position of the arms, which are extended horizontally both in the same line.

Muscles exercised: the deltoideus and infraspinatus lift the arms horizontally; the scapulæ are fixed backward by the rhomboideus and trapezius (middle part). For muscles exercised in the walk forward, fall standing position (extensors of back and legs), see Photos. X. and XI.

This extending and flexing the arms alternately in a horizontal plane has an expanding influence upon the chest, stimulates the respiration, and develops the arm muscles.

Photo. XIII. Stretch standing, heels rising, knee bending.—The arms are stretched up parallel, the fingers closed and extended, the shoulders back, the neck and back extended, the knees bent sideways and outwardly rotated, and the heels close and lifted. This exercise influences the extensor muscles of the body and expands the lungs, and is good for strengthening the spine and the nervous system. For the muscles exercised, see figures describing arm stretching and heels rising. Knee bending, the flexors act in the bending, then the extensors resisting are brought into action and are mainly exercised; these latter muscles are the gluteus maximus and the gluteus minimus (three glutei), pyriformis internus, semimembranosus and semi-tendinosus, and abductor magnus.

Physical Development in the Treatment of Disease and Deformity.—Most of the affections benefited by the movement cure are of the chronic type, and require for the most part the second or duplicated variety of exercises. In the majority of these diseases, too, what is required is to restore and develop the entire body: in other words, their cure involves the vital principle of physical culture,—the acquirement and preservation of health. In addition to this, in others it will be necessary to perform certain specific exercises intended to stimulate and restore directly the affected region or organ. These need not here be described in full, as they have already been given in more or less detail in other parts of this work.

In considering the subject of movements in their particular application to children, the employment of such apparatus as dumb-bells, Indian clubs, chest-weights, rowing-machines, etc., must not be overlooked. The length of this article, however, will not permit of their description in a proper manner, so that those interested are referred to the excellent writings of Maclaren, Sargent, Blakie, Ralfe, Ball, Dowd, Oswald, Schaible, and others. Nor must the subject of passive and active movements by means of the beautifully-constructed apparatus employed at Baden Baden and similar resorts be slighted. These systems possess a voluminous literature of their own, and are neither specially adapted for childhood and adolescence nor properly to be considered within the limits of this article.

In conclusion, attention may again be directed to the tendency to develop some parts at the expense of others,—the lower extremities of runners, the back and forearms of rowing men, etc.,—and to the necessity of insisting as far as possible upon the complete development of the body; in other words, emphasizing the statement of Dr. Parkes that, when a single muscle or group of muscles is exercised to too great an extent, these after growing to a great size begin to waste, which does not seem to be the case *when all the muscles of the body are exercised.*

MASSAGE.

By WILLIAM A. EDWARDS, M.D.

Massage, from the French word *masser*, literally means kneading, but in its application of to-day it denotes the communication of motion to the tissues from an external source, in contradistinction to the various movements, Swedish, localized, and remedial gymnastics. The latter terms are used to designate motions of the entire limb or limbs or of the trunk through the joints.

Historical.—The application of massage to medicine is of ancient date; indeed, as Weiss and Ritterfeld tell us, documentary evidence shows that the Chinese recognized its importance three thousand years before our time, and that remarkable book of the Hindoos, the *Susruta*, contains descriptions of mechano-therapy; the Brahmins continue even up to the present time the method that was in use thousands of years ago. The Chinese publications of centuries ago present illustrations which show clearly the correctness of the ideas of the Chinese physicians at that time concerning massage and medical gymnastics. Duhalde informs us that the schools then established have been maintained, until now they form part of the regular curriculum of the educated Chinese physician, and that it was probably from the Hindoos and Chinese that the Greeks and Romans obtained their information on masso-therapeutics. It was not until the internal disintegration of the Empire had commenced that Rome introduced the brutal exhibit of her circus sports in place of the more refined exercises learned from her Grecian neighbors; and finally the Christianity of the Middle Ages, in abolishing all Roman customs, made no exception of mechano-therapy, which consequently fell into disuse until about 1680, when Borelli called attention to its peculiar virtues. In 1740, Fuller, in England, published a little work which attracted much attention, and which appears to have been the turning-point in a revival of the popularity of massage, as the works of Börner and Gehricke (1748) appeared in rapid succession, followed in 1781 by that of Clement Joseph Tissot, later by those of Barthez and the two Webers, and by John Pugh's (1794) treatise on the science of muscular action, which in turn was followed by Barclay's Edinburgh work (1808), "The Muscular Motion of the Human Body," which Schreiber

(1887) considers worthy of special commendation. Balfour added his testimony in 1819, and Pravaz in 1827; but the greatest advance was made by Blache in 1855, and by Ling, whose work at the Central Institute of Gymnastics at Stockholm extended from 1806 until his death in 1839.

Schreiber tells us that the number of magazine articles and pamphlets from all sources up to 1874 ranged from one to four a year, increasing in 1879 to nineteen publications in various languages: this increase demonstrates the general interest in the subject that was awakening in the entire medical world. Most of the larger cities have erected institutes for sanitary gymnastics, and in the last few years it has won for itself a place in all the standard works on therapeutics and general medicine.

Effects.—The effects of massage or mechanical movements may be properly considered under two subdivisions, (*a*) primary and (*b*) secondary. Under the former we attempt the removal of exudates, extravasations, vegetations, and adhesions; under the latter, stimulation of the muscular and nervous system by increasing the circulation and setting on foot cell-changes and metamorphosis of tissue, and furthermore directly affecting the process of general nutrition. The effect of centripetal stroking of the body is to increase at once the rate of flow of the lymphatic and the venous circulation in the part; this has been demonstrated beyond peradventure by Von Mosengeil's well-known experiments upon the effect of massage in causing absorption from about the joints. This observer injected finely-levigated black India-ink into the joints of rabbits, and those joints which were subjected to massage showed a progressive decrease in size, while the others remained large. After death the India-ink was found, in those limbs which had been manipulated, scattered through the thigh and as numerous foci in the areolar tissue.

Classification.—We adopt the classification of Mezger, which is generally accepted to-day,—viz.: 1, effleurage (stroking); 2, frictions (friction); 3, pétrissage (kneading); 4, tapotement (percussion). The first manipulation consists in stroking with the palm of the hand or its radial border, or with the tips of the fingers, or with the thumb alone, and the force applied may be the gentlest possible or the heaviest pressure made by reinforcing the operating hand with the other laid on top of it. Where deep effect is desired, as the removal of exudations in the tendons or the intermuscular tissue, the thumb or several fingers are used, the tips being held nearly perpendicular to the surface, and the degree of penetration is dependent upon the amount of pressure exerted. When working in bony regions, only moderate pressure must be used; when concerned with large muscular masses, the fleshy cushions of the palm of the hand must be brought into requisition, and the patient so placed that the masseur can be aided by the weight of the upper part of his trunk in giving force to the stroke. As a rule, the strokes are to be made centripetally,—that is, towards the central organ of the circulation; in certain rare cases this rule may be disregarded, and the direction of the stroke may be centrifugal.

Friction consists in the use of the thumbs or the tips of the fingers in strong, forcible circular rubbings, followed by centripetal stroking. The object of these manipulations is to act upon deposits in diseased parts in such a manner as to distribute them among healthy tissues, and, inasmuch as pathological deposits may exist in any tissue, we can formulate no general rule for the direction which the frictions may take, but it should rather be our aim to reach sound tissue, and consequently the friction may be centrifugal if the healthy parts lie in that direction.

Pétrissage, or kneading, is performed with the tips of the thumbs or with the index finger and the thumb, and consists in picking up a muscle or other tissue and subjecting it to firm pressure, either by the thumbs and fingers or by the fingers and the dense tissue that may underlie it.

Tapotement, or percussion, is usually divided into four sections: 1st. Clapping with the palm of the hand or with an instrument especially constructed for the purpose. 2d. Hacking, which is performed with the ulnar border of the hand or with the extended fingers, depending upon the impression desired to be created; with the fingers the motion is made from the wrist-joint, with the edge of the hand it is either from the elbow- or from the shoulder-joint. 3d. Punctuation, which is performed with the tips of the fingers, and is usually applied upon the head or upon the præcordia. 4th. Beating with the clinched hand; usually applied over the thick muscles of the thigh.

Local Massage.—Having considered the special forms of massage, it will be well to review its application to the various parts of the body, which we will term local massage.

Massage of the leg commences with stroking from the foot to the hip, and then friction from the interosseous muscles upward, followed by stroking, which in turn is followed by kneading, and the treatment of the limb is completed by hacking over the muscular parts. The arm receives a similar treatment, which is best applied in the semi-flexed position.

The Chest.—Here again we commence by stroking with the hands on each side of the sternum, manipulating upward and outward with circular movements. The præcordia is to receive circular punctuation: too much strength must not be used, and the treatment of this region may be terminated by hacking and clapping. The back should receive much care, and is to be treated by downward stroking from the base of the skull to the sacrum, taking care to avoid the spinous processes of the vertebræ; this is to be followed by friction with the tips of the fingers in the same general direction, making the movement more lateral, so as to include the postero-lateral aspect of the trunk. Then spread the hands over the back and knead with the thumbs between the vertebræ, and administer hacking up and down the back several times,—some authorities say ten times, but this would certainly be more than a child could stand. It is well to finish by stroking and clapping, particularly the latter, and especially on the right side.

The Abdomen.—The patient lying upon the back, with the legs flexed on the thighs and the thighs on the abdomen,—to secure perfect relaxation, which is essential,—the masseur commences on the right side by spreading the right hand over the abdomen and pressing with the heel of the hand and the fingers alternately; this manipulation is to be carried out in the direction of the transverse colon. Kneading is next applied by the tips of the fingers.

The *head* is best treated by stroking, friction, hacking, and shaking. The first is applied principally to the forehead: with "the thumbs between the eyebrows, the stroke is to be carried firmly over the temples to the ears, both thumbs working together." Friction of the entire head with the palm of the hand, hacking with both hands, making circles over the head in all directions, and gentle shaking by clasping the forehead with both hands and shaking the head carefully and deliberately, will, as a rule, be the best method of procedure.

In *massage of the face* only two motions are used,—stroking and friction. With the index finger in the mouth, the thumbs stroke the muscles of the cheek, and by the thumb and the index finger the muscles are picked up and subjected to rotatory movements.

Massage of the Throat and Neck.—Von Gerst advises that the patient be "stripped to the middle of the chest, and stand with head thrown back and shoulders relaxed; deep, full, and regular breathing by the patient is essential, else the return venous circulation will be impeded. Each stroke consists of three parts. First, the open hands, with the palms upward, are placed, with their ulnar borders in the right and left cervical fossæ, between the head and the neck, so that the tip of the little finger and the last joint of the ring finger shall rest upon the mastoid process behind the ear, and the ball of the little finger under the horizontal branch of the lower jaw. A centripetal movement is now begun with the ulnar borders of the palms thus placed in the superior cervical region, and is performed as follows. While the ulnar border is moving towards the middle of the neck, both hands perform a rotation on their long axes, so that the radial border turns upward and inward towards the head and finally reaches the position first occupied by the ulnar border, and thus the entire palm has come in contact with the neck and is now employed in giving the stroking." A slight degree of pressure is to be exerted by the balls of the thumb upon the jugular veins, and by the palmar surface of the fingers upon the venous and lymphatic vessels along the lateral cervical regions. At the supraclavicular fossa the hand again turns upon its long axis, and the radial border of the palm once more comes into use. The lateral cornua of the hyoid bone and the larynx are to be avoided; pressure on either produces pain and inclination to cough.

MOVEMENTS.—The various Swedish movements are peculiarly applicable to the growing child, and, when judiciously used, will do much to produce a symmetrical growth, preserve health, and correct vicious tendencies.

THE APPLICATION OF MASSAGE AND SWEDISH MOVEMENTS IN THE TREATMENT OF DISEASE.—We will first consider the conditions which demand *neck-massage*, as in this region, owing to the numerous superficial veins and the distribution of the carotid arteries, we can act almost directly upon both circulations. Indeed, massage in this region has been compared to copious bloodletting without its disadvantages; consequently it is most advantageous in congested conditions of the brain and its membranes. Being a rapid method of depletion, it is efficacious in sunstroke, also in headache and hemicrania when these are of the congested type; but when the latter occurs in weak, anæmic, nervous children, massage of the neck is useless; our efforts then would better be directed to manipulation of the scalp, forehead, and temple, bearing in mind, however, that in these children massage in this region is apt to produce hypnotism. Vretlind believes that in many instances hemicrania is due to chronic myositis, in which case, of course, the muscles must be carefully manipulated to remove the indurations. Walter Johnson goes even further, and says that the necks of nearly all his patients who had suffered for any length of time from head-affections were swollen and indurated, with, probably, enlarged and swollen glands in the neighborhood.

Massage is said to have been used with the most gratifying results in the so-called wasting diseases of children.

Peripheral neuralgias which are not dependent upon central nervous disease or deep-seated pressure (tumors) are peculiarly amenable to the effects of massage,—percussion or kneading. Sciatica is often speedily relieved, particularly if it be rheumatic and come under treatment before alteration has taken place in the sheath or the neurilemma.

The so-called *sensitive points* met with in different parts of the body in nervous girls at or about adolescence are often entirely removed by massage. These points when located over the spinous processes of the vertebræ are often responsible for hysterical outbursts.

Cramp, when peripheral and due to over-use, over-tension, or irritation of the nerves, is to be treated by percussion, kneading, and strong stroking of the muscle or group of muscles affected.

Chorea.—If the child is violent, it should be held supine upon a mattress for from ten to fifteen minutes, while the masseur applies gentle stroking with the palm of the hand over the entire body; the time occupied in this application is to be rapidly increased to an hour and to be repeated every three or four days. In a short time regular passive movements are to be added to the treatment, care being taken to overcome the tension of the antagonistic muscles, so that within eight or ten days the child can take a few voluntary active muscular movements. Within the next week gymnastic exercises are introduced, which must be of the simplest form, and are to be combined with simple voluntary movements of the limbs and trunk. It is well to have the patient imitate the movements of the masseur, in order to exercise his will-power. Rhythmic movements timed by music are of inestimable benefit for the exercise of the child's will and brain. The case is apt to improve up to a certain point and then reach a stationary period, in that event sorely taxing the physician's skill; but a happy termination will usually be brought about by kindness, persuasion, and encouragement. Blache states that of one hundred and eight cases of chorea in childhood treated as above recorded not one relapsed.

It is not our province in this article to refer to the benefits to be derived from massage in the peripheral palsies of motor nerves, in lead-poisoning, or in the local anæsthesias; but we desire to call special attention to its use in anterior poliomyelitis,—*infantile spinal paralysis.* It must, of course, be applied only after the acute manifestations of the disease have subsided and all evidences of irritation in the central nervous system have passed away: then, and then only, will massage be of benefit; furthermore, after massage has been persisted in for a time, the muscles will respond to electric stimulation. Von Mosengeil mentions a case in which at the expiration of four months electric excitability was completely restored. Norström adds his testimony to the efficacy of the method, but Murrell considers that some of the improvement is due to the retrogressive character of the disease.

Post-diphtheritic paralyses, as Kellgren tells us, are often at once and permanently relieved by manipulation; he records the case of a girl under treatment from June 23 to August 16, eighteen séances in all, who was discharged cured and in whom no relapse occurred.

Angina pectoris has often been relieved by vigorous stroking and kneading over the heart. Mühlberger records the case of a young man in whom the relief was marked, the severity of the attacks being much lessened.

Abdominal massage is of benefit in many cases, as in this way we can act upon the circulation of the blood and lymph and stimulate the secretions and excretions of the entire gastro-intestinal tract, to say nothing of its effect upon effusions or new growths or fæcal accumulations. Hence, as Lee says, abdominal massage is indicated in acute or chronic gastric and intestinal catarrhs, dyspepsias, cardialgia, dilatation of the stomach, intestinal obstruction, tympanites when not dependent upon inflammatory action, ascites, and, finally, all the sequelæ of peritoneal inflammation, as peritoneal or extra-peritoneal exudations or adhesions, provided, of course, that all signs of inflammation have subsided. In *habitual constipation,* combined with pelvic gymnastics its effect is most happy; indeed, even when the accumulation is so excessive that occlusion is threatened, it is often possible by very gentle massage to move the hardened fæcal mass onward towards the sigmoid flexure so that it may be reached and softened by injections.

The liver and spleen are directly accessible to manipulation when they are enlarged, and even when they are normal in size they may be indirectly affected by massage: hence it is indicated in hepatic congestion, enlargement, or jaundice, and in splenic engorgement; gymnastic exercises should be added to the treatment.

In the anæmatoses, as *chlorosis* and *anæmia*, general massage of the whole body, in combination with the usual plan of treatment in such cases, will often hasten the return of the blood to normal. We have already called attention to the effect of massage upon the blood- and lymph-circulation: its effect on superficial *œdema* will likewise be most happy, whether the œdema be a manifestation of a general disease or be due to local obstruction. Massage is peculiarly grateful to those patients who present œdema of the lower extremities, and is particularly to be recommended in the œdema of scarlatina, where it not only acts directly upon the circulation of the parts, but also assists in restoring the skin to the normal.

In diseases of the *heart*, massage is capable of affording much relief. Gendrin formulated the apt expression that "massage constitutes a sort of accessory venous heart."

Several careful observers demonstrate beyond question that massage causes an elevation in the surface-temperature. For example, Mills has recorded observations on the general and axillary temperature in a girl nine years of age who had partial paralysis of the right arm and both legs following scarlet fever; the temperature showed the following changes:

Before Treatment.	After Treatment.
99.5° F.	100° F.
99.3° F.	100° F.
99° F.	99.5° F.
99° F.	99° F.
99° F.	99.5° F.

Weir Mitchell is even more decided upon this point, and remarks that he has frequently seen the strangely cold limbs of children suffering from infantile paralysis gain from 6° to 10° F. during an hour's massage.

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PROPHYLAXIS OF DISEASE IN CHILDREN.

By J. WELLINGTON BYERS, M.D.

Prophylaxis may be designated as a series of methods or procedures whereby contagious disease is restricted and prevented by suppressing or removing its predisposing conditions and destroying or modifying its exciting causes.

Viewing the subject in the light of this definition, it will be seen that preventive medicine is largely dependent upon a knowledge of the etiology of disease, and that its sphere is precursory. In order, therefore, to obtain a satisfactory understanding of its principles and methods, such as may be peculiarly applicable to contagion among children, it will be necessary to examine into those circumstances and to discuss those influences which are ordinarily presumed either to favor or to retard the origin, the development, and the distribution of disease.

Observation shows very generally that the conditions which usually constitute human environment, including such factors as air, water, food, clothing, habitation, climate, and telluric influences, have a constant and powerful effect in shaping the type and advent of morbid phenomena, and that they often furnish the means by which the immediate or exciting causes of disease reach, invade, and are eliminated from the body. Hence the whole system of prophylaxis purposes to understand these conditions and to take such advantage of them as will control or destroy their morbific agencies and influences. While it is true that investigations with etiological data are highly essential and conducive to a philosophical or scientific interpretation and practice of preventive methods, still it must not be concluded that its progress in the past or its prospect for the future has been or is solely contingent upon the exact amount of information possessed concerning the causation of the disease, since there are numerous and valuable facts associated with its history which are not dependent upon these features for their utility. Indeed, experience pretty clearly demonstrates that it is eminently possible to achieve a very efficient system of preventing disease with little or no knowledge of its exciting causes. Many of the practices now in vogue were originated long before the era of the germ theory; still they are now none the less reliable. A number of disorders corroborate this and illustrate the methods of prevention in a high degree, though their causes are quite unknown. This is exemplified in all the acute exanthemata and in diphtheria, each of which shows unmistakable evidences of amenability to isolation and disinfection, yet the personality of their specific agents is conjectural. Therefore it must never be concluded that for prophylaxis to be efficient the causes of disease must be known, since experience teaches the contrary.

As set forth in the definition, there are two general methods employed in attempting to restrict and prevent the spread of contagious disease. The first of these is preparatory and defensive, and treats of those conditions, externally in the media and internally in the body, which invite or predispose to the occurrence of morbidity or which foster and sustain its actual exciting causes. The second class in contradistinction to this is aggressive and destructive, and purposes to seek out, attack, and kill the specific germs of contagious disease or to render their surroundings so inimical that they will perish. This latter method constitutes disinfection. As preliminary to a consideration of these two divisions of the subject, it must be observed, however, that the disorders especially referred to here as preventable and avoidable have the common features that they are transmissible mediately or immediately from sick to seemingly well though susceptible persons, and that they are each presumably due to or caused by a specific germ or contagion, which is, as a rule, a living unicellular vegetable micro-organism of the general class bacteria. Bearing this in mind will better enable us to perceive the scope and plan of prophylaxis. All zymotic diseases, while not originating in peculiar environment, so far as we are now aware, yet are its inevitable outcome. In other words, there must always be susceptibility upon the part of the infected subject and pathogenicity upon the part of the germ, before disease can arise and manifest itself. If there should be anything in the external media or in the internal conditions of the body calculated to hinder or frustrate the operations of the contagion, and it fails to germinate or reproduce, the individual escapes. Hence there must always be a certain amount of co-operation among all the factors concerned in disease-processes in order for the disease to exist. As has been stated, these conditions relate to the exciting cause so as to foster and assist it, or to the body so as to debilitate it and render it vulnerable or susceptible to pathogenic agents. A satisfactory understanding of how these influences act to produce these results is obtainable by an investigation of the relations of the two factors of predisposing conditions and exciting causes.

Before proceeding to a discussion of these, however, I deem it necessary by way of parenthesis to mention the misconceptions which have arisen in regard to the use of the terms predisposing *conditions* and exciting *causes.* It has been customary, under the older notions of disease-etiology, to employ the latter of these when describing circumstances antecedent and related to its occurrence. In the light of recent developments this is both wrong and illogical, and should be dispensed with, from the fact that we now know full well that none of the agencies or conditions usually embraced by these phrases are at all capable of generating or acting as a cause of specific disease.

Everything that happens must either have an adequate cause—that is, an agent—or be spontaneous; still, there is a material and broad difference, when speaking of contagious disease, between substances and conditions, between agents and agencies. The first are definite, an entity; the latter, a state or relation. To illustrate practically, take the instance of erysipelas following a wound. Here the wound acts as a condition which renders the entrance of the erysipelatous virus possible, and without which it could not enter or progress, yet the wound is in no sense a cause of erysipelatous disease and would never under any circumstances originate or produce it in the absence of the specific poison. It is a contributory or predisposing condition, which assists the virus, and not a cause of the disease. This is a distinction seldom insisted upon in etiology heretofore, though, as a means of a clear idea of the causation of pathogenesis, such as will be of service in antagonizing its advances and encroachments, its propriety cannot be gainsaid. In fact, it is fundamental to quite a number of the methods pursued in prophylaxis, and they owe their practicability and efficiency to the fact that disease-conditions and disease-causes are distinct from each other. It is by studying and classifying these separate influences that we learn to modify and remove them and establish a scientific basis for preventive medicine.

As has been remarked, predisposing conditions may be made to include all those external states which determine the mode of life, propagation, and distribution of disease-germs outside the body, and also those internal influences which alter or control the resistance of the body to their invasion. With regard to the conditions which chiefly influence the body so as to render it susceptible to disease-agents, there are two kinds, the internal and the external, some of which are avoidable and others inevitable. It does not lie within the purpose of this article to enter into details as to how and why these conditions produce their effects, or how and why the removal of them gives exemption and immunity. We must accept them as facts fully corroborated by a long series of reliable experience. As to the rationale and mechanism of the physiological processes that lead to protection from disease, there are many vastly interesting phenomena which recent advances in bacteriology and chemistry have brought forward. The germ theory of disease, together with the labors of Metschnikoff and his coadjutors in the phagocytic actions of cells, opens up a new and luminous prospect for all medicine, and ere long we shall possibly be equipped with the facilities for knowing precisely and seeing actually the processes of immunity and protection going on. Undoubtedly these, in addition to the experiments now being conducted throughout the world by means of attenuated viruses inoculated for the purpose of preventing and anticipating infectious disease, foreshadow great possibilities for all measures of prophylaxis. Again, the introduction for similar purposes of ptomaines, leucomaines, and such other drugs and synthetical chemicals as have analogous molecular composition or constitution to the specific poisons of the pathogenic germs, will eventually place preventive medicine in the foremost rank of beneficent science.

Among the external conditions that produce tendencies towards disease are foul air, damp soil, filthy habitations, poverty of food and clothing, defective sewerage, and the like. All of these, by lowering the vital forces and depressing the systemic functions, induce a condition of susceptibility to morbid agents. They prepare the soil for disease-germs and create proclivities favoring their development. The ancients, while knowing nothing of the final vital reactions of the tissues, or how they operated to protect the system, recognized the great importance of hygiene and sanitation in furthering these events and controlling and warding off the approaches of disease. Hence the Greeks and Romans never failed to inculcate regulations pertaining to hygiene. Moses, the law-giver, laid down a sanitary code, with severe penalties attached for infringement, and gave directions for the disinfection of persons, places, and things, so thorough and effective that even this advanced age can find little room for improvement. Therefore there is nothing new in the statement that health protects itself and is the means of its own defence.

Recent developments in histology and bacteriology show that, while the cells of the body possess the ordinary functions of growth, reproduction, and assimilation, they have in addition aptitudes or inherent powers for discriminating between natural and foreign substances in the economy, and that they seize upon, devour, and cast off the latter in a most marvellous and perfect manner. In order to do this, however, and keep the system protected from the invasion of noxious substances, the cells must be sustained at the normal standard of healthy action, their pabulum and stimuli the best, such as hygienic influences are capable of furnishing, since if they be depressed by the presence of predisposing conditions they are unfit to cope with the enemy the micro-organism, and they perish. Therefore, since the vital organism is qualified to protect itself, it should be assisted by all the means at hand, and everything removed that tends to hinder or cripple its efforts. The experiments recently made by Roger and Charrin confirm this in a remarkable degree: they find that, even when the serum of the blood is impoverished, it alone may determine susceptibility and render the entrance of the germ possible, bacteria being far less active and virulent in it the nearer it approaches the standard of healthy blood. When we recall the very general prevalence of germs, the wisdom and necessity of exemption and protection being dependent upon something outside from and superior to human knowledge and precautions become obvious.

Among the internal states or conditions which create liability to disease-agents may be mentioned age, sex, nation, race, certain diseases and drugs. As to the exact nature and conformations of the tissues which permit the invasions of the morbific substances, we know these principally by the result,—susceptibility. It is highly probable, however, that this subjective condition is largely influenced by circumstances related to the nutrition of the body as a whole and the finer cells of the tissues in particular, and it is only when they depart from this standard that the influence of the pathogenic germ is capable of doing harm. From the statements already made in connection with the theory of the cellular warfare which goes on between the disease-germ and the cells of the organism, we see that disease is kept off or prevented by an active and discriminating process of the body, and that susceptibility is the absence of these resources or a failure to exert them. As yet, we are not in a position to specify exactly what the germ must and must not find in order successfully to invade, multiply in, and set up disease in the body. We know in a general way that there must be a condition of susceptibility, and that the germ must meet with a combination of circumstances favorable to its growth and development, among which may be mentioned suitable pabulum and proper and agreeable temperature, and that it must nowhere encounter any substances or conditions which either check or injure it. It is known that exciting causes can act only upon systems predisposed towards their reception, but whether this liability is chemical, mechanical, or vital, in addition to what has been said, we are not as yet prepared to state. Susceptibility is primarily weakness, though there are doubtless other influences which alter and lessen the system's natural protective powers, in kind as well as in degree.

Hence, when debility is present, howsoever induced, whether by external conditions of environment or by reason of peculiar internal states, the system is lowered and rendered incapable of contending in a successful and physiological manner with the pathogenic intruders, and the body succumbs to disease. Therefore, if we wish successfully to assist and protect the system, we should remove in advance all those conditions and influences which have a tendency either to favor the exciting causes or to depress the systemic functions of the body. Sanitation fortifies the normal actions and removes all depressing conditions, while disinfection modifies and destroys the exciting causes.

Thus having briefly outlined the necessities, reasons, and philosophy of prophylaxis, we will now pass to an enumeration of the methods and details by which these objects may be accomplished.

Whenever there is reason to suppose that an outbreak of infectious disease is imminent, there should be a thorough overhauling of everything calculated to facilitate its approach or protract its stay. In all these undertakings there should be system and regularity, the prime object being always kept in view. The first case or cases of disease should be at once isolated, and all visitors and susceptible members of the family warned and excluded from the apartments or premises. All sources of decaying matter should be at once freely sprinkled with chloride of lime, or wet with a solution of the same, and removed. There should be free ventilation and absolute cleanliness as far as is possible. When the case subsides, all bedding, clothing, furniture, rooms, and persons should be thoroughly submitted to such processes of disinfection as will best eradicate every vestige of contagious matter. The belief that certain germs are the efficient causes of contagious disease has led to the employment of a great number of experiments for the destruction of bacteria by means of heat and chemical substances, and thereby the doctrine of disinfection has been established and developed. Some diseases are far more contagious than others, measles and whooping-cough being feebly so, and scarlet fever and diphtheria very much so.

By a disinfectant is meant, in the language of the definition given by the American Public Health Association, "any agent capable of destroying the infectious agency of infectious material." Most of our knowledge concerning the efficacy of chemical disinfectants is drawn from their influences upon putrefying matter of organic origin, and it has been found that what will arrest putrefaction will also destroy the germs of disease, and *vice versa.* Only a few diseases are now considered to be directly concerned with filth. Cleanliness of the surroundings has little or nothing to do with small-pox, measles, scarlet fever, or whooping-cough, and no amount of sanitary effort will influence them in the least. Such diseases, however, as consumption, diphtheria, the various dysenteries and diarrhœas, and cholera infantum, are directly concerned with filth in relation to water-supply, drainage, and sewerage. (Billings.)

With regard to the mode through which disinfectants exert their influence, several explanations have been offered. Some are thought to act by oxidization, others by their property of coagulating albumen, and still others in a way either entirely unknown or but imperfectly understood. What is a disinfectant for one disease of this character, however, is a disinfectant for all, and it will only be necessary to review a few of the most reliable and important ones.

Generally, in the absence of spores (and no human disease has any, so far as we know), a heat of 250° F., sulphur dioxide in fumes, and solutions of the bichloride of mercury will destroy all pathogenic germs. A number of disinfectants are known to evolve oxygen freely, and are thought to hasten in this manner the disintegration of the noxious matters.

In each particular disease there is always some peculiar or special portion of the body involved which is occupied by and reproduces the exciting causes of infection. It is to these parts that we must always first direct our attention when beginning to disinfect for the disease. Fortunately for prophylaxis, the exact location of the centres for breeding is known in quite a number of diseases, and we can attack these seats directly. In all the exanthemata this is the case, and in them it is the skin and the mucous membranes which should receive the most attention; while in typhoid fever and cholera it is the discharge from the bowels, and possibly from the kidneys also, which we must particularly attend to.

For personal disinfection, Labarraque's solution, diluted with twenty parts of water, is suitable for washing and bathing the body. A weak carbolic solution, or a one-per-cent. solution of chloride of lime, will also be found efficient for the same purposes. Oily disinfectant inunction for the skin is one of the best methods of preventing the detachment of epithelia and pus, and should be used from the commencement of measles, small-pox, scarlet fever, and chicken-pox. It is found better and more agreeable when mixed with camphorated olive oil or with carbolic acid and olive oil. The throat and fauces in these diseases should also be washed with Condy's fluid or a weak solution of sulphurous acid. All discharges from the nose, eyes, and mouth should be received upon rags and immediately burned, or, if upon towels and handkerchiefs, these should be put in a solution of chloride of lime. The stools should be disinfected by a solution of bichloride of mercury, one-half ounce to the gallon of water, and all clothing treated by placing it in boiling water to which have been added two ounces of chloride of lime for each gallon. With regard to the disinfection of the apartments, furniture, and hangings, an observance of the directions in the general remarks that are to follow will be amply sufficient for all purposes.

HEAT.—Extremes of temperature have long been known to be disastrous to animal and vegetable life, and are doubtless among the most efficient and satisfactory agencies that can be employed for disinfection. Of course it is not necessary to say that fire will destroy the principles of infection, since it is complete combustion. Heat in particular has a powerful and constant effect upon all albuminoid substances, both in coagulating and in desiccating. It is undoubtedly the best means of disinfecting bulky material, such as bedding, curtains, and clothing, and, if they be properly exposed to its action, all the contained life or contagion will certainly be destroyed. Heat is employed as a disinfectant in several ways, among which may be mentioned dry, moist or boiling, and steam heat. Drs. Parsons and Klein have made numerous and elaborate experiments both on the degrees of heat and on the time necessary for disinfection. The results of their investigation show that dry heat is best adapted to the usual purposes of disinfection. Ordinarily this kind of heat can be supplied by a common laundry drying-closet or a baker's oven, and will be sufficiently powerful to destroy all contagion unaccompanied by spores. Dry heat has one principal objection, however: it penetrates bulky and badly-conducting substances very slowly, and the time usually allotted for the destruction of germs is far too short for it to be effective. Hence if the drying-closet or oven be used the articles should remain in it at least four hours, and the temperature should not be below 200° or 225° F., the latter being much better. With respect to the amount of heat that can be borne by ordinary fabrics, scorching is said to occur at different temperatures in different materials. White woollens are always soonest affected, and should be carefully watched. To avoid these bad effects in them, the temperature should never exceed 250° F.; and even this may in a majority of cases be too much for the finest woollens. Dry heat is generally applicable to all that class of goods which can be boiled, and is said to be materially aided in penetration by the addition of a certain amount of moisture, though the moisture is of no assistance in facilitating the destruction of the germs.

Dr. Henry has been successful in destroying the infective principle of scarlet fever, contained in clothing, by means of a dry heat of only 140° F. kept up for three hours. Most authorities, however, recommend a temperature of at least 225° F. for all kinds of exanthematous disease, such as small-pox, measles, chicken-pox, and the like. Sternberg's investigations demonstrate that the various germs perish at the following temperatures:

Typhoid bacillus	132.8° F.	Cholera bacteria (Koch)	125.6° F.
Anthrax bacillus	129.2° F.	Micrococcus Pasteurii	140° F.
Pneumococcus	136.4° F.	Staphylococcus p. aureus	136.4° F.
Tuberculosis bacillus	212° F.		

Koch has found that bacteria in general, free from spores, cannot resist a temperature of 212° F. for many minutes, and that if it is continued for an hour and a half they invariably perish.

As to the action and results of moist heat or boiling as a disinfectant, experiences are not very satisfactory. Still, if the boiling be kept up for several hours, and a little carbolic acid, or chloride of lime or of zinc, be added to the water, the results can be relied upon as effective. Should there be any doubt about the destruction of the germ when lodged in clothing or other articles of similar character, we should not hesitate to employ the highest dry heat compatible with the safety of the goods. The finest fabrics will withstand for a reasonable time an elevation of 225° F., and linens, cottons, and woollens may be trusted to it without the apprehension of any injury or danger. Two hours of dry heat such as has been described will be amply sufficient, and no evil consequence will follow either to the safety of the goods or in the form of disease.

Steam is considered to be a rapid and powerful disinfectant, experience showing pretty conclusively that at 212° F. it will destroy all contagion, and the complete penetration by steam for five minutes is found capable of thoroughly disinfecting. Koch, Parsons, Klein, and Loefler all are agreed upon this particular. Steam penetrates far more rapidly than dry heat, and is much more destructive, in the time occupied, to all germs. A pressure of twenty-five pounds is said to assist materially in its destructive action.

Chemical Disinfectants.—These are employed in the form of gases, liquids, and solids.

Gaseous substances are employed principally for aerial fumigation, and are applicable for the disinfection of apartments, etc. I shall not undertake to enumerate the various articles of this class that have been used from time to time, but shall simply confine myself to a description of a few of the leading ones, such as experience demonstrates to be effective.

A number of chemical substances are known to evolve oxygen very freely when brought in contact with organic bases, and in this manner disintegration of the obnoxious matters is presumed to take place. Chlorine, bromine, nitrous fumes, and ferric compounds are supposed to disinfect to some degree by this method. With respect to the chemical reactions that take place in the use of the other substances which will be mentioned farther on, very little appears to be known.

Sulphur Dioxide.—The most available, inexpensive, and reliable disinfectant that can be employed for fumigating apartments and preventing the spread of exanthematous diseases is sulphur dioxide, (SO_2). The Greeks and Romans were acquainted with the preservative properties of this gas, and used it as an antifermentative in wine. It is two and a quarter times as heavy as air, and is usually generated by burning sulphur. It has a peculiarly pungent and disagreeable odor, which is perceived when a sulphur match is ignited. Guyton de Morveau, who first studied its action, was of the opinion that it would completely disinfect miasms. Its chemical action is supposed to be due to its power to deoxidize, the resultant acid coagulating albuminous matter. For the disinfection of rooms recently vacated by diseased persons, three pounds of sulphur should be burned for each thousand cubic feet of air space. The sulphur can best be ignited by pouring over it two or three ounces of alcohol and applying a lighted match. The room during the entire period of fumigation should be kept completely closed, and about twenty-four hours afterwards opened and thorough ventilation allowed. Before using this agent, however, all carpets should be taken up, the wall-paper removed and burned, the hangings thoroughly loosened, and every part exposed so that the gas can penetrate everywhere. That sulphur dioxide has a direct action upon the vitality of the germs of disease follows from the statement made by Sternberg, who says, "In the proportion of one to two thousand, in an aqueous solution, SO_2 kills micrococci in two hours." Koch asserts that anthrax-bacilli are destroyed in a one-per-cent. solution in thirty minutes. The experiments of Baxter led him to the conclusion that "it is the most powerful volatile disinfectant known." Sulphurous acid and sulphur dioxide are both destroyed by chlorine and permanganate of potassium, and should never be used in conjunction with either. The great solubility of sulphurous acid renders it one of the most valuable substances with which to disinfect liquids. Dr. Edson, of the New York Board of Health, in a recent report, says that sulphur dioxide is the most practicable and reliable means that he has ever seen employed for the disinfection of the exanthemata and diphtheria.

Chlorine.—This is a pale yellowish-green gas at ordinary temperatures, about two and a half times as heavy as ordinary air. It is a powerful oxidizing agent, and extremely irritating to the air-passages when inhaled. It decomposes ammonia and sulphuretted hydrogen, and affects all compounds arising from the putrefaction of organic matter. It is commonly used for the same purpose as sulphur dioxide, and, like that agent, renders the occupation of the apartments temporarily impossible. It bleaches organic matters and destroys odors, either by withdrawing hydrogen or by direct oxidation. Chlorine is usually generated by means of chloride of lime, moistened either with water or with dilute sulphurous acid. There are several popular disinfectants which contain this gas in sufficient proportions to be of service: among these may be mentioned Platt's chlorides, Labarraque's solution, and Javelle water. Chloride of lime is often used without the addition of an acid, being scattered as a powder in vaults, privies, and gutters. Bromine and iodine have properties similar to chlorine, though they are not so powerful in their action, and should not be relied upon when better substances can be procured. Iodine can be liberated from the scales by simply applying heat underneath a plate upon which they have been scattered.

Fumigation should always be regarded in the light of an accessory, and should be, when practicable, accompanied by the processes of baking, steaming, and washing. All the wood-work—walls, floors, and furniture—must be rubbed down and washed with a solution of carbolic acid, one pint to the gallon of water, or a solution of bichloride of mercury, an ounce to the gallon of water, and the clothing and bedding baked, or washed in a solution of chloride of lime. A failure to make these processes complete by leaving a single place or article overlooked will create a new centre for infection, and all the work will have been in vain.

Carbolic Acid.—An important advance was made in disinfectants when the virtues of this substance were discovered and brought forward. The great advantage of it, as a liquid, is that it is slightly volatile, and therefore capable of being sprinkled in apartments and upon substances where it will penetrate every corner and crevice and be of service. It requires about twenty-five parts of water for thorough solution, and in this proportion is a powerful disinfecting liquid against all contagion. It coagulates albumen in the proportion given, and enters readily into union with organic substances wherever it meets them. Dr. Sansom has shown that a mixture of carbolic acid and glycerin is a delicate and effective application for disinfecting disease in the skin. Its chief property and that of the compounds associated with it is the power of destroying vegetable and animal organisms and preventing putrefaction and fermentation in them.

Bichloride of mercury in solution is the most powerful and remarkable disinfectant known to science. Investigation shows that it is a deadly poison to all the lesser forms of life, which it kills instantly when employed in proper quantities. It cannot be used in connection with lead, tin, or copper, owing to its corroding qualities. Its power to destroy germs, even in dilute solution, is unique. In the proportion of one to five hundred it destroys vitality in ten seconds, in one to two thousand in one and one-third minutes, and in one to twenty thousand in from fifteen to twenty minutes. Thus it will be seen that *time* is an element always to be considered when the solutions are used in ordinary proportions.

The fact that corrosive chloride combines with albumen leads to the conclusion that it interferes somewhat with the process of destruction, and this is always to be remembered when using it as a disinfectant.

The following solution is recommended by Sternberg as quite capable of killing all the germs of infectious diseases: bichloride of mercury, four ounces; sulphate of copper, one pound; water, one gallon.

Dr. Parsons recommends the following solution as suitable for clothing, excreta, etc.: bichloride of mercury, half an ounce; hydrochloric acid, one ounce; aniline blue, five grains; water, five gallons. Mix. This is the standard Solution No. 1 of the Public Health Association, and has been tested thoroughly. It should be labelled "poison."

SEWAGE.—A large number of substances have been proposed for the disinfection of sewers, cesspools, and water-closets. The mercurial mineral salts are not applicable to sewers or drains, owing to the fact that they corrode the metallic pipes. Carbolic acid, crude, dissolved in water in the proportion of one to fifty, is a very efficient arrester of decomposition in drains and cesspools, and is in every way preferable. Calvert's powder, composed of about twenty-five parts of carbolic acid and seventy-five parts of alumina and silica, is applicable to the same conditions of use as quicklime.

The solid absorbent disinfectants, such as dry earth, charcoal, sulphate of iron, chloride of zinc, and chloride of lime, will be found to be efficient in drains and sinks under ordinary circumstances. It should be strictly enjoined, however, that under no circumstances are the dejecta of yellow fever, cholera, and typhoid fever patients to be emptied into sewers or privy-vaults until they have been thoroughly disinfected by the standard solution of mercuric chloride which has been recommended. Filth such as is found in closets, sinks, and privies, it should be remembered, is the chief source of nutrition for the different disease-germs, and therefore such places cannot be too clean nor avoided too widely. Let them be cleaned out often, after being disinfected; and do not allow accumulations to take place under any circumstances, always remembering that bad odors are the best, if not a certain, indication that something is out of order and that there is danger ahead.

Sternberg advocates a dilute chloride-of-lime powder composed of one pound of chloride of lime and nine pounds of plaster of Paris. This is clean to handle, and can be sprinkled over everything fearlessly.

Perchloride of iron is useful for the disinfection of sewage, and when added to it throws down a precipitate of ferric oxide, which is due to its action upon the sulphide of ammonium nearly always present in sewage. It is conjectured that this reaction leads to the liberation of sulphur, which in turn acts as a disinfectant.

Permanganate of potassium prevents putrefaction in sewage for a short time, and also acts as a deodorant; but it is necessary to use it in large and expensive quantities to get these results, and hence it is not practical.

SCHOOL-HYGIENE.

By D. F. LINCOLN, M.D.

THE subject before us is readily divided into the two branches of personal and external hygiene. Under the former we shall consider the facts of good or poor health in the individual, his habits and occupations as a scholar, and their influence upon his development. In doing this, the resources of medicine and educational science should be combined. The second part of the paper will include such architectural and engineering details as seem useful in establishing external conditions favorable to health and a normal development.

I. PERSONAL HYGIENE.

It is obvious that we must here exclude from consideration a great number of matters which apply equally to all children, whether inmates of schools or not, and that we must limit the discussion to such things as are specially under the influence of the school and its work. Under this limitation we find that the subject unavoidably presents itself in the form of a list of complaints: it might with equal propriety be termed a "School-Pathology."

The affections which originate in school-influences may be roughly divided into those in which overwork is, in a general way, the chief cause, and those which are not so caused. Overwork and depressed vitality are largely responsible for the following complaints in school-children: dyspepsia, headache, nervous derangements, chorea, epilepsy, neurasthenia, backache, menstrual disorders, and, in some cases, consumption. Spinal deformities, and diseases of the eyes, are conveniently treated apart from this list of affections. The subject of physical education, and a discussion of school-programmes of daily work, conclude this part of the subject.

AILMENTS FROM DEPRESSED VITALITY.

Physical debility, in one form or another, is plainly a chief cause of the ailments enumerated below, although in some cases it is necessary to allow largely for the presence of other causes, as in the case of eye-diseases and spinal deformity.

The public schools of this country have often been accused of overworking the pupils. In the discussions of this subject it has naturally happened that physicians have been prominent as accusers, while, just as naturally, many teachers have shown a tendency to excuse. Some of the most eminent educational authorities, however, have been most severe in their comments on the existing conditions of school-life.

It is to be feared that the schools of Boston set the example of "high-pressure education" to the rest of the country. Such is the testimony of the late John D. Philbrick,[1] so long superintendent of the schools of that city, who gives a striking picture of the way in which it was introduced.

According to Philbrick, the schools were in a lethargic state previous to 1845, when the influence of Horace Mann aroused them. At that time the oldest classes of the grammar-schools (about the age of fifteen) were first subjected to *competitive written examinations.* This woke them up to an intense activity; "the highest kind of high pressure was inaugurated in a day." The competitive examination was discontinued after a few years, but the fear of it survived, and efforts were constantly made to revive it.

Examinations at all stages, and very frequently repeated, are a characteristic of recent plans of education. They are extremely useful, in fact. But they are worse than futile when instituted merely to make a visible showing for work already well done, or when a child who has been faithful for a year is kept in needless but anxious suspense about his promotion. Examinations have also a habit of coming at the close of the spring term, when everybody feels tired; some schools wisely suspend other work, to give as much ease as possible, at that time. Much of the English experience with competitive examinations goes to show that they exert a most noxious influence upon the youth who are being bred up by the forcing process for the purpose of passing them, in hopes of getting appointments.

Measured by the standard of the German schools, our children do not have much overwork to complain of. Ten hours a day, study and recitation, is a common requirement in gymnasia (classical schools) for boys of ten to fourteen years of age; with us the work done in "high schools" from the age of twelve to eighteen varies from six to about seven and a half hours for average pupils; in colleges it is about eight hours, and at West Point and Annapolis nine or ten at most. These requirements for American schools are not excessive. But it is beyond a doubt that we compel younger children to attend too long. If a child enters a primary school at five, he is kept three hours in the morning and two in the afternoon,—or fully two hours too long for his good. He is kept in, nominally at work, far beyond the period for which he has the power to use his mind at the work. The researches of Edwin Chadwick have furnished us with data governing this point, which have never been set aside. He states that a child from five to seven years old is able to attend to one subject for about fifteen minutes, which should be the length of a lesson; from seven to ten years, about twenty minutes; from ten to twelve years, about twenty-five minutes; from twelve to sixteen or eighteen years, about thirty minutes. The total power of attention for one day is somewhat in proportion to this. It is a disgrace to our communities that they insist on having the little ones sent, more to be taken care of than taught, for the same number of hours that make a banker's day. Every minute in school, after their power of attention is exhausted, is given to forming the habit of inattention, which is clear loss to education. That health must suffer, is certain.

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[1] Education, February, 1886.

The city of St. Louis fixes the age for admission at seven; but there are numerous kindergartens which take younger children. The kindergarten is one of the best charities of modern times; it teaches neglected children habits of neatness, order, punctuality, civility; feeds, washes, and clothes them when necessary; keeps them half a day in an atmosphere of physical purity and health, and must be classed as the best evidence of what schools may do for hygiene. On the other hand, the kindergartens for children of wealthy parents are not wholly free from the charge of over-stimulating their pupils. There is a constant tendency among the new teachers to urge and arouse children who are doing well enough already: I quote the words of a very experienced senior teacher. Some children, in fact, are too much aroused, and have to be removed; but the danger is understood; and, on the whole, these children also receive moral lessons that are of inestimable value.

The fault of the old-fashioned school was in neglecting the pupil's understanding of the subject and his interest in it. These points have now been so thoroughly studied that it seems as if lessons had become far too interesting for some children. A bright boy, making no progress in a common school, is transferred to a "Quincy" school and becomes devoted to study, but he has to be taken out every few months to rest his brain. The teacher ought of right to be taken out for the same purpose, but she holds out—by the aid of coffee.

A very exaggerated notion is entertained by some parents regarding the value of primary work: as if children at the age of five could be said to be students in the proper sense of the word. Schooling at that age means something radically different from what comes later. In the words of W. T. Harris, "We do not look so much to the gain in intellectual possessions as to the training of the will into correct habits, during the years previous to the seventh."[1] In protest against the popular delusion about *losing no time*, there is an occasional expression of individual will, like this: "I kept my little girl out of school till she was eight (or ten) years old, and now she is up with the rest." There is a certain number of bright excitable children who are benefited by this postponement of school-life.

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[1] St. Louis School Report, 1872–73, p. 18.

A general view of the state of our schools has often been attempted; the results arrived at have not been entirely concordant, but all reports show the nature, at least, of the evils complained of, however widely the estimate of their extent may differ.

As suggestive of the state of things at present, the writer gives the results of two inquiries, one favorable, the other not.

The first case is that of the high school at Cleveland, Ohio, investigated by authorized medical authority in 1881.[1] It is stated that nearly seventy-five per cent. of the girls had left school on account of ill health, or partly so, and that thirty-three per cent. of the boys were compelled to leave on account of physical troubles. Ill health increased almost uniformly in proportion to the amount of outside study, and inversely to the amount of recreation indulged in. The following table divides the girls into four classes, according to the number of hours of study.

Statistics relating to the Health of the One Hundred and Eighty-Six Girls belonging to the Cleveland High School.

STATE OF HEALTH.		HOURS STUDIED OUT OF SCHOOL.			
		Under two.	From two to four.	From four to six.	Over six.
Health poorer while at school . . .	per cent.	29	70	93	100
Health very poor while at school .	“	14	40	66	100
Appetite deranged while at school .	“	7	44	47	69
Sleep deranged while at school .	“	7	18	37	69
Troubled with headache while at school	“	22	62	81	100
Troubled with back- or side-ache while at school	“	14	34	57	45
Troubled with weak eyes while at school	“	29	39	36	42
Eyes failed since entering school .	“	14	13	18	17
Menstrual symptoms were known .	number	5	40	31	9
Menstruation as usual while at school	per cent.	80	50	19	67
Menstruation deranged while at school	“	20	50	81	33
Menstruation frequent or profuse while at school	“	20	28	26	11
Menstruation scanty, irregular, painful while at school	“	. .	35	71	33

“The parents of seventy-six of the one hundred and eighty-six girls attributed their trouble in part to the stair-climbing. The irregularity of meals necessitated by the one-session system, the worry about rank and examinations, were arraigned as causes by others.”

The remark of Dr. William Goodell, of Philadelphia, has the same tendency: “So commonly do I find ill health associated with brilliant scholarship, that one of the first questions I put to a young lady seeking my advice is, ‘Did you stand high at school?’ ”

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[1] See New York Medical Record, November 12, 1881.

The case of the young lady who read one hundred and ten bound novels in six months while attending the Hartford high school shows that the blame is not all on one side.

The more favorable view is taken by President William De Witt Hyde, of Bowdoin College, who has just collected statistics based on the answers of one hundred and fifty teachers and physicians. Of these one hundred and fifty, one hundred said there was no overwork in the schools of their city; twenty replied, "Not generally, but in individual cases;" ten replied, "Some nervous high-school girls;" fifteen did not answer directly; while only five (four physicians and one teacher) found overwork in school a prevalent evil. Nearly all attributed whatever evil there is, not to the amount of work required, but to the worry and anxiety occasioned by an iron-clad system of grading by too frequent and severe tests (*i.e.*, examinations: compare Philbrick's statements).

It is likely that these respondents are correct in denying the frequency of overwork, if that be measured by the number of hours. But the effects of *anxiety* are worse than those of carrying heavy loads.

It is a universal complaint among teachers that girls ruin their health by social dissipation. The complaint is justified by the facts, and it applies to almost all ages in school. The fault is in the age we live in, which exacts too much and too early display, and expresses the height of its contempt by the word "slow."

The parents of school-boys and school-girls ought to consider that "society" has not yet begun for them,—that school is entitled to their entire strength; in return for which, the school ought to see that the children grow into the possession of firm health. Many boarding-schools deserve praise for their success in this matter, and it often happens that children of rich and indulgent parents are never quite well except when at boarding-school, where regular hours are kept and sweetmeats are not allowed to be received from home. A return to old-fashioned, English notions about the value of play seems to be making, also, in boys' schools. But it is the girls that give most anxiety, because of their readiness to undertake double tasks.

The higher schools of Copenhagen have been recently made the object of research by Hertel, in an important contribution, since translated into English. The result in figures is as follows:

	HEALTHY.	SICKLY.	IMPERFECT REPORT.	TOTAL.
Boys	1900	978	263	3141
Girls	644	477	90	1211

In girls the percentage of the sickly increases from the age of twelve, equals that of the well at fourteen, and exceeds it by ten per cent. at sixteen. The term "sickly" includes anæmia, scrofula, nervousness, headache, nosebleed, curvature of spine, eye-disease; also consumption, organic weakness of the heart, etc.

The boys' work is six hours a day at seven years, rising to ten hours, often more, at seventeen and eighteen. The girls at eleven years work eight hours; at from fourteen to sixteen, about nine hours, or rather more than the boys.

The work of Hertel shows a state of things which is probably not unlike what is found in a large part of Germany.

DYSPEPSIA.—This is an established national trait of Americans, and a familiar symptom of overwork at school. The first point to be noticed here is the fact that it is not (as seems to be popularly supposed) a local trouble, to be cured by some doses of medicine, but a symptom of general want of force in the system, to be cured by fresh air, exercise, food, sleep, and good regimen in general.

The school is responsible for dyspepsia, in some cases, by interfering with the pupils' opportunities for regular meals. The old plan gave two hours of free time at noon, during which children had a good dinner; the new plan, already introduced into high schools and beginning to creep into those of lower grade, keeps the child from nine to two o'clock, with no food except the wretched "lunch" of cake and sweets, dismissing him in an exhausted state from the day's work, to seek for more cake or pie in the cupboard, or else to await the family supper or dinner with what patience he may command.

The privilege of having a whole afternoon to one's self is so highly esteemed that we shall not probably see a return to the old plan. A modification, however, by which an hour's recess is given midway in the session affords ample time for the consumption of a proper lunch, as is the custom in one of the Chicago fitting-schools.

Children often lose appetite during the course of a school year, nor is this always evidence of overpressure, but sometimes of mere confinement to the house and want of exercise. Some, particularly girls, have no appetite for breakfast: they must not be allowed to indulge this want of appetite. Very many think it worse to be tardy than to lose a breakfast: they perhaps are lazy at times in the morning, or have been up late at an entertainment; or they may live a great way from school, and may leave home before the family are quite ready for breakfast. Many teachers notice children occasionally coming to school in a famished state from such causes; it is a duty to send them home at once, with advice.

Other children there are in whom this failure of appetite is a warning to investigate the day's doings. It is fully as bad when children acquire a habit of depending on a cup of tea or coffee at breakfast.

HEADACHE.—The causes of headache are as various as those of dyspepsia.

If there is distinct excess of mental work, this will often produce increased irritability of the brain, and disturbances in the circulation of that organ. There is not a very great amount of this overwork in our own schools, perhaps. In Germany, and in the countries that have formed their educational systems upon her models, excessive study is the rule, and the result is coming to light in some of the more recent statistical reports, as well as in a general popular protest against the cruel exactions that are made.

Guillaume mentions headache by the name of "céphalalgie scolaire." He found two hundred and ninety-six among the seven hundred and thirty-one pupils of the Collège Municipal at Neuchâtel who suffered frequently from headache. The girls suffered twice as often as the boys; the younger children were also great sufferers. Becker found among three thousand five hundred and sixty-four pupils at Darmstadt twenty-seven per cent. suffering more or less from headache; in the highest class of the gymnasium over eighty per cent. complained. With headache Guillaume mentions the frequency of nosebleed,—a complaint which may perhaps be disregarded among us.

Dr. J. Crichton Browne has lately given statistics taken among the half-fed inmates of the London Board schools. He draws a strong picture of the unhappy, under-vitalized existence of this class, but it remains open to some doubt whether the blame should be laid so heavily upon the school-work as is done by him.

Prof. N. J. Bystroff, of St. Petersburg, has found headache in five per cent. of the children at the age of eight, rising to from twenty-eight to forty per cent. at from fourteen to eighteen years. He examined seven thousand four hundred and seventy-eight children of both sexes.

The headaches suffered by hypermetropic children are cured at once by suitable (convex) glasses, and by no other treatment. The public ought to become aware of this rather common class of cases. The patients are literally unable to accommodate their eyes for reading without hurtful efforts.

NERVOUS DERANGEMENT.—The term nervous derangement covers a wide field. One of the common forms among school-children is sleeplessness, or restless sleep disturbed by dreams. Chorea is brought on in some predisposed children by school-work. Either of these conditions should give instant warning.

A fair statement of the general condition of city public-school children has been given by Dr. C. F. Folsom:[1] "Pale faces, languid work, poor appetite, disturbed sleep, headache, and what is vaguely called nervousness, are more common among them than they should be among children of their ages. I doubt whether there is an exaggerated prevalence of manifest or well-marked diseases of the nervous system among them. If due to the school-drill, my impression is that they come for the most part later in life, after the children have left school, and because of constitutions weakened during the school-years, instead of strengthened, as they should be."

The impression which the appearance of city school-children made on the writer, when a visitor, precisely corresponded with the above statement.

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[1] Six Lectures on School-Hygiene, Ginn & Co., 1885.

Some children are extremely sensitive to the influence of their comrades. They are unfit to mingle in a crowd; they lose the power of expressing themselves in reciting; their manner betrays mental pain and constraint. The rigid air of discipline in large schools keeps many in an unnatural state. Young girls entering college sometimes suffer greatly from being obliged to live in the midst of crowds, with sò much less of personal freedom than young men enjoy under like circumstances.

CHOREA (St. Vitus's dance) is mainly a disease of the time of bodily development: the greater part of the cases occur from the sixth to the fifteenth year, which includes the second dentition and puberty. It belongs to the class of diseases which may spread by psychical contagion among children. Among the first symptoms is a change of temper from cheerfulness to fretfulness or apathy, and along with this the powers of attention and memory fail in a way which the teacher may be the first to notice. A child suffering from chorea, therefore, is unfitted for associating with school-children or performing school-work; and the first step in the treatment must be to remove it from school and stop all head-work at home. There is a certain number of children who possess a predisposition to complaints of this sort, and are not fit members of ordinary schools, with the strain on the faculties which seems a necessary attendant upon our system of large classes and fixed tasks. Such children may develop well, if educated quietly and with much open-air freedom.

EPILEPSY.—Children liable to attacks of epileptic fits are not proper inmates of school-houses. They are often backward, or even feeble-minded. They are usually peculiar in temper,—easily excited and falling into ungovernable rages, given to lying, and licentious. It is impossible to manage them by the ordinary course of discipline; they are not understood by the average teacher, and do not belong in the common school, but in those special establishments where their nature is understood. Besides the moral danger to which their presence exposes the scholars, the occurrence of an attack in the presence of young people is a thing to be greatly dreaded. Fright is a recognized cause of epilepsy in well persons; and a person in a fit is a spectacle quite ugly enough to frighten others into fits. Add to this the facts that childhood is eminently susceptible to nervous impressions, whether of fright or otherwise, and that most cases of epilepsy originate in childhood.

Of epilepsy as a possible consequence of overwork in school, little may be said. The connection "is not proved, but we cannot wholly reject the possibility of it."[1]

NEURASTHENIA, OR BREAK-DOWN.—A complete failure of strength, bodily and mental, is sometimes the reward of excessive zeal in study. A partial break-down, implying a year's semi-illness, with a recovery to one-half the former working-powers, is rather common. High schools, normal schools, and colleges furnish the cases. The following one[1] is peculiar, because it seems to point to defective mental training as a cause.

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[1] Nothnagel, in Ziemssen's Cyclopædia.

A young man of good antecedents and physique entered a normal school. His memory was good, but he could not grasp the principles of the instruction, which was intended to develop judgment in teaching rather than to cram with information. He failed in his work because he could not make memory take the place of judgment; spent protracted hours in listless endeavor; became dyspeptic, lost muscle, developed what seemed to be hysterical symptoms, had headache and neuralgia (unknown before). In a few months he went home. Several years of rest and light work on the farm did not wholly restore him, and he still complained, "I am tired."

Break-down is notoriously common in young women, and excess of work or some other distinct cause is usually traceable. It may occur shortly after the graduation, rather unexpectedly.

BACKACHE.—This is not the name of a disease, but is used here as designating a class of cases described with admirable vigor by Haward,[2]—cases, not of spinal disease, nor of uterine disorder, but of over-fatigue.

"It is very common," says Haward, "to see cases in which such symptoms (backache and weakness of the spinal muscles) are the more obvious evidences of over-fatigue, and in which a careful examination will reveal other signs of the same evil. This is especially the case with young girls of feeble circulation (evidenced by their cold hands and feet), whose enthusiasm for work is in excess of their physical powers. They rise early, study before breakfast, sit long hours before the piano or easel, or attend long and frequent religious services, retire to rest late, take insufficient or innutritious food, and still further exhaust themselves by irregular and fatiguing exercise undertaken with the idea of 'working off the effect of over-study.' At last comes the break-down; the poor girl, who has been cramming into one day the work of six, and who has been held up by the fond and foolish mother as an example of industry, piety, and intellectual excellence, finds herself exhausted and ill. She cannot sit upright, her back aches terribly, her brain feels weak, and in her depression and anxiety she thinks she has some serious spinal disease."

DISORDERED MENSES.—Painful or irregular menstruation is to be classed with neuralgia, anæmia, headache, and the like, as a symptom of over-pressure.

In 1873 a work was published which attracted universal attention in America, attacking, as it seemed to do, some cherished features in American education. The book, Dr. Clarke's "Sex in Education," was certainly written with the intention of stirring up discussion; and it succeeded. The author affirmed that he had seen vast numbers of women whose health had broken down, as he thought, owing to neglect of the menstrual function at the formative period, and especially owing to over-study, or, rather, uninterrupted study, during this period. The purpose was to show that girls needed special care while the menses were developing; that the healthy performance of the function is so important that no sacrifices are too great which further its proper establishment; that for many girls hard study was one of the worst things that could be done during the monthly periods; that a rest was imperatively called for by nature, and must be granted "for a single day, for two or three days, or half-work for two or three days." The inference drawn from these important truisms was that girls cannot stand the strain of working side by side with boys in high schools and colleges, where it seems necessary to treat all alike on every day of the year. If Dr. Clarke had refrained from certain galling expressions, the tone of the rejoinders would have been milder; but his end was attained, and the public was the gainer from the prominence given to the question.

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[1] W. H. Rouse, in Michigan Board of Health Report, 1880.

[2] Treatise on Orthopædic Surgery, p. 148.

The replies made to Dr. Clarke showed that many women are, apparently, complete exceptions to his rule in regard to an absolute need of rest every month. One such exceptional lady, in her book, incidentally states that *she* has stood ten hours a day in a store for five years, without the least trouble; a remark which may be improved by us as the occasion of proposing a new rule for mixed high schools,—namely, that in the upper classes *girls ought never to rise in recitation.* Opinions may certainly differ on the point of rules for school-government, but at this day there are many masters who are accustomed to make allowance for girls, even to the extent of never giving penalties for absence. If this does not meet the case, the remedy ought to work through the feminine tact of assistants. A little want of tact may spoil the arrangement; a word of complaint about frequent absences may appeal to the pride of the girls in such a way that they will accept no more favors. It was Clarke's opinion that girls could get through as much work as boys, on the whole, "working in their own way."

The principal of the St. Louis Normal School states[1] that "excuses relieving the students temporarily from work are granted, whenever asked for on account of sickness, without further explanation; and I may say that it is my belief that this privilege has been of great advantage." The school in question is exclusively for young women.

CONSUMPTION.—There is reason for believing that this disease is rather frequently caused by school-influences, though it would be hard to say how frequently. The story of the cases brings to mind those of "general breakdown,"—the familiar "nervous prostration;" the general list of causes is the same,—bad air, continued overwork without rest for repair of waste,—and the difference in the effect is doubtless largely due to hereditary predisposition.

Bad air is notoriously influential in causing consumption. "The impure condition of the air of our houses, be they factories, public buildings, or dwelling-houses, has much to do with the great prevalence of such diseases as phthisis pulmonalis, bronchitis, and pneumonia, which together make up nearly one-quarter of the total mortality. . . . Unventilated and crowded workshops and schools are, moreover, the nurseries of strumous diseases in general, which sap the strength of the community."[1]

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[1] Report of Public Schools for 1878–79.

The following observations by Parkes[2] bear upon this point: "Usually a person who is compelled to breathe such an atmosphere [vitiated by respiration] is at the same time sedentary, and perhaps remains in a constrained position for several hours, or possibly is also under-fed or intemperate. But, allowing the fullest effect to all other agencies, there is no doubt that the breathing the vitiated atmosphere of respiration has a most injurious effect on the health. Persons soon become pale, and partially lose their appetite, and after a time decline in muscular strength and spirits. The aëration and nutrition of the blood seem to be interfered with, and the general tone of the system falls below par. Of special diseases it appears pretty clearly that pulmonary affections are more common. Such persons do certainly appear to furnish a most undue percentage of phthisical cases; that is, of destructive lung-disease of some kind."

Carmichael (1810) and Neil Arnott (1832) describe cases where the diet of schools was excellent, and the only causes for the excessive phthisis were the foul air and the want of exercise. In fact, medical testimony lies all in the same direction. The presence of dust in the air is a very frequent cause of bronchitis, asthma, or pneumonia in various trades, and ought to be mentioned here. Consumption has been a terrible scourge to the British and other foreign armies, chiefly owing to the impure air of the barracks. The same is true of the navy; of prisons in general; of monkeys in menageries, and other unhappy confined creatures.

In America the experience of Prof. H. I. Bowditch as a specialist has led him to utter repeated warnings to the misguided parents who urge their weakly children beyond their powers. The following quotation[3] outlines the history of this class of cases:

"A young person, male or female, walks into my study for the purpose of consultation in regard to health. I observe great paleness of face, extreme emaciation, and trembling steps, combined with a slight cough, and evidently more or less difficulty of breath. These, if combined with a certain intellectual expression of the face, almost immediately enable me to foreshadow a history somewhat as follows:

"Stimulated by ambition to be in the front rank of scholarship, and desiring, owing to poverty or the known wishes of parents, to obtain rapidly an education, the poor, scarcely developed child has been laboring for months, always overworking intellectually, and at times also physically.

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[1] Fox, Sanitary Examinations of Water, Air, and Food.

[2] Hygiene, 1873, p. 115.

[3] From article in the Youth's Companion, August, 1880.

Perhaps the victim has been carried many miles daily to and from school. Study at school, in the cars, and after return at night, sometimes twelve to fourteen hours, has been the daily rule. [In one case the girl went forty miles daily to and from school, in all weather, winter and summer.]

"Of course, utter prostration is the result. The appetite fails or becomes capricious under severe fatigue and irregularity of meals. Gradually a cough is noticed, and it is thought 'a cold has been taken.' . . .

"The cough and the educational race continue on together. Finally a failure of strength manifests itself, and then, for the first time, the parents begin to look with concern at the appearance of their child. But neither child nor parent thinks of giving up school. Perhaps it is towards the end of the term. 'Only a few weeks more,' and the coveted prize will be gained, and then rest and cure can be attempted. . . . At last the long-wished-for goal is reached. The first honors are gained, but they are now of little comfort, for all strength, which has been artificially kept up by the excitement of the race, suddenly leaves the patient, and the pupil does nothing afterwards. . . . All ideas of cure, or even of partial relief, have disappeared." . . .

It is important to note that the blame for such abuse of life is placed upon the parents, rather than upon any stimulus supplied by the teachers.

If a child has hereditary tendencies to consumption, it is imperatively necessary to select "a proper, reasonable, well-ventilated school (especially one in which the frequent opening of windows is avoided). No overwork of mind or body should be permitted. If the health fail at all, absolute removal from school is required; travel or anything else should be undertaken that will interest and keep the pupil from books and out of doors, and let the education, so called, take care of itself."

In another place[1] the same eminent authority remarks that "in a consumptive family the steadfast rule should be, *that the mind be wholly subservient to the body's welfare.*"

In a report on the causes or antecedents of consumption,[2] the opinions of two hundred and ten correspondents were summed up as follows. The question having been put, "Is consumption ever caused by over-study at school or college?" the answer "yes" was given by one hundred and forty-six; "yes, indirectly," by seven; "no," by twenty-one; "doubtful," by ten; and twenty-six gave no answer.

The circumstance that residence on a damp soil is one of the most powerful predisposing agents to consumption ought to have its influence when the site of school-houses is selected.

Here we may end the description of the ailments commonly grouped together when the effects of overwork are described. Spinal deformity and near-sight are susceptible of being classified with these, for weakness is an element in both. It is more suitable, on the whole, to describe them separately, on account both of their special importance and of their mechanical relations to desks and seats.

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[1] Massachusetts Board of Health, Fifth Report, 1874, p. 50.

[2] Ibid., Fourth Report.

SPINAL DEFORMITIES.

Under this heading belong two distinct affections,—lateral curvature and round shoulders. With the latter is associated the "hollow back" or excessive curve at the loins. Lateral curvature is the object of our present study.

Lateral Curvature, in the popular eye, is an inelegance of person, surmountable by the aid of the dress-maker. The physician sees in it a consequence and a cause of low vitality.

The great majority of cases of this curvature originate in children from the age of five or six upward, and in young persons who have lately been in school. This might be thought a mere coincidence; for the school-period is necessarily the period of development, and the curvature is a disease of development. But there is evidence that school work and customs are genuine causes,—not by any means the sole causes, but rather prominent ones.

The origin of lateral curvature depends chiefly on two things,—weakness of the muscles which support the spine, and bad positions of the body. Weakness, however, is not a necessary circumstance, though an extremely common and often important one. A bad position constantly maintained will twist the most athletic frame. A very muscular person may be forced to stand in a one-sided position by the circumstance of having one leg shorter than the other. Fig. 1 gives a rough but sufficient idea of the way in which the body is supported on the legs. If the right leg is shortened (Fig. 2), the pelvis or hip-bone will be tilted to the right, and the lower part of the spinal column, being firmly attached to the pelvis, will tilt with it. If the whole spine remained straight, the person would be thrown off his balance; the spine must therefore curve to the left at a higher part; and furthermore, to make up for the overweight thrown to the right below, there occurs a "compensating" curve to the left at the height of the shoulders. In addition to this, the spine is in parts twisted, with a gimlet-motion; but this is not represented here.

Fig. 1.

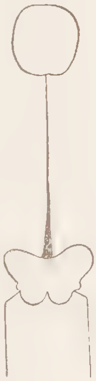
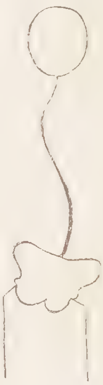


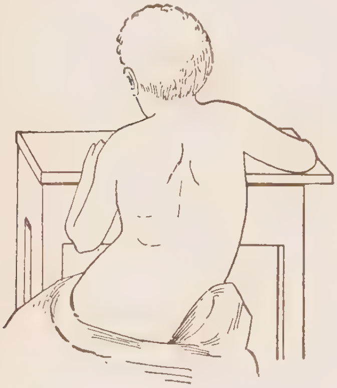
Fig. 2



A similar thing happens when children stand on one leg,—a position which practically shortens the other leg. "A most pernicious habit, and one which is very often to be noticed in school-girls (I think I have observed it in girls more frequently than in boys), is that while we are talking to them, or during recitations, especially if they are much interested in what is going on, they are standing on one leg. This position is assumed involuntarily, and it is always, or almost always, one and the same leg on which the weight is thrown. The effect of this is easily understood: one side of the pelvis is lifted up, curving the spine in the loins; the opposite leg is advanced in front of the other, twisting the pelvis and rotating the vertebræ. Of course the curve of compensation takes place between the shoulders. One is depressed, the shoulder-blade gradually projecting, and with the change, and in fact assisting to produce it, occurs the spiral twist."[1]

Many curvatures begin in the region of the shoulders; of these, beyond doubt, the cause is largely to be found in false positions in writing or drawing. "I have visited rooms in which drawing was taught," writes Brown, "where all,—male and female,—with scarcely an exception, were sitting in a position not only to curve but to twist or rotate the spine, and in most the position was such as to produce a triple curve." The deformed attitudes (so to speak) assumed in writing are powerfully described by Liebreich. Such occupations can hardly be conducted in entirely normal postures, but a great deal can be done to correct the grosser faults. One cause of the defect is the raising of the right shoulder by a high desk or table, as is seen in the sketch (Fig. 3) borrowed from Guillaume.

FIG. 3.



Another frequent cause exists when the desk is too far from the seat, and the pupil is forced to bend over his work in an unbalanced posture, which cannot be maintained; in a short time, if not at once, he leans one side forward, puts elbow on desk or knee, and head on hand, and gets his spine into the shape of a corkscrew.

It is desirable that every person directing or teaching a school should have a notion of the proportions suitable for desks and seats. Farther on, this matter is illustrated with a few representative figures. But it is of the greatest consequence to remember that no seats or desks can be devised which will remove the *original weakness of muscle,* which ranks as one of the two chief causes, and would by many be named as the one important cause. Children cannot be made strong by supports.

The part which weakness of muscle plays in developing spinal curvature is plainly shown by the class of persons most affected,—namely, girls and women. Adams (1865) gave one hundred and fifty-one cases among the female sex, and twenty-two among males; Knorr (1860) gave sixty among females, and twelve among males; Klopsch estimates that from eighty-four to eighty-nine per cent. are females. These are among actual patients. But the disease exists far more widely than any one is likely to know. Guillaume (in 1864) found in the public schools of Neuchâtel, among three hundred and fifty boys, eighteen per cent. affected; and among three hundred and eighty-one girls, forty-one per cent.

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[1] Buckminster Brown, Lecture before the American Social Science Association, 1879.

That muscular weakness is a chief cause is further shown by the treatment which is successful, consisting essentially of prolonged and perfect *rest* to the weak muscles, by reclining, combined with occasional moderate yet active *exertion* of the muscles by appropriate exercises. Cases that are not far advanced may do well by using scientifically directed gymnastics. Some need never be told that they have "anything the matter with the spine," provided they can be got to change their habits of living.

These principles need to be applied to the *prevention* of spinal curvature in schools. The preventive treatment should consist, when possible, of very varied muscular activity of an active sort, taken at proper times. The best kinds are out-door games of an athletic tendency,—leaping, running, coasting, skating, climbing, and all sorts of "hard play." For boys, wrestling, foot-ball, and boxing, and many more, may be added. No doubt, children must be kept from excesses, such as trying to make a high score with the skipping-rope. Some sports have a tendency to develop curvature,—croquet, decidedly; tennis,—when played with one hand; base-ball; horse-back-riding on a one-sided saddle. Ladies should use two saddles, one for each side alternately. Sports liberally indulged in are, with this class of exceptions, the usual and natural preventive of spinal curvature among boys; if girls played out of doors as boys do, they would have little trouble of that sort.

The physical indolence of girls, however we may deplore it, is not to be overcome at once. It arises in part from their unwillingness to assert themselves as boys do; their readiness to submit to custom; and their power of sacrificing comfort (*i.e.*, health) for the sake of propriety. The misguided sense of decorum, which prevents even walking in many cases, is the same feeling that, neglecting certain of the lower functions of the body, leads to the prevalent habit of constipation, and occasionally to disease of the bladder. Every argument, therefore, in favor of bodily training, or of the teaching of gymnastics in schools, should apply with double force to the female sex.

The programme of a girl's life consumes the greater part of the day in sedentary occupations. Sewing, piano-practice, drawing-lessons, embroidery, are among the added burdens of the life of girls at home. If parents cannot be induced to take active steps for their children's physical training (and I fear they cannot), the school must undertake the task, on behalf, at least, of the girls.

The existence of spinal deformity has not yet received due attention from our public. It is hard to get at the facts. There are very few collected observations of masses of children. Parents would dread the exposure of their children; but perhaps the popular mind would not object to a lady surgeon for girls. No figures are procurable from female colleges, though the very great prevalence of curvature is admitted. The late Dr. J. C.

Warren in 1830 stated that of the well-educated females within his sphere, about one-half were affected with some degree of distortion of the spine. It is not a wild guess to suppose that this is nearly true at present.

Spinal curvature is not only a product of low vitality, but it does harm by permanently fixing vitality at a low standard. The spirometric observations of Schildbach (Amsterdam, 1862) showed that the respiratory capacity of this class of children at the ages of from thirteen to seventeen was lessened by one-third, and in some cases by one-half,—a matter of the gravest importance in the maintenance of life.

Brown calls attention to the fact that too much mental stimulus has an undoubted effect as one of the predisposing causes of spinal curvature. Mental rest combined with other appropriate conditions will sometimes cure the trouble in its incipient stage.

The disease may exist in a fully-developed form without any apparent change in the direction of the spine as seen from behind, even when the body is stripped: this is because the twisting may be confined to the bodies of the vertebræ, which are out of sight. The first thing usually noticed is that "the shoulder grows out," or else is higher than the other one. The ribs partake, and the chest is twisted out of shape.

Patients may sometimes be kept in school who need special seats. This is a matter for the surgeon to decide.

DESKS AND SEATS.

The improvement which has been made in American school desks and seats within half a century is very great. Few city schools are now unprovided with "modern" furniture, which in most cases is decidedly better than the old. It is to be hoped that the excellence already attained will not stand in the way of further progress. We have been quite successful in reaching our ideal of *comfort;* but we ought not to forget that the subject has been much studied, from various other points of view, by German and other investigators, and with results which certainly differ from ours.

Bad desks are chargeable with aiding the formation of two of the most important "school-diseases,"—near-sight and spinal curvature. They cause the first by compelling pupils to hold the eyes too near the object, and by favoring a stooping position at work. Spinal curvature is very much assisted by the twisted postures which children take, especially in writing.

Let it be understood that it is not our object to make desks and seats which a scholar can occupy with comfort, assuming and maintaining one "normal" position, for hours at a time; no, nor for one hour. It is not possible to do this; and, if it were, it must needs injure the child. The discipline of a school is a precious thing, but it should not interfere with the child's need for change of attitude; nor must the teacher fancy that in prescribing fixed attitudes he is following the dictates of "medical science." Attitudes assumed for a few moments, for purposes of respect and attention, may properly be formal; attitudes in study should be decent, but may be as varied as possible, subject to correction when they become injurious. The teacher should be a judge of the latter fact. Especially should liberty be given to the younger classes. There is indeed a great difference between the mobility of a kindergarten and the studious self-possession of a high-school class at the age of sixteen or eighteen.

Strength cannot be gained by maintaining any one posture, unless in the sense that a comfortable posture gives strength by resting the muscles. If a comfortable position is given, let the child not be kept in it till rest itself is fatigue.

As standing is undesirable exercise, so is sitting in a chair without a back. It will not make the child's back strong, but only causes fatigue, and drives the child to take all kinds of unsuitable positions for relief.

The young pupil should have a seat and desk so well adapted to its form that it will be tempted to take the most correct position, as being the most comfortable. It will not keep this position long, however comfortable it may be, but it will return to it after making its little excursions and changes, and will by degrees become accustomed to a normal position without much being said about the matter.

Little children may properly be taught to sit still, facing squarely to the front, for five minutes at a time, when circumstances favor it. They may be taught, by degrees, to sit ten minutes, but not through a school-session; every *rest* must be made up by a corresponding *activity*,—a change or a movement, a song or a bit of gymnastic drill. Movement is a child's way of resting: rest is a kind of work, to be taught by degrees.

As regards faulty positions, *stooping* contracts the chest and compresses the abdominal organs. The child, sitting erect, and wishing to bow the head towards the book, may be shown that a very slight movement will accomplish that object,—a hinge-movement at the upper part of the neck, and not at the shoulders. The trunk does not need the support of the elbows on the desk. One-sided positions easily become habitual, and are then exceedingly objectionable: they are chiefly caused by propping the arm or elbow on the desk.

A comfortable back for the chair is best secured, not by giving a series of marked curves intended to follow the natural curves of the body, but by giving, first of all, an emphatic support to the lower part of the spine. The writer was recently shown a rather stiff-looking seat, of which the dealer remarked that all those who sat down in it at first said "no," but if he could induce them to remain sitting fifteen minutes he was sure to sell it. The seat in question has a flat bottom, sloping a little down and back; the back is tilted, and is composed of two flat surfaces set together at an angle so small as hardly to be noticeable,—the projection being one-quarter of an inch from a straight line. (See Fig. 4.) This chair, supporting the pelvis solidly, gives great comfort. A somewhat greater projection of the lower middle part of the back might be useful.

This principle, which the writer is convinced is of the greatest value, may be carried out in other ways. The matter is not wholly settled, as may be seen by comparing Figs. 5, 6, 7, given by different authorities. Liebreich's chair (Fig. 5) is intended to support the pelvis by following its outline by a curve up into the small of the back : the projection of the curve may be too great. Fig. 6 (Varrentrapp's), and the unshaded spaces in Figs. 7 and 8, give the impression of stiffness ; they come to the height of the elbow, and give support solely by a horizontal cross-bar at the top, allowing a little open space below the bar. It is noticeable that many of the later German plans proposed by scientific men give support in this way. Their object is to enable the child to sit erect while writing, with the aid of a partial support, not necessarily used at all moments : it is thought that such a support gives the habit of a correct attitude. Buchner was an inspector of schools :

Fig. 4.

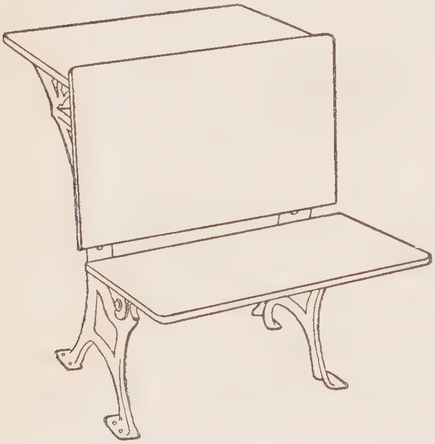
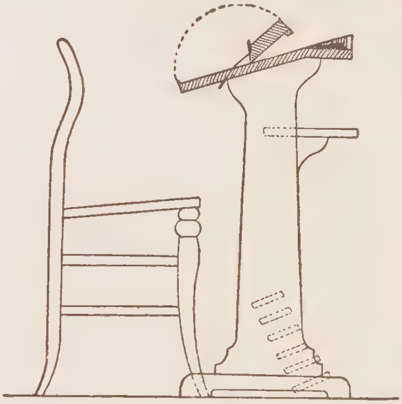
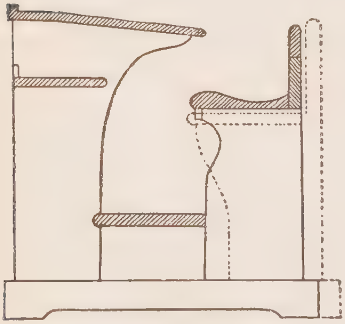


Fig. 5.



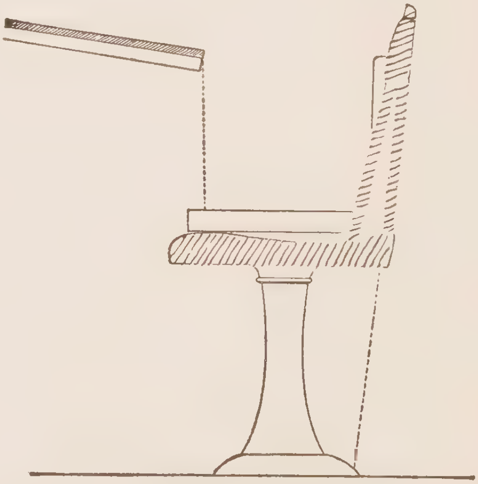
Liebreich's desk and seat.

Fig. 6.



Varrentrapp's desk and adjustable seat.

Fig. 7.



Varrentrapp's seat and desk compared with a Boston school-chair and desk (see Fig. 9) for corresponding ages.

he says, "The children very soon feel that the perpendicular piece supporting the small of the back corresponds with the structure of the body much better than the slanting back which supports the shoulders. I often used to ask the children whether they would not like to have a rest for the shoulders, as well as for the back, but the girls always answered in the negative." Cohn and Fahrner are also in favor of the low support. It is of wood, two and a half to three inches broad, and long enough to be reached by both elbows, which may rest upon it when the child is not writing. It must not be placed too high, or it fails to give due support.

It may be doubted whether a low back-rest of this kind, or a slightly-inclined rest for leaning back, supporting shoulders as well as pelvis, is, on the whole, the better. The present writer is not in a position to decide; yet a choice must be made, since it seems impossible to secure a resting-chair which shall also give support in writing.

The popular American school-seat, with its back curved like a long italic *ſ*, is not an ideal model. It is comfortable,—at least some are, for there are varieties. Some of them let the body slide down so that upright sitting is impossible; others are too low, which tends to increase the constriction of the abdomen; as a rule, they give the body a round-shouldered position, being essentially lounging-chairs with the head-rest cut off. Fig. 8 gives a good pattern.

Our common wooden chair, with the back-sticks set in a curved line, quite fails to support the pelvis; in fact, its deficiency in this respect is a positive annoyance. Fig. 7 shows a chair with this fault. It is built thus to give strength; this is better secured in an old pattern which prolongs the middle piece down to the pedestal, following the dotted line, and so is able to bring the side-pieces in line with the middle piece.

Fig. 5 gives Liebreich's desk and seat, as designed for the London School Board. The desk remains the same; the accommodation for different ages is made by changing the chair and moving the foot-rest. The shape of the seat is slightly different for the two sexes. The lid is hinged so that it can be thrown into a convenient book-holder for reading. In writing, the chair is placed so near that the edge of the desk just touches the body. The height of the seat is correct when the sloping line of the desk, prolonged, just touches the elbows.

Fig. 6 gives the design for desk and seat published by the late Dr. Georg Varrentrapp, of Frankfort-on-the-Main, in the *Vierteljahrsschrift für Gesundheitspflege* for 1869. It is the one from which the unshaded spaces in Fig. 7 are taken. The desk remains the same for different ages; the seats are of different sizes, the dotted outlines corresponding to larger pupils.

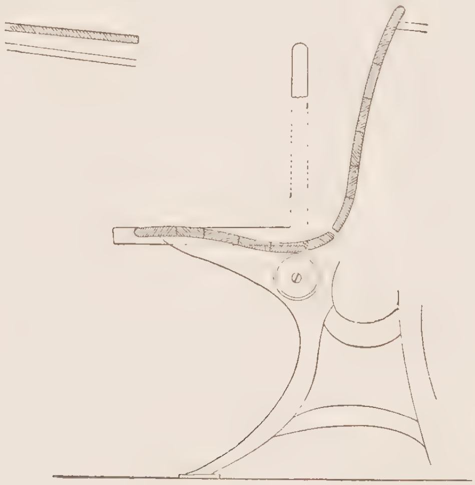
Fig. 7 gives a side-view, drawn to scale, of a highly-approved American school-seat, of a size intended for pupils from ten to twelve years old. The position of the lid of the desk is also given. The unshaded spaces show the position of corresponding parts of the model designed by Varrentrapp, of dimensions suited for children of the average height of one hundred and forty-three centimetres, which for American children represents a little over twelve years. The lower edge of the desk, measured from the seat, is 4½ centimetres = 1.8 inches higher in the American than in the German seat. The German back-rest is on a level with the desk, and the pupil while reading can easily prop his elbows upon it, maintaining an erect posture.

The dimensions, in centimetres, are as follows:

	Height of Seat.	Height of Desk.	Difference in Heights.	Height of Back-rest.	Desk to Back-rest.	Seat projects under Desk.
American	36.9	64.8	27.9	(35)	30	5.2
Varrentrapp	40.2	63.6	23.4	23.4	26	2.6

Fig. 8 gives the side-elevation of two full-sized desk-seats, drawn to scale. The American desk is the largest size of one of the most popular kinds. To correspond with this, Buchner's tables were taken, and the dimensions calculated for a person five feet six inches in height; the lines of desk and seat are given with shading for the latter case. The difference between the heights of the desks is nearly 4 centimetres = 1.6 inches. This difference is increased, practically, by the downward and backward curve of the American seat.

Fig. 8.



American curved-back seat and desk (shaded), and Buchner's pattern, for corresponding ages.

The dimensions, in centimetres, are as follows:

	Height of Seat.	Height of Desk.	Difference in Heights.	Height of Back-rest.	Desk to Back-rest.	Seat projects under Desk.
American	43	76	33	(44)	44	1 or 2
German	42.9	72.1	29.2	32.1	23.8½	5.2

Fig. 9 is the American model from which the drawing Fig. 7 is taken. The fact that the desk-lid is considerably higher than the pupil's elbows is correctly shown. The occupant cannot be supported while sitting upright, and when sitting back his eyes will be too far from the book for ordinary work.

Fig. 9.

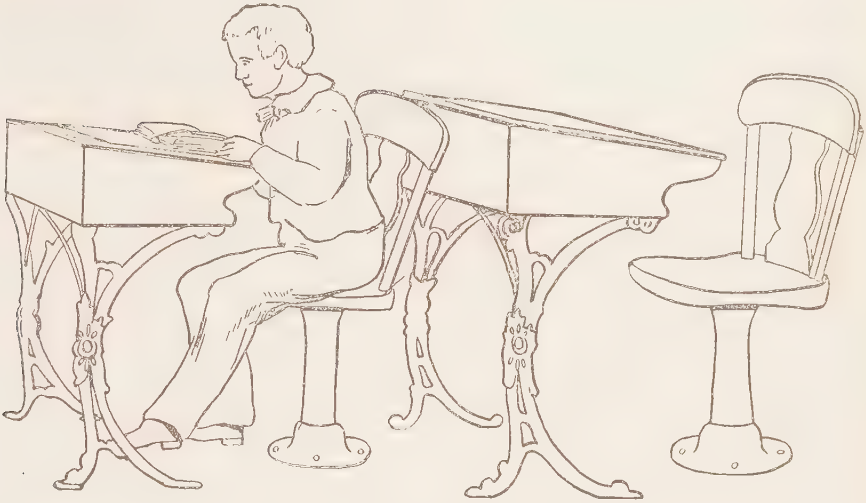
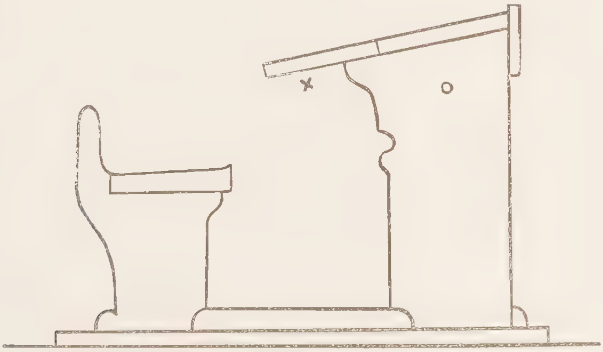


Fig. 10 shows the Belgian model in the Philadelphia Exhibition of 1876. It carries out very well the idea of supporting the sacrum; there is a turn-back lid, and the under side of the hinged part has a cushion at *x* for the use of the girls in sewing; there is a peg for the hat at *o*. The seat, however, is much too far from the desk.

Fig. 10.



Stooping, cramped, awkward positions are not very likely to be assumed in a desk and seat well adapted to the occupant, except in the acts of writing, ciphering, and drawing. Bad postures in these occupations are far worse than in others, from the seeming naturalness with which they are assumed, and the certainty with which they become habitual. If a child can be taught from the beginning to write in a proper attitude, one of the commonest and worst of school-faults will be broken up. If there is to be a reform in this matter, let it begin at the beginning; let the youngest classes be the first to receive the ideal desk (whatever that be), and let the change go on, following their progress in the schools. Time ought to be devoted to acquiring the habit of sitting well, especially at the beginning of school-life.

The correct posture in writing is one which does not twist the body or neck. The pupil faces the desk squarely; the shoulders are equally distant from the lid of the desk; the trunk is erect and nearly touches the desk. The lid must be high enough just to support the forearms, but not so high as to raise them; they rest lightly on the lid, but do not sustain the weight of the body. This posture cannot at first be maintained long. Perhaps five minutes at a time is all that we can expect of beginners; at all events, nothing is more certain than that children begin to lapse from this uprightness by that time. What can be done then? The effort to force fifty children to keep in one position cannot succeed beyond a brief time, and the bad position must not be permitted: hence, as soon as fatigue begins, it is best to give a total rest by letting the children sit back, stand, etc., and then resume writing.

The "systems" of penmanship in vogue do not satisfy the demand here made; they do tend to twist the spine. In some cases the pupil is directed to turn squarely to the left, letting the whole right forearm swing over the desk-lid, on the elbow as a pivot. This raises the right shoulder. In others the direction is to turn partly to the left, or partly to the right. The positions assumed in these cases are almost invariably faulty: one or the other shoulder is raised; the head leans to the right or left, and sinks by degrees until the ear may come in contact with the hand and the nose almost grazes the paper, the spine meantime assuming various curves.

FIG. 11.

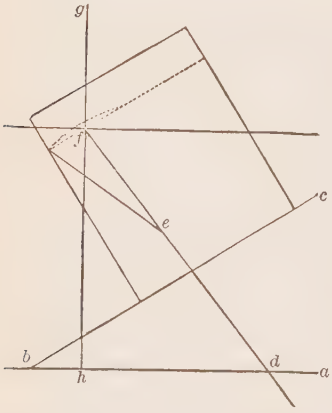


Fig. 11 illustrates the position of the manuscript on the desk. It is copied (with a little simplification) from an article by Staffel in the *Centralblatt für allgemeine Gesundheitspflege*, 1884, p. 45. If the pupil sits in the correct position, facing squarely to the edge of the desk ab, and looking in the direction hg, df represents the axis of the right arm, f being the point of the pen and e the place where the wrist touches the paper. The left hand steadies and adjusts the paper, and must be near the right hand, so that the two forearms point inward and nearly meet at the hands. To correspond with this, the paper is tilted thirty degrees from the perpendicular, towards the left, which enables the hand more easily to follow the direction of the ruled lines on the paper. This tilting of the paper is a natural device,—the reader has probably often practised it without special thought; it is, however, mechanically and physiologically the correct plan for easy writing.

The letters acquire a slant of thirty degrees when the paper is held in this position. This is owing to the fact that the most natural and easy way of making down-strokes is to make them parallel to the line *gh*,—or perpendicular, as the paper is commonly held. Children and blind learners begin with these strokes.

Some additional points require mention:

1. The chair is often too high for young scholars. The most convenient plan may be to provide footstools.

2. The seat, from back to front, ought to be long enough to support nearly the whole thigh. A more or less spoon-shaped hollow in the seat is commonly thought desirable. The curve of many settees is such as to produce pain at the point where the bones (tuberosities of the ischium) rest on the wood: the support is not wide enough.

3. Seats must have backs. The straight upright back reaching to the shoulders is bad; a straight back slightly tilted is not bad. American seats are commonly curved, with curved backs, as in Figs. 8 and 9.

4. The edge of the desks should come up to, or overlap, the edge of the seat. The recognition of this fact is a recent discovery: desks used formerly to be separated from seats by a space sufficient to enable the occupant to rise in his place, but since desks are now made separate or in pairs, it is only necessary to step into the aisle.

5. Most of our best desks are too high, relatively to the seat. The reason for making them high is, doubtless, to prevent the pupil from stooping. Something is certainly gained in reading, by this plan,—at least, in convenience of reading,—but it interferes with correct positions in writing. The elbows, hanging freely, should be only just below the level of the lid.

For near-sighted children, the higher desk may be a necessity in writing. If the desk is made as low as is here recommended, a portable arrangement resembling a writing-desk may be placed on the desk.

DISEASES OF THE EYES.

Near-Sightedness, with some other difficulties, composes one of the most important divisions of our subject. To some extent the production of myopia is doubtless due to constitutional weakness or to depressing causes acting temporarily. Landolt considered hardships and poor fare the leading causes, but his opinion seems an exaggerated one. Loring has argued forcibly in favor of more active sport for growing youth, and has shown how confinement to the house, short hours of relaxation, and undesirable fare, must be considered important causes of the excessive prevalence of near-sight on the European continent.

Other facts which may illustrate this point are the excess of near-sight in cities; the general prevalence of poor health among the same classes of students that are subject to near-sight; the readiness with which the eye may be injured by work performed before breakfast, during fatigue, or after recovery from acute fevers. An associated fact is the low vitality prevalent among blind people,—which may, however, be an effect rather than a cause.

As regards map-drawing, the best plan is to use large paper, make strong outlines, and insert few details, the object being by no means the production of handsome work, but the fixing of leading facts on the pupil's mind. In penmanship and sewing, and still more in embroidery, harm may easily be done.

Interesting remarks upon the most desirable form of type for clearness are made in Dr. Jeffries's article, Massachusetts Board of Health Report, 1882–83: they are from Javal. Many school-books are excellent in this respect; many are still bad.

The pupils' most common neglect (says Soldan) is in regard to their eyesight. They injure the eye by reading by a lamp close to the head, without a shade: the object of the latter is quite as much to keep off heat as light. They work in the evening instead of by day. They read novels six hours and then study two. They defer the most taxing work (such as drawing) to the last minute.

LIGHT.—Defective lighting is one of the chief faults of school-buildings. The difficulty of satisfying the requirements in cities is stated under the head of "Site for Schools." For a northern climate, a very free exposure to sunlight is desirable. Large trees often need removal. If possible, direct sunlight should enter every room at some hour of the day.

A sufficient light implies light which easily reaches the back of the room. Lighting from one side, as practised by the Germans, is thought by most of our architects insufficient. In fact, a room with sixty scholars and an allowance of two hundred and fifty cubic feet of space per head will necessarily be too deep for good unilateral lighting. The simplest remedy is to make windows on one side and at the back. This principle, carried out, gives us the square school-house with four corner rooms on a floor, or, as in the Cleveland model, with six rooms on a floor. It has the advantage of natural draughts. If the combined size of all the sashes equals one-fifth or one-sixth the area of the floor, it is usually said that the supply of windows is sufficient. Small windows are not the fault of modern school-houses.

To get the best effect, windows must reach within a few inches of the ceiling. They ought to have square tops, not the Gothic shape adopted in the St. Louis model; they must have no heavy projecting outside ornaments to cut off light. Instead of Italian awnings for summer, they should be guarded with blinds on the inside. It is hard to find screens that will at once exclude the sun's rays and admit enough light and wind. Neither white nor yellow nor red screens are pleasant when the sun is on them: a neutral gray is best. A neutral light tint is suitable for the walls.

Lighting from both sides is well enough for small school-houses of one room. There is, however, a preference for light coming from the scholar's left hand, especially in writing. Rear windows may be added if thought necessary; they give a general increase of light: their worst point is that they try the eyes of teachers, but that can be relieved in two ways,—either by a rather dark shade rolling up from the bottom, or by placing the windows about six feet above the floor, so that the direct rays do not strike the teacher's eye when looking at the class. It is a cardinal rule that no one shall be forced to face the windows while reading or otherwise exercising his sight: therefore no windows must be in front of the scholars. Blackboards are generally put wherever there is room; those between windows ought to be little used; their surface must be a dead black, not glossy.

This, however, gives but one aspect of a wide question. Other important causes—perhaps much more important—are the following.

Excessive use, even under favorable conditions, wearies the eye. It seems well proved that, in general, students who spend longer hours over home lessons are affected by near-sight in larger proportion. The practice of working without rest for long periods is worse than working many hours with pauses.

Poor light has always been considered one of the leading causes. It not only fatigues the eye, but also induces the pupil to bring the eye close to the book.

Constant attention to near objects doubtless has its effect, even when they are "near" only in the sense of being bounded by the walls of streets. City children live in a narrow horizon. The youthful eye has a marvellous power of seeing things at the distance of two or three inches; and many things in school-life conspire to bring about habits based on this power of adaptation,—badly-proportioned desks, poor type and ink and paper, poor light, excess of light. The eye that is laboring at too short a distance is enabled to do so by the action of the "muscle of accommodation" in the eyeball, which arranges the focus by changing the shape of that organ. Such an eye is working in a state of tension, which tends, if long continued, to produce a permanent change in the form of the globe, making it longer from back to front, which constitutes the chief characteristic peculiarity of the near-sighted eye. Very few, if any, children will obey physiological laws of distance without being compelled to do so. There are certain faults in school furniture that favor the bad habit: too great height of desk relatively to seat; separation of desk from seat by an interval, instead of having the desk partly over the seat. These, and bad positions in writing, have been mentioned already.

Anything tending to cause congestion of the eye aids in forming near-sight. Among these causes are tight clothing (corsets, neckties, collars, belts); indigestion, particularly constipation; overheated rooms, with bad ventilation; overwork of the brain, especially if it causes headache. Fulness of the blood-vessels stretches the eyeball and assists the tendency to a change of shape as above noticed.

All these influences have their effect chiefly during childhood: few persons become near-sighted after they are grown up, though an increase of near-sight is not rare. This circumstance is one of many which warn us that bodily development is liable to be impeded or distorted in every direction by false education. Every plan which aids in strengthening the constitution of children may be considered as a contribution to the health of their eyes. During childhood the tissues are less firm, more elastic, than later in life; they are more quickly renewed, more easily distorted. The geometrical deformity of the eyeball is produced by pressure at this tender age. The remedy seems to require not only that we lessen the daily amount of pressure, but also that we encourage those active habits which will make the fibre stronger and more resisting.

Hereditary influence is undeniably powerful. What may be the effect of several generations of accumulated tendency in studious families cannot be predicted: a learned friend of the writer's suggests that the result may not be blindness, but a permanent type of myopism, in which children will be born short-sighted and will not need to become so. But in speculating about the future it is necessary to take account of other tendencies. Far-sight is quite common among children; and the action of inheritance seems not to be limited to the repetition of identical defects, but to reproduce both anomalies—the long eye and the short eye—with a certain degree of indifference. The existence of a tendency to the normal eye is probable, independently of the elimination of unfit persons from the race.

Blindness, or an approach to it, is the tendency of a certain number of cases of near-sight; the retina becomes gradually detached from the back of the eye, and becomes incapable of receiving exact impressions. On this account (as well as for reasons above given) the "near-sighted eye is a diseased eye."

Test-types, or large cards on which lines of letters of graded sizes are distinctly printed, are a ready means of estimating the degree of near-sight in the hands of school-principals. To give such observations full value, atropine and the ophthalmoscope, in expert hands, are required. A much-used eye is apt to be in a state of tension which makes it temporarily more near-sighted than it really is; atropine relaxes the tension.

There exists a prejudice against the use of glasses, which is natural enough. But if near-sight is considerable, so that a child really cannot work well in an erect position, it is necessary to allow a pair of very weak glasses. The matter cannot be determined by directions given in an article like the present: the decision and choice must be left to the physician. A limit or minimum distance at which the book may be held from the eye should be stated, and children advised and corrected of their bad practices. The least distance, recommended by the Commission d'Hygiène des Écoles of Paris in 1884, was twenty-five centimetres for children in the lowest schools and thirty-three for those from eight to twelve years old,—about ten and thirteen inches respectively. Fifteen inches is proper for those of larger stature, but would be impossible for little children.

HYPERMETROPIA, or long-sight, is not uncommon among school-children. Its effect, when it is of a high degree, is to make the act of reading difficult or painful, and sometimes to put an end to a child's career in school. Those subject to it read fairly well for a time, but after a while become conscious of effort in the act of seeing. The eyes feel strained, and the letters become somewhat blurred. There is a desire to rest the eyes, or to close them firmly, or to compress them with the hand. A fresh start is made, and a second rest has to be taken after a shorter period. Sometimes the habit of holding the book close to the eye is acquired, which makes the case seem to be precisely the opposite of long-sight. The choice of glasses (convex, or old-sighted) should be directed by a competent physician, for the purpose of enabling such children to work without suffering. One effect of the disease is the production of internal squint.

ASTIGMATISM is rather common, and gives as much annoyance as near-sight. It depends on an incorrect curvature of the front of the eyeball (cornea). It is known by producing a blurred look in lines that run in one given direction: some people see horizontal lines badly, some perpendicular ones; in others there is an oblique axis of indistinctness. If at all troublesome, this defect ought to be remedied by glasses,—as it can be perfectly. It is not a disease in the sense that near-sight is.

PHYSICAL TRAINING.

The connection between physical training and general education is obvious. The principle being granted, it remains for us to consider how much the school and the college of to-day should be required to give of their energies to the furthering of this end.

A system of calisthenics is at present widely used in public schools, with distinct benefit. It is not probable that the system will ever be abandoned. Most readers must have seen the pleasant sight of a roomful of children engaged in the simple but vigorous movements of the arms which serve so well as vents for superfluous energy. These trained movements are an indispensable part of primary-school work, and are of great use in the intermediate grades, but are of subordinate value (as now practised) for older pupils. They are quite difficult enough for little children, but above the age of twelve scholars begin to look down on them as childish, and with good reason, for they lack one essential element,—they do not call forth exertion to overcome resistance. For better work, scholars should have light dumb-bells and wands, and more space to use them than can be found in an occupied room.

The immediate benefit of exercise, however, does not depend on any large amount of development that it imparts. Very simple exercises, without any apparatus, practised twice a day or oftener for five minutes at a time, do a great deal of good. Far from breaking up the discipline of the school, they make it easier, by relaxing the tension of mind and by introducing the element of pleasure. The eyes are relieved at the same time. A piano adds pleasure, but is not essential. The windows should be opened at the instant the signal is given, by having one boy assigned to each.

The facility with which calisthenics are practised should not blind us to the imperfections of the system. The movements employed are limited to such as can be safely made in a room full of desks: the pupils ought to have a much wider range of action, in an open hall, large enough for fifty or sixty to exercise in.

A good type of light gymnastics adapted to use by classes is furnished by Amherst College. In that system every student is required to attend unless physically unfitted. The work is done in classes, to the sound of the piano, under a leader; students attend four days in the week, half an hour at a time. Most of the work is done with wooden dumb-bells; there is also considerable running, and some marching. The exercises are eminently cheerful; compulsory attendance meets with the acquiescence and support of almost all the students. In fact, the exercise is a union of recreation and amusement with work.

This moderate amount of exercise is sufficient for nine-tenths of the men. For those of unusually muscular frame, heavy gymnastic apparatus is provided; for a few, special developing apparatus is needed; all are sufficiently under control and observation. No serious accident has occurred since the opening of the gymnasium in 1859.

The direction is in the hands of Prof. Edward Hitchcock, M.D.; the duties of his position include teaching gymnastics, physiology, and elocution as connected with bodily movement; he is expected to be acquainted with the health of each student, and is required to furnish the excuse whenever sickness compels a student's absence. The department is dignified by marks given for attendance and deportment, and its head is a member of the faculty.

The object aimed at has always been the promotion of health and power of work, rather than the development of muscle or the performance of feats of agility and strength. As evidence of the success which is attained, Prof. Hitchcock states that sickness among the classes diminishes regularly from the Freshman year up, being in the Senior year little more than half as great as in the Freshman.

A system of this sort reaches a large number of young men who much need it. Few are so judicious and persevering as to lay down a plan of gymnastics and adhere to it. Much of the apparatus in ordinary gymnasiums is unsuited for the beginner: its effect is to exhaust and rack his frame and discourage his efforts. The most complete outfit of apparatus, and the best instruction, will not insure the attendance of the very class of men who need the training most. Nothing is, on the whole, so well suited to the average needs as a class-system resembling that of Amherst.

At Harvard there is everything except compulsory class-work. Careful measurements of the person enable the superintendent, Prof. Sargent, to assign special exercise to each young man, defined in kind and amount, for the purpose of strengthening his weak points. His apparatus and methods have been adopted by a considerable number of colleges, including some for women.

A new feature of Dr. Sargent's work consists in the taking of photographs of gymnasts—front, back, and side views of the naked body—at the beginning of their course, to be compared with those hereafter to be taken on leaving college. It has been the writer's privilege to look through this list, now numbering several hundred; and it must be confessed that the most striking thing about them is the rarity of a handsome body, or of even a well balanced and proportioned body. Deflection of the spine is quite common. Athletic oarsmen show, by the twist in their upper backs, which side of the boat they are accustomed to row in; but most of the curvatures do not seem to arise from excess of muscle. A prevalent droop of the right shoulder is noticeable. It is thought that this may originate in carrying weights in the right hand, and perhaps in the musket drill of the public schools.

Another noticeable thing is the frequency of the hollow back,—a peculiarity of form which may be hereditary, but may originate in weakness of the supporting muscles of the spine. Weakness of the upper part of the trunk, allowing the shoulders to fall forward and the neck to stoop, may be the first step, and the saddle-back may be merely the compensatory curve.

Smallness of the chest, and consequent want of lung-power, constitute a failing that it is really of great importance to correct. There can be no safe athletic training for men whose chests are small: they run the risk of inflicting permanent injury upon heart or lungs by the efforts made with their large muscles. It is well known that a small chest predisposes to consumption. Our boys need not be athletes,—it would be better for the mass not to attempt to compete in that line,—but they ought to have better breathing-capacity than they now have. *Play* is better than any gymnastics, from a certain point of view; but a full allowance of the prevalent sports does not give to boys a good chest-development.

The late Archibald Maclaren, describing the English school-boy as coming under his observation, said, "I find that almost every youth at the time of passing from the schools to the University has, as it were, a considerable amount of attainable power and material capacity undeveloped; his body, or rather a portion of it, is in arrears in this respect, and as arrears, and as a recoverable debt, the youth may fairly view it." During the youth's first term of two months, with properly-administered exercise, the chest will expand, in all ordinary circumstances, two inches, and in peculiar circumstances he has known the increase to reach double that amount.

Most sports develop the *legs* satisfactorily,—walking, running, leaping, foot-ball, cricket, fencing, tennis, racket, fives,—but some of these give in addition an excess of work to the right arm. The result of sports without gymnastic training is a frequency of pigeon-breast, hollow breast, drooping shoulders, and stooping. There is also an occasional excessive upward growth without corresponding expansion of the chest. These are the results of abundant play in English schools for the better classes, where play is a regular part of the day's business; they represent the best that a boy's play can do for his development.

Military drill was brought into favor by the war; its supposed end is to furnish large numbers of men ready trained to service in case of emergency. Some military men find that the real result is a conceit of knowledge and indisposition to enter the militia. Its best side is the moral side: it raises self-respect, and promotes obedience by showing the practical need of it. It is, further, as good exercise as many games are, and shares with sport the element of interest and pleasure. Schools known to the writer have given two hours out of the weekly programme to drill, and have found that the week's work as a total was not lessened. The objections are that the exercise is taken in a cramped position, every movement being executed to pattern, and that the number of movements is extremely few, so that the exercise is not at all a typical one for developing the body; also, that the musket is too heavy, and that it is carried chiefly in the right hand for convenience. The so-called setting-up drill consists of light gymnastics, the object of which is to give the soldier a good position at the outset: this is not and cannot be properly carried out in schools unless time is taken,—and the time is already taken for musket drill.

The amount of time taken by Maclaren to correct the shape and growth of boys in his own school was one hour of gymnastics weekly,—this, in addition to abundant *play.*

The children in the turner classes practise an hour twice a week, somewhat after the general plan of Amherst, aiming to give the girls more of grace (*e.g.*, by a variety of dancing movements) and the boys more of muscularity. Their work is well worth inspecting.

The British soldier, on entering the army, is put through daily gymnastics from one to one and a half hours daily for three or six months. It is unnecessary to describe the practice in other armies.

In our public schools the friends of reform should not be satisfied with less than half an hour twice a week, under trained teachers. An hour twice a week might afterwards be thought desirable,—the method to be that of light gymnastics, to some extent imitating that of Amherst. The teachers may be specialists at a moderate salary; or the work may be done by such of the regular teachers as have special gifts for it, as is the case in Germany. As regards the amount of work to be done, or the teaching force, the city of Frankfort-on-the-Main is a good illustration: there are twelve thousand children in the public schools of that place, and the number of hours given to gymnastics is equivalent to the constant services of seventeen and a half teachers. The city has special halls with apparatus, of the length of from twenty to twenty-five metres, nine or ten metres wide, and from five to five and six-tenths metres high.

THE ROUTINE OF A SCHOOL-DAY.—It will not be without profit to study the arrangement of time and occupation made for cases where a young person's whole time is under control. In making the plan it is necessary to have a clear idea of the amount of work that is desirable. Time must be assigned for play, and for gymnastic lessons: either or both may be made compulsory (as foot-ball is in some of the great English schools).

The following is an outline of a day's work in one of the best American boarding-schools for preparation for college:

SUMMER.	WINTER.	
6.30	7	Rise.
7	7.30	Breakfast.
7.45	8.15	Prayers.
8	8.30	Study and recitations four and three-fourths or four and one-half hours.
12.45	1	Intermission.
1	1.15	Dinner.
2	. . .	Study-session in summer one-half hour (none in winter), then play-time.
2 30	2.15	Play-time, summer three and one-half, winter two and three-fourths hours.
. . .	5	Quiet in session, study optional.
6	6	Supper, one-half hour.
6.30	6.30	Intermission.
6.45	6.45	Prayers.
7	7	Study session in school-room until bedtime.
8.30	8.30	Youngest boys (twelve and thirteen) go to bed.
9.15	9.15	Middle boys (fourteen and fifteen) go to bed.
10	10	Older boys go to bed.

The amount of sleep allowed for is from eight and a half to ten and a half hours, according to age and season. The boys all have the gift of sleep, and use all the time allowed. Study and recitations for the youngest boys, six and a half or six and three-fourths hours; for the oldest, about eight hours. Play, two and three-fourths or three and one-half hours, according to season, in a solid lump, besides some intermissions too short for serious play. There is a twenty-acre lot to play in; in winter they use the gymnasium at their option, under control of a tutor. Detention for punishment is assigned to the afternoon play-hour: most boys average one hour a week at most; mischievous boys suffer longer detention, but in no case to their physical harm. Sweetmeats are not expected to be sent from home; if discovered, they are confiscated, or are served at the boys' table so that many share.

Compare with this the routine of a large boarding-school for girls, of very good standing, in the same part of the country:

6.30	Rise.
7.10	Breakfast.
8	Recitation, forty-five minutes.
8.45	Prayers.
9	Recitations and study, four hours.
1	Dinner.
2.15	Walk, in which all join.
3	Study and recitation, two and one-fourth hours.
5.15	Recreation,—free time.
6	Supper, followed by recreation.
7	Prayers.
7.15	Study, one and three-fourths hours.
9	Bedtime.
9.30	Lights out.

Here are nine hours assigned for sleep, and eight and three-fourths hours for study; but three-fourths of an hour is taken out every day for gymnastic exercise in classes. All take a walk of three-fourths of an hour. There is apparently a considerable amount of time left free. The contrast between girls and boys is seen in the compulsion exercised in regard to all exercise, which is doubtless necessary. The required hours of work are likely to be too long for some girls; and if music and letter-writing and literary societies and prayer-meetings are added, girls are likely to be burdened. Visits to the pupils' homes in term-time are properly forbidden.

Detention is an effective means of punishment when not carried too far; but when a boy's Saturday forenoon, or even his whole day, is spent in silent confinement for a series of small faults, the effect is bad, morally and physically. An occasional good whipping is far better.

The chief objection to corporal punishment is perhaps its effect on teachers. Without exactly making them cruel, it presents a temptation to hasty and often excessive action, afterwards regretted. Girls should not be punished in that way; boys seldom, and with conscientious reflection, without anger.

The city of Cleveland requires that every case of corporal punishment shall be reported to the superintendent of schools, in blanks containing the following headings: date; offence; general character; home influences; means employed for reform; whether parents were previously notified of misconduct, and what answer was given; whether ever previously referred to the principal of the school or the superintendent, and how often; result of punishment.[1] This represents fully the present tendency to caution.

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[1] Report of Schools, 1875–76.

II. EXTERNAL HYGIENE.

SITE OF THE SCHOOL-HOUSE.

One of the first points to consider in selecting a site is dryness of the soil and air. If possible, we should choose a spot considerably elevated above ponds or wet ground, on soil which dries quickly after rain, and where evening mists do not hang. In damp, low-lying spots consumption is apt to prevail; a tendency to rheumatism, catarrh, and neuralgia is common; diphtheria often prefers low, damp regions, and so does intermittent fever.

A house may possess a damp atmosphere amid wholesome surroundings, if the cellar is damp. Even high land is often full of water. In the country it is usually possible to choose a dry spot, but in cities many otherwise desirable lots contain underground springs, or watercourses which have been dammed by the filling in of new streets, and it may be necessary to drain or water-proof the cellar. A wet cellar is never necessary or allowable.

Next to the general healthfulness of a spot we may consider the question of light, one of the most puzzling and unsatisfactory of all the problems of school-science in large cities. Wherever we build, we must expect that others will by and by occupy the adjoining land with houses of unknown height. There ought to be an open space on all sides large enough to secure a good light in any such contingency. How difficult it is to accomplish this in cities may be seen in New York. In 1880 the *Sanitary Engineer* gave prizes for designs for school-houses to be placed on lots one hundred feet square (that being a usual size) not situated on street-corners. In making the award, the committee came to the inevitable conclusion that these conditions made it "impossible to secure sufficient light without either overcrowding of class-rooms, or an unsatisfactory arrangement of corridors, stairs, etc." Not want of foresight, but the cost of land, prevents proper lighting in cases like that of New York; but there are many small yet growing cities which, with culpable want of foresight, are building school-houses within six feet of boundary-lines.

Noise and other nuisances ought to be avoided. It is possible to foresee the growth of traffic on certain lines, and avoid them: by doing this we are the more likely to retain comparatively good light. In some places the law properly forbids the maintenance of liquor-shops near schools. The neighborhood of police-stations, with the occasional view of noisy prisoners haled before justice, is most objectionable. Engine-houses are also bad neighbors. In short, all sources of noise or excitement and the neighborhood of crowds are to be avoided.

The purchase of land enough to give a space sixty feet wide all around the building should be a minimum requisition.

PROTECTION AGAINST FIRE.

Fires in school-houses can generally be traced to the heating-apparatus. Nothing special need be said regarding the precautions needed in the case of common stoves; but, as open fireplaces are now placed in some schools, it may be well to call attention to a possible danger from the conduction of heat through the hearth and back to wood-work in contact with them. A double back of masonry, with air-space, is required,—the hearth supported by a brick arch. Some builders carelessly run beams into the brick-work of chimneys.

A tin ventilator running in a floor to a chimney has been known to cause a fire: a quantity of light material was put into a grate in the story below, and the flame and hot air, reaching back from the chimney into the tin flue, heated it so that the planks caught fire.

Special care ought to be taken to protect wood-work against the heat of hot-water or steam pipes. Hot-water pipes may in some cases reach a temperature much above 212° F. A properly-arranged boiler for steam-heating, it is said, will not give superheated steam, and the heaters will not scorch wood-work; but with coil boilers, if not kept properly supplied with water, superheating might occur. There is much evidence to show that fires do take place just in the neighborhood of pipes; and they should be protected most scrupulously by the steam-fitter. Pipes may be suspended by iron straps in a groove lined with bright tin, allowing a space of one inch between pipes and tin.

It has been proposed[1] to provide large valves, easily opened, at the roof, so as to draw out great quantities of air or smoke at the beginning, thus relieving the occupants of the danger and alarm which smoke occasions: also to provide extra flues in walls, communicating with the floor-spaces or wainscot-spaces where smoke is generated, to carry smoke off.

Floors should be brought up to brick walls, so as to cut off all upward currents behind the furring. A method of doing this has been patented.

Stairs should be as strong and substantial as possible, and practically fireproof. "They should be the last to burn or fall in the structure. In ordinary construction they are among the first places to catch fire and the most efficient means of rapidly conveying it from floor to floor. Stairs can be built at a reasonable outlay that are convenient, easy of access, well lighted, and absolutely fireproof." (W. R. Briggs.) Iron frames enclosed in a separate brick tower at each end of the building are the fullest realization of this idea. There ought to be two, on opposite sides, so that one would be left free in any case.

Five or six feet is a proper width for the stairs in large buildings; the hall into which they empty should be considerably wider, and the front door-way from six to ten feet, with doors set to open outward. School-room doors should open outward. A panic *may* arise at any place, with or without good cause. Spiral stairs or wedge-shaped stairs are to be forbidden. A landing half-way up, with some spare room, is desirable. There must be plenty of light, and the *impression* of security must be imparted.

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[1] "Lomb Prize Essays," published by the American Public Health Association, 1886.

The balustrade is not altogether the best arrangement; a well, in fact, is not needed for reasons of construction, but is useful for discipline, enabling the scholars to be seen easily. The loss of fifteen children's lives by the breaking of the balusters during a panic, while the crowd was descending a stairway three feet wide, in the Notre Dame Academy of New York, is an instance in point. There were nine hundred children in that building. It is said that they had been carefully instructed in the "fire-drill;" but the fire-drill does not meet the case when the stairway is insufficient. With good arrangements, eight hundred children ought to be got out of a school-house in a minute and a quarter; they ought, for practice, to have to do it without notice as often as once a month. It is certain that there are a good many fire-traps just as bad as the Notre Dame: the writer has seen much worse.

VENTILATION AND HEATING.

This subject is admittedly of the first importance. The school is the place for work, and bad air at once impairs the working-power. More than this, the effect of bad air is to deteriorate the whole constitution: there is little exaggeration in the statement that all diseases are either caused or are made more severe by bad air. Several notorious "school-diseases" are rather closely connected with this cause,—dyspepsia, headache, nervous debility, anæmia, scrofula, consumption, various affections of the eyes,—the special discussion of which is found elsewhere.

The impurities of air may be divided into three classes: 1, dust, smoke, stenches, gases from heaters, and other defilements which are independent of the presence of scholars, and should be entirely got rid of; 2, carbonic acid from the lungs; and, 3, organic matter exhaled from the lungs and skin. The last two are unavoidable, and must be allowed for in ventilating.

Carbonic acid gas, in the quantity found in ordinary badly-ventilated rooms, is not probably of itself a serious source of injury. Men who go incautiously to the bottom of wells or vats sometimes become unconscious, and perish unless rescued, owing to the presence of nearly pure carbonic acid; but in rooms the amount present seldom exceeds five or six parts in one thousand, which quantity cannot be very actively injurious except in so far as it slightly lessens the proportion of oxygen. The lethargy of a close lecture-room seems to resemble the stupor of asphyxia, but in reality it is genuine sleep, caused by heat, bodily fatigue, an easy seat, a monotonous voice, weariness from continued passive listening,—all greatly aggravated by the bad air, no doubt. But carbonic acid by itself does not produce the violent symptoms of poisoning which are familiar from the description of the "Black Hole." Expired air freed of carbonic acid does produce such symptoms. The eminently noxious agent, then, appears to be, not carbonic acid, but the animal exhalations which accompany it in the breath.

The process of analyzing air for the organic impurities is difficult and uncertain. It is therefore necessary to depend upon the comparatively easy and certain test for carbonic acid, which corresponds quite nearly in proportion with the organic impurities and is safely taken as their index.

The atmosphere, when pure, contains normally about four parts of carbonic acid in ten thousand. Recent analyses seem to indicate that three and a half parts is nearer the truth; but it varies somewhat, being larger in cities. Assuming four as the rule, the question arises, "Supposing the air of a room to be constantly polluted by breathing, at what stage shall we say that it becomes unsuitable for further use?" The question is answered variously. Pettenkofer proposed seven as a standard of maximum amount of carbonic acid; Degen, six and six-tenths; while Parke, who may be regarded as the best authority in our language, sets it at six. That is, the permissible added impurities correspond to the addition of three, two and six-tenths, or two parts of carbonic acid in ten thousand. Parke bases his standard upon the personal experience that air at six seems pure, so that a person coming from the outer air perceives no trace of odor, or difference between the outer air and the room in point of freshness, while if the carbonic acid exceeds six the air usually begins to be perceptibly impure. When it reaches nine or ten the air is what is called close and fusty; above this it becomes disagreeable. After a person has been a few minutes in a room the odor becomes imperceptible, and he no longer can judge "by the nose."

It is abundantly proved that in our climate, and for large bodies of persons, ordinary (so-called "natural") means of ventilation by windows, fireplaces, and holes in walls are entirely inadequate, and must give place to the systematic use of flues of sizes suited to the supply required.

The allowance of fresh air per head is based on the datum just given for *permissible degree of added* impurity. It is, unfortunately, the case that the impurity cannot be got rid of by itself; it mixes so rapidly with the air that it seems best, on the whole, to consider that the mixture takes place at once, and that our only remedy is to *dilute* the air by letting out some that is foul and letting in some that is pure. We have, therefore, the problem in this form: How much fresh air is needed to dilute one person's exhalations to a given point?

Adult men exhale about six-tenths of a cubic foot of carbonic acid per hour while at rest (Pettenkofer, Parke). The equation becomes, then, 2 : 10,000 : : 0.6 : 3000, showing that one man's exhalation, diluted with three thousand cubic feet of air, impregnates it in the proportion of two parts in ten thousand. The hourly supply required is three thousand cubic feet per man. The calculations of Roth and Lex give three thousand five hundred.

This statement applies to rooms constantly occupied, and to adults. When the room is to be occupied but three or four hours at a time, and is thoroughly aired in the interval, the amount may be reduced to two thousand five hundred cubic feet per hour (Billings), or about forty per minute. Assuming that children require nearly as much as adults, the *standard of thirty cubic feet per head and minute* seems a fair one,—as reported by the special committee on plans for public schools, given in the *Sanitary Engineer* for March 1, 1880, and repeated in the report of a commission on the public schools of the District of Columbia.[1] This is the *minimum requisition.*

It may be questioned whether children require the full supply of air assigned to adults. They are smaller; the work of transformation of material, though active, is in some degree proportioned to their consumption of food, which equals that of adults at fourteen, but is much below it at six and eight; they are very active, yet doubtless do not perform as much absolute muscular work as adults. Figures quoted in the Lomb Prize Essays (page 73) show that children under ten expire about one-half as much carbonic acid as adults.[2]

The views of De Chaumont, as given in a report made to the International Congress of Education held at Brussels in 1880, are presented by Billings.[3] Assuming that adult men exhale two hundred and sixty-six cubic centimetres of carbonic acid per hour at rest for every kilogramme of their weight, he makes what seems sufficient allowance for increase due to movement, speaking, etc., and assumes three hundred and forty-six cubic centimetres per kilogramme for children. Taking Quetelet's tables of weights at different ages, he finds that the supply of fresh air required in order to keep up the standard of purity (six in ten thousand) would be—

AGES.	CUBIC FEET PER HOUR.	AGES.	CUBIC FEET PER HOUR.	AGES.	CUBIC FEET PER HOUR.
4	910	10	1575	16	3220
6	1085	12	1875	Adults	4130
8	1350	14	2450		

To this Billings remarks, "The question of the amount of carbonic acid exhaled has little or nothing to do with the matter, except in so far as it is an index of the amount of organic matter given off; and it is probable that the difference between the amount of organic matter excreted by a child of five and one of fifteen is by no means so great as would be indicated by the carbonic-acid tests. I should allow in a school-room or hospital very nearly the same amount of air-supply per head for children of all ages over five years. . . . The standard which I would fix should be . . . for each person in a class-room not less than thirty cubic feet of fresh air per minute."

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[1] Mis. Doc. No. 35, House of Rep., 47th Congress, 1st Session.

[2] Adult man, .6 of a cubic foot (16.8 to 22.6 litres); girl of seven or eight, .29 of a cubic foot; girl of eight or nine, .34; child of ten, .28; boy of twelve or thirteen, .37; young woman of seventeen, .37; boy of fifteen, .5.

[3] Op. cit., p. 162.

It is a fact that children do not stand bad air as well as grown people. Not only do they require large supplies to correspond with a rapid rate of growth, but they are more liable to lose health from any given cause; they are more delicate,—sooner depressed, and quicker to recover.

"I think it best," said Simon, "that children and adults should be deemed to require equal allowances of air and ventilation."

CUBIC SPACE REQUIRED PER HEAD.—A common size for school-rooms, where liberal ideas prevail, is about twenty-eight by thirty-two and thirteen and a half feet high, giving twelve thousand and ninety-six cubic feet. Beyond these limits the size becomes inconvenient for the purpose of instruction. Let us assume a class numbering forty; the cubic space per head is three hundred feet, a liberal allowance. But forty persons require forty times thirty—*i.e.*, twelve hundred—cubic feet of fresh air per minute, and seventy-two thousand per hour: so that in the room supposed the air must be wholly changed six times in an hour.

Take the minimum requisition for New York city schools, seventy cubic feet of space per head; if the scholars are to receive the same allowance of fresh air, the room must be wholly re-charged with air twenty-five times an hour! It is absurd to suppose such a case as possible: the children would live in a whirlwind. It is a good deal to speak of changing six times an hour; yet this can be done without risking a draught, whereas a more rapid rate is considered likely to give annoyance.

These calculations give the reason for requiring liberal space, as usually stated. There are other reasons, however. A large room has more windows, and in other ways favors "natural" ventilation. The airings at recess give a larger store of fresh air to draw from. There is a tendency for the state of the air to grow steadily worse from the beginning to the end of a session: this progression is far more rapid in crowded schools. This is obvious enough, and is common matter of experience. Liberal space is very desirable, even if there is no thorough system of ventilation.

On the other hand, it seems a pity to spoil a good cause by excessive claims; as when one thousand cubic feet of space per head is named as desirable, in several places in the second report of the Ontario Provincial Board of Health.

The following list of requirements is reduced to feet from Billings:[1]

Belgium, law	157
Educational League, Belgium, proposes	336
Holland, average	129
Haarlem, eighty-nine schools	158

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[1] Op. cit., p. 163.

English Board schools, about	125–150
Bavaria, law (at eight years)	136
" " (at twelve years)	196
Dresden	107
Frankfort, Medical Society recommends	297–322
Basle, law	213–237
Sweden, primary	187–264
" higher	269–350
New York City (for various ages)	70–100

METHODS OF VENTILATION.—The heated flue is at present the arrangement applicable to the greatest number of cases of school-house ventilation. Its size must be determined in accordance with the number of pupils in the room or house. In most cases one general discharge-flue is sufficient. It should be located centrally, so as to lose no heat to the outer air. It consists of a straight brick shaft, rising from the cellar to the roof, and capped above the roof to protect it from downward gusts of wind. To increase the current, the air is warmed by having the smoke-flue of the heater pass up through the middle. The velocity of the ascending air depends on many circumstances: if the weather outside is cold, if the chimney is high, if the heat is concentrated, the current is swifter than under the opposite conditions. A simple formula for the *theoretical* velocity is the following, from Billings:

$$v = 8\sqrt{\frac{(t - t') \times h}{491}},$$

In which $v =$ velocity,
$t =$ temperature in the chimney,
$t' =$ temperature of the outer air,
$h =$ height of the chimney.

If the temperature out of doors is 40°, that of the chimney 100°, and the chimney is 50 feet high, the result is

$$v = 8\sqrt{\frac{60 \times 50}{491}} = 8\sqrt{6.11} = 20.$$

This velocity of twenty feet per second is *greatly diminished* by friction and other circumstances. The velocity desired is five feet,[1] which will discharge five cubic feet of air per second for every square foot of the cross-section of the chimney. This, be it remembered, is only an average performance, corresponding to a difference of 60° between the chimney and out-doors.

If a pupil requires one-half of a cubic foot per second, every square foot in the section of the chimney corresponds to ten pupils. A school of four hundred pupils requires a shaft eight by ten feet, inside measurement, not allowing for the smoke-flue and for space taken up by entering pipes and guards.

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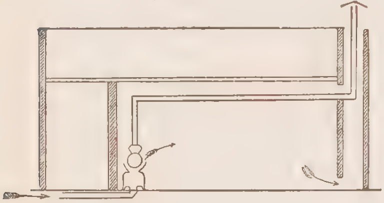
[1] "For ordinary and the most favorable circumstances the actual velocity in the flue is best if it be established at about five feet per second."—PROF. W. P. TROWBRIDGE, in *Sanitary Engineer*.

Schemes of Ventilation by Flues.—To pass the smoke-pipe up the ventilator-flue is an obvious thought. The draught, without some such arrangement, is seldom as strong as we require. One of the early applications of the principle is shown in Fig. 12, taken from Guillaume (1874), where it is called "Mott's system,"—apparently a combination of the ventilating-stove and the chimney-ventilator. This represents a plan which can be put in use in almost any country school,—the only difficulty being the inadequate size of the flue. For example, in a one-story building the draught will be feeble, owing to the shortness of the chimney; twenty-five pupils would require under those circumstances a flue two by two and a half feet in section. Such a chimney would need an efficient guard against rain. The outlet should be narrowed, to increase the velocity at that part. The danger would be that wind would occasionally (or regularly) blow down one side of the flue while the hot air rose on the other. This danger would become a certainty if the flue were made two by a partition.

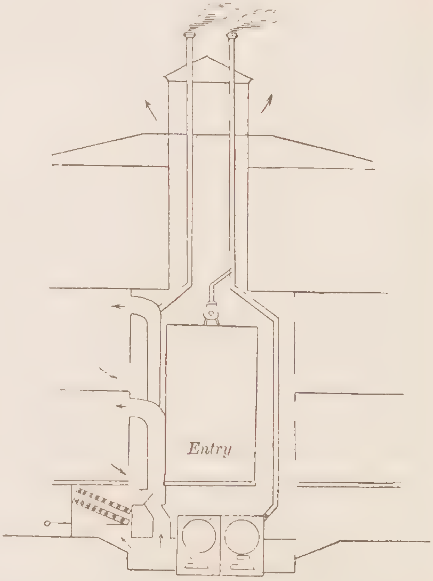
Fig. 12.



Sheet iron or wrought iron rusts out rather soon; for the pipe in the chimney cast iron is best.

Branch flues of moderate size leading from the rooms will have a slower current, hence must be larger in proportion; a class of fifty might require two horizontal ducts, each two feet square,—a requirement which would prove awkward to the architect. In view of the great size and of the expensive complexity of these arrangements, it is best, when possible, to have no flues or ducts leading to the main flue, but to place each room in contact with the latter, so as to deliver its foul air directly into the shaft.

Fig. 13.



In illustration of this method, Figs. 13–15 are given, representing the system as applied to an old-fashioned square building with four nearly square rooms on each floor and a wide entry running through the middle.

The plan, as explained by the engineer, is a copy, with certain modifications, of the plan adopted by Mr. W. R. Briggs for the Bridgeport High School. Fig. 13 shows a section of the house as seen by a person looking through the entry: rooms on the right and left; the third story occupied by a hall; a flue on each side of the entry, uniting at the third story, and warmed by the smoke-flues of the two boilers. The flues are of wood, lined with tin. Where the two unite, a brick floor is laid on an arch, to support an iron stove to be used when the boilers are not heated. This part of the shaft is entered by a door from the hall.

Fig. 14.

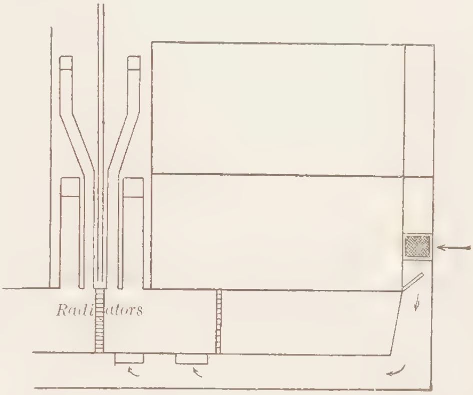
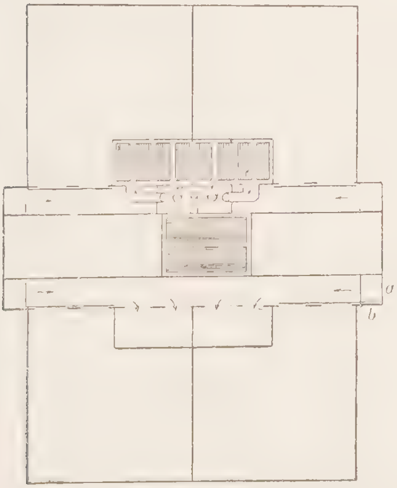


Fig. 14 is a partial section in the direction from front to rear, showing the intake of air by a screen window, and the way the air is carried up from the heaters to the tops of the rooms that are thus supplied.

The intake stands three or four feet above the ground, as is seen, at the corner of a projection in the porch. A flap-valve is fastened with hinges like a common door, and swings back and forth, covering windows *a* and *b* alternately according to the direction of the wind; this prevents the air from blowing through both windows at once and deranging the supply.

Fig. 15 gives the cellar-plan. The fresh air in entering passes through two long channels to the heated chambers, of which there are four, each supplying the two rooms directly over it. There are four coils used for each room, making thirty-two in all; they are disposed in two layers (Fig. 13); each can be shut off separately, giving an economical control of the heat sent to each room. The hall in the third story is heated by upright coils at the sides of the hall, with air-supply taken through the side of the building. One of the shafts is shown in Fig. 14, with its four air-tubes and one smoke-flue.

Fig. 15.



In introducing the system it was found desirable to employ steam heat; an excavation had to be made to receive the boilers. At the right and left of the boilers are seen the air-supply ducts in section, communicating outwardly with the brick coil-chambers, and upward with the small chamber where cold and hot air are mixed before entering the rooms. A valve is placed so that the cold or the hot air may be entirely shut off, or different proportions may be used; the valves are controlled from the school-rooms to which they pertain. A slider of sheet-iron regulates the admission of air to the heaters according to the weather.

The preceding is an instance of thorough work. The next best thing, for those of limited means, is to avoid blunders and waste. The following points are offered with this purpose.

Fireplaces are partial remedies for bad ventilation. They are now occasionally placed in new school-houses, both for this reason, and also in the expectation that they will be used for fires in mild weather; but the trouble of attending to such fires is too great for an average janitor. Their ventilating power is small compared with the demand, and may be roughly stated as sufficient for ten persons while the fire is going. As a heater the fireplace wastes from seventy-five to ninety per cent. of the heat.

Stoves arranged like the "portable furnaces" that are set up in cellars are useful. The plan is to place a screen of zinc or galvanized iron around the stove, leaving an interval of a few inches for an air-space; a hole in the floor, communicating with a pipe led out of doors, supplies a current of fresh air underneath the stove, which becomes warmed and rises into the room. The screen comes down to the floor; a valve regulates the supply of cold air.

Such a stove is shown in Fig. 12, an old design applicable to the case of a country school-house. The stove is set at a distance from the chimney, to get more heat from the pipe; the latter ought to have a strip of bright tin hung below it, to shield the heads of the scholars. The upper ventilator cools the room.

Ventilating-stoves of various patterns are sold, based on the above principle. They cannot supply a quantity of air commensurate with the wants of a school, but they are good as far as they go, and doubtless economize fuel.

Fig. 16.

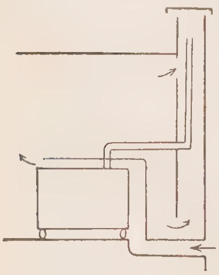
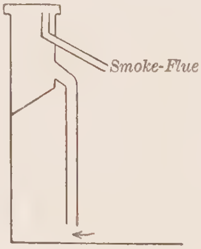


Fig. 17.



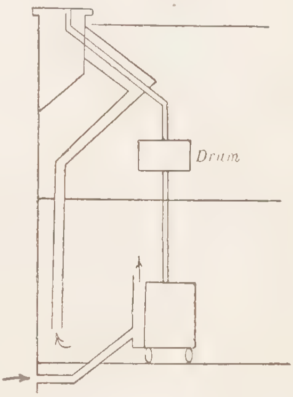
Figs. 16–18 show how the principle of ventilating rooms by heated flues may be carried out. They are adapted from Mr. Jacokes's illustrations in the Seventh Report, Michigan Board of Health.

Fig. 16 shows air entering by a pipe beneath the floor to the casing around the stove, which is four inches and six inches wide all around. Foul air escapes by the heated chimney. The upper outlet requires a valve.

Fig. 17 shows the suction of the chimney applied at the level of the floor by carrying a pipe down.

Fig. 18 combines two stories. An opening in the enlarged part of the ventilator in the upper story may be utilized for that room.

FIG. 18.



AIR-SUPPLY.—The advice has been given to make the ventilation of a house depend on a single shaft. By doing so we avoid the possibility of having two shafts "drawing against each other." Two or more shafts, however, of equal height and equally heated, need not interfere with each other, provided each has a full supply of air. This point may be overlooked, with bad results, as it often is in the chimneys of dwelling-houses.

In the illustrations (Figs. 14, 15) a full supply of air is indicated as entering by two large conduits, lined with masonry, running across the floor of the cellar and opening at each end at a point four feet above the ground. The conduits are large enough for a man to walk through them with considerable stooping. The inlets are so arranged with a flap-valve as to catch the wind; there are also regulator-valves at a point below the inlets and again at the entrances to the radiator chambers. The heat of the air entering school-rooms varied from 80° to 105° F.; on leaving the rooms it is below 70° F.: the outlets must, therefore, be larger than the inlets,—a good deal larger, practically, in order to favor escape at a low velocity.

One cannot help seeing how intimately the problems of heating and of ventilation are connected. It is the part of economy to make the details correspond. Waste heat from stoves, etc., should be utilized, when possible, to heat ventilator-shafts; and the latter must be provided with a due supply of waste air. The immense quantities of air that are discharged must be replaced; and it seems evident that the replacement should be made by air that is already warmed to some extent, if not fully. There are very few buildings where this can be done at present: hence advice about opening windows is of universal application.

As school-houses now are, windows must be opened. In country districts, especially, both sashes ought to move easily up and down. In exposed places double windows are applicable; they must not be fastened, but must be freely movable. A double pair of sashes with the lower outer one raised and the upper inner one lowered gives a tolerably safe arrangement. Window-boards are often placed under the lower sash, filling the space entirely, the air in this case entering by the crack between the two sashes. Or the board may be set at the distance of an inch from the lower sash, in such a way that when the sash is raised two inches the air is deflected upward. All such contrivances need to be watched closely, or they will occasionally give rise to dangerous draughts.

It is a good plan to make the curtains roll at the bottom, so that the tops of windows can be opened freely in summer without injuring the curtains.

Pierced window-panes, gauze shields to be placed before cracks in windows, Eureka ventilators (little slanting apertures in outer walls), and a variety of other contrivances, have more or less value, but do not meet the wants of school-rooms: if they let in a good supply it causes an unendurable draught. No window-supply can be sufficient in cold weather: a crack opened in every window and well watched is a proper measure; as a supplementary arrangement it should be an inflexible rule to devote five minutes at the close of each hour to some drill like light gymnastics, the windows being opened instantly at the beginning of the exercise and closed after it. At recesses the same should be done, and scholars should be made to leave the room.

The writer has seen a great many school-houses in which tin ventilating-tubes have been placed long after their erection. The result may be stated as follows. There is generally a distinct draught in the pipes, and the air in the rooms invariably remains poor. It is said that there is "marked improvement," and this is probably true. The ventilation secured by these pipes may amount to one-fifth of what is needed, more or less. They are not specially heated, and their dimensions are inadequate. Such a system may work admirably when applied to a series of rooms containing one or two persons (rooms for practising music, private study-rooms). The usual plan, in the absence of an adequate shaft, is to build a large wooden box in the attic under the eaves and place in it, around the sides, a series of steam-coils. The box opens upward by louvers, and receives at the sides and bottom the tin tubes leading from the rooms. A draught is certainly obtained in this way, but it is an expensive way. The power of a chimney depends on its length,—*i.e.*, on the length of its warmed part; and in this case we have the equivalent of a chimney only about twelve feet high, placed just below the roof. An instance may be given. The S—— school, a large, spacious building with sixteen rooms and probably eight hundred pupils, was the subject of serious complaints on account of bad ventilation. A system of large tin flues, opening in the attic to a number of such boxes as are described, was introduced about five years ago. The principal of the school considers that a very beneficial change has occurred, a result partly due to the improvement made in the sewerage-arrangements. The consumption of coal previous to the change was one hundred and fifty tons per year; the average of three years since the change is one hundred and seventy. It is probable that the added consumption is chiefly due to the radiators in the attic, which roughly correspond in surface with the increase.

The size of these flues is eighteen inches for school-rooms (two to each room). This is evidently not a sufficient ventilation.

Ventilation by flues must be planned to give spacious passages, with air at a low velocity. Horizontal flues, sharp angles, roughness of the inner surface, retard the current; so do narrow flues: the smallest diameter should not be less than five inches. Round tin pipes are unobjectionable, but must be boxed in where they are exposed to cool currents. The discharge should not be broken at top without good reason. A patent cap may be useful to prevent down-draughts, but cannot be relied on as a source of draught. A shaft may open into a louvered cupola with exposure to all sides, in which case the shelters should be movable and be kept closed on the windward side. The top of the shaft should be closed when school is not in session, to prevent cold air from descending.

COST OF VENTILATION.—In the first place, the motive power need not cost anything: the heated shaft, if properly built, requires nothing but the heat of the smoke-flue. A ton of coal may be used in spring and fall for the stove in the shaft. The box in the attic costs in proportion to the length of pipe placed in it.

In the second place, an immense amount of heat is thrown away in the foul air which is expelled. There is no alternative: it must be wasted. But there is reason to think that less is wasted than might be feared. The waste goes on for only a quarter of the twenty-four hours, at any rate, while heating must go on all the time, holidays included. Thirty hours a week is only eighteen per cent. of the time; though, on the other hand, the temperature maintained is considerably higher than is required after sessions. The heat expended is divisible into two parts,—one used to keep up the temperature, as against the cooling effect of the outer air; the other used to warm (from 20° to 70° F., we will suppose) the air which is thrown away by the ventilators. Certain estimates by "Thermus" in the *Sanitary Engineer* assign an expenditure of eighty-six and a half pounds of coal per hour for the former object, in a building containing two hundred and fifty thousand cubic feet. For warming the supply of fresh air for four hundred pupils in such a building, at eighteen hundred cubic feet per head and hour, the expense would be seventy-four pounds of coal per hour. It is highly probable that we are obliged to expend one-third of the latter amount already, even with the poor results we have. Our people will not endure closed windows, as the Germans do; the recesses are frequent; there is a complete airing-out twice a day at least,—all of which must be considered as actual ventilation, and be subtracted from the expense of seventy-four, leaving, say, twenty-four actually spent and fifty more desired to be spent per hour. The summing up is as follows:

Daily Expenditures.

Necessary heat, 6 hours, at 86.5 pounds	519
Necessary heat, 18 hours, at 43 pounds	774
	1,293
Heated air now thrown away (by open windows, etc.), 6 hours, at 24 pounds	144
	1.437
Heated air proposed to be expended, in addition, 6 hours, at 50 pounds	300
Total per diem on school-days	1,737

One Week's Expenditures.

Necessary expenditure, 5 days, at 1437 pounds	7,185
Necessary expenditure, 2 days, at 1293 pounds	2,586
Proposed ventilation, 5 days, at 300 pounds	1,500
	11,271

Proposed ventilation costs thirteen per cent. of total estimate.

The chief reason why ventilation is not more widely introduced is the expense; and this may furnish the excuse for dwelling so long on the question of expense, and may mitigate criticism of the accuracy of figures for which the writer has had to depend on himself. The totals seem rather large. It is perfectly plain, however, that to make ventilation three times as effective as it is at present will not require the expenditure of twice the amount of coal: the estimate requires only one-seventh more. It is certain, also, that we now pay, of necessity, a considerable sum for a wretched, half-way result, with colds, catarrhs, rheumatisms, earaches, etc., thrown in. We are forced to keep windows open; the ideal system does away with that necessity. Air discharged through windows might as well be sent through shafts, as far as economy is concerned: open windows cost heat, surely.

As regards actual cost, the Bridgeport High School building requires from one hundred and fifty to one hundred and sixty tons of coal annually, of which ten should be deducted as due to the use of rooms for evening schools.

It contains fourteen class-rooms, each twenty-nine by thirty-eight feet and thirteen feet high; library, office, hall fifty by one hundred feet and twenty-two feet ceiling, water-closets, hall-ways averaging sixteen feet wide, and cloak-rooms, all ventilated. The number of pupils is about seven hundred.

The school-house represented in Figs. 13–15 has consumed an average of sixty-five tons per year for eight class-rooms, each containing about forty pupils, besides hall-ways, and a hall occupying the entire third story. There was abundant heat.

Steam-Fans for Ventilation.—The advantage of having mechanical power is that the result is independent of weather or temperature. The heated flue is recommended for buildings of moderate size, at all events. For large buildings, in cities where competent men can be had at low wages to run engines, it is probable that the expense will not prove a serious objection. Arrangements may perhaps be made by which one engineer could oversee the running of several engines. A two-horse engine furnishes power enough to ventilate a house for eight hundred children. A large fan in the attic, in a receiver to which the various flues are led, and another in the basement to ventilate the closets, would be a good arrangement. The plan is on trial in Boston, but results in figures are not given.

The heated flue will necessarily act with greatly lessened force in warm weather.

If steam could be supplied from a constant source outside a building, or if a shaft run by outside power could be used, the question of economy might be made easier.

It is necessary to supply a distinct flue for each room, allowing no opening from other rooms, or else, if a common shaft is used by several rooms, precautions must be taken to prevent foul air escaping from it into upper rooms. This is a very common fault. In the Bridgeport flue, the air from each room entering the shaft is at once thrown upward by a deflector, which serves the further purpose of turning aside currents from below. The deflector consists of a rounded piece of sheet metal, and reaches up beyond the orifice. Such deflectors are not applicable to ordinary, small, unwarmed flues.

If inclined to a conservative view, we may direct that inlets for hot air be placed near the floor-level, as has been the custom heretofore. The "Bridgeport" plan has the novelty of placing them near the ceiling. By experiments published in the *Sanitary Engineer*, it has been shown that the temperature of an unoccupied closed room under those conditions was remarkably equable, having a difference of five degrees between the floor and points five and one-half feet above it. This equability is said to be due to the way in which the air circulates. Entering quite hot at the side remote from windows, it spreads out in a layer under the ceiling and works its way to the windows, where it descends under the influence of a cool current from the surface of the glass; thence it goes back to the point of exit under the platform or thereabouts, having completed the tour of the room. Further experiments should be made to determine the circulation in other buildings, and the analysis of the air made.

The place for exits for foul air is near the ceiling for summer ventilation or in the evening when lamps or gas are burning and there is heat to be got rid of. In the winter it is at the floor-level: a higher point lets too much heat escape, and a ceiling register might discharge the entire supply of fresh warm air before it had time to mingle with the air of the room.

The Ruttan system of heating employs a heated shaft of brick, to which the foul air is brought by flues of any required length. The air leaves the rooms by perforations along the side wall near the floor, and is carried first through channels under the floor, and imparts some warmth to the latter. The writer cannot give details as to results.

The Gouge ventilator is a tube of metal, which can be inserted in old buildings, and seems to have worked well. It uses a gas-flame or a lamp to heat a small column of air, which shoots up into a larger tube and induces a movement of the whole column of air.

"Direct" steam heat does not permit of good ventilation. If a powerful exhaust-draught were to be applied to a room thus heated, the question would arise, "Where does the supply come from?" If windows are closed, the supply comes from entries, and indirectly from cellars to a large extent, which can hardly fail to be unwholesome.

Radiators set against the wall of a room, with an opening behind them leading to the open air, may be supposed to furnish some fresh warm air to the room. They will not *ventilate* it. The amount of fresh air entering by one such opening depends on the size, and on many other points, but, as a rule, they are calculated to effect very little except heating.

Much has been made of the need for moisture in the air. In our climate moisture is so seldom abundant during the school-year that a dry in-door air is not noticed as a contrast. It is probable that good ventilation, with lowering of the prevalent excessive temperature, is what we need to relieve the confined, dull, oppressed feelings that are referred to "burning of the air by furnaces." At the same time there is some ground for complaint if furnaces are heated excessively or leak gas. The gas which escapes is deleterious, for it contains not only the sulphurous acid which gives it the pungent odor, but also some carbonic oxide, which is apt to cause headache. The weight of testimony at present goes to show that the latter gas is not likely to "pass through the pores of cast-iron furnaces," either black or red-hot.

Furnaces of ample size and good make are not objectionable. They ought to be larger than can ever possibly be wanted. The chief trouble is well known: they will not send a column of air many feet horizontally, but should be placed directly under the rooms to be heated. In case of exposure to the weather, they can be set on a windward side.

Steam-heating requires the attendance of an intelligent man. In fact, the whole business of a janitor is capable of being "pushed" or neglected,—like other kinds of business. But for managing a boiler the man should have given proof of special competency.

The temperature of a school-room is commonly required to be about 68° F. in our climate; in Europe, about 60°, though there is a difference between places. Judged from the latter standard, it is curious to find a regulation in Springfield, Massachusetts, that if the temperature does not exceed 60° F. half an hour after the opening of school, the class shall be dismissed. In summer there ought to be a regulation for dismissing when the thermometer reaches 82° or 85° F., or some point indicating that study is no longer profitable.

A warmer for the feet should be placed in the entry-way. The writer has seen one large enough for twenty to stand on at once, composed of a plate of iron with steam coils underneath.

ANALYSIS OF AIR.—A convenient method of testing air for carbonic acid, simple enough to be used by persons who are not practical chemists, and yet accurate enough for practical purposes, is a great desideratum. Several such methods have been proposed:

1. Lange's method, described in Buck's "Hygiene and Public Health," vol. i. p. 624. Six bottles, of known capacity, forming a graded series of different sizes, are made perfectly dry and clean; they are then filled with the air to be tested, and closely corked. The smallest one is then charged

with a dose of fifteen cubic centimetres of lime-water, clear and fresh, is recorked, and well shaken. The presence of a certain amount of carbonic acid is requisite in order to produce "distinct turbidity" of the lime-water, by the formation of carbonate of lime. If turbidity appears in the first bottle used, we infer the presence of that required amount of carbonic acid in the quantity of air that the bottle contains; and the *proportion* is estimated by making the latter amount a denominator with the amount of carbonic acid as numerator. In short, if the reaction succeeds in the smallest bottle, it shows a high proportion of carbonic acid in the air; if it does not succeed until we have tested all the bottles and reached the largest, the proportion is small. The first objection to this method is its clumsiness, with the weight of the apparatus; another objection, and a nearly fatal one, is the uncertainty of the word "turbidity." To aid the eye, a piece of paper with a mark is gummed to one side of each bottle as a test of the turbidity.

2. The method given in the Lomb Prize Essays, 1886, p. 80, depends on the same principle. The six bottles are, however, first filled with water, and the air is introduced by suddenly emptying them. They are all then charged with lime-water colored pink by phenol-phthalein; the dose, half an ounce to each. All are shaken at once, in a frame. A given amount of carbonic acid will neutralize the given quantity of lime-water; complete neutralization is indicated by disappearance of the pink tint. The size of the bottles being known, the proportion of carbonic acid is calculated as above. The results were compared with simultaneous analyses made by the late Prof. Nichols, of the Massachusetts Institute of Technology, and an encouraging degree of accuracy was obtained. The operation, however, is described as long and fatiguing; the apparatus is bulky and heavy.

3. Lange's second method[1] requires the use of *one* bottle of moderate size (fifty cubic centimetres). The shape of the bottle ought to be stated, but is not. The charge is seven centimetres of baryta-water. The cork fits tightly, and is pierced by two glass tubes, one of which dips into the fluid. By means of a rubber tube and ball-syringe of known size (giving twenty-three cubic centimetres of air when pressed), successive doses of air are pumped in, or, rather, are sucked in, through the long glass tube. After each introduction of air, the bottle is shaken well, and the observer notices whether "turbidity" occurs. The objections to this apparatus are, the ambiguity of the term "turbidity;" the tendency of baryta-water to undergo change in contact with air, vitiating the accuracy of the experiment when made; the fouling of the tubes in shaking; the uncertainty that the operator feels how long he ought to shake the bottle; and the fact that baryta-water is a poison.

It may be well to remark here that the bottles sold by dealers in chemical glass-ware do not correspond accurately with any standard measurement, but must be tested beforehand, and the table for practical use made out by the observer for his own bottles. The rubber bulb-syringe, also, is not fixed in its capacity : a selection approaching the standard must be made by the experimenter.

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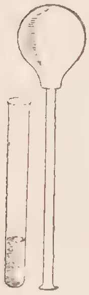
[1] Buck, loc. cit., p. 625.

4. Owen's process, given in Billings's "Ventilation and Heating," p. 24, combines Nos. 2 and 3. Air is drawn by an aspirator through a series of glass bulbs blown in a tube, charged with the pink solution of lime-water and phenol-phthalein. A loss of color is significant of saturation with carbonic acid. The writer was informed by the late Prof. Nichols that this apparatus gave results widely varying from the truth, in his own experience. The reason for this would seem to be the incompleteness of the reaction, due to the comparatively short time the aspired air was in contact with the fluid. The same incomplete reaction may doubtless be charged to Lange's second method.

5. Wolpert's method (see Fig. 19) can be recommended for further test as a practical one. As described by the author,[1] the apparatus is as follows :

"A cylindrical glass vessel, twelve centimetres long and twelve millimetres wide [shaped like a test-tube], is marked on the side with a line corresponding to the surface of its proper charge of three cubic centimetres of lime-water. On the bottom [outside] is the date 1882, black on a white ground. Clear, saturated lime-water being poured in to the level of the line, a glass tube attached to a rubber bulb-syringe is passed to the bottom of the cylinder, and air is repeatedly pumped in through the fluid until the mark 1882 can no longer be seen distinctly. The bulb, pressed at its back with the thumb, yields about twenty-eight cubic centimetres of air each time. [There is no valve ; the tube is removed after each discharge.] The apparatus is not shaken, except at the very last, when we wish to be sure of the exact degree of turbidity. If the reaction is sufficient, the mark 1882, barely visible through the lime-water, becomes invisible after a moment's shaking."

FIG. 19.



The table of values is constructed upon the basis of one discharge of the bulb, filled with air containing two hundred parts of carbonic acid in ten thousand. This air produces the requisite opacity with *one* discharge. It is inferred that if *two* discharges are required, the amount of carbonic acid present is only one-half as great, or one hundred parts in ten thousand ; or, in general terms, the number 200, divided by the number of times the bulb is emptied, gives the number of parts of carbonic acid per ten thousand.

It is not safe to speak of the exactness of this as compared with laboratory processes, but, as far as tried in practical use, it gives encouraging results. It is very simple and portable, and is easily cleaned with a little vinegar. A little more or less of lime-water makes no difference in the result, provided the dimensions of the glass are accurate. As far as can be judged, the results are the same with very rapid discharges of the bulb as with quite slow discharges.

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[1] Centralblatt für Allgemeine Gesundheitspflege, ii. 236.

The *absolute* accuracy of this empirical process may easily be questioned, but considerable *relative* accuracy may be hoped for. It is to be wished that its action may be tested by simultaneous exact analysis of air in the same room, or, still better, by the use of known mixtures of air and carbonic acid in proper receivers. A valve-arrangement might be thought desirable, in order to avoid the trouble of removing the ball and tube every time; but a valve would introduce a fresh element of uncertainty.

The author's table is given here.

Table for Wolpert's Air-Tester.

NUMBER OF FILLINGS.	PARTS OF CO_2 IN TEN THOUSAND.	NUMBER OF FILLINGS.	PARTS OF CO_2 IN TEN THOUSAND.	NUMBER OF FILLINGS.	PARTS OF CO_2 IN TEN THOUSAND.	NUMBER OF FILLINGS.	PARTS OF CO_2 IN TEN THOUSAND.
1	200	16	12.5	31	6.4	46	4.3
2	100	17	12	32	6.3	47	4.2
3	67	18	11	33	6.1	48	4.1
4	50	19	10.5	34	5.9	49	4.1
5	40	20	10	35	5.7	50	4.0
6	33	21	9.5	36	5.5	51	3.9
7	29	22	9.1	37	5.4	52	3.9
8	25	23	8.7	38	5.3	53	3.8
9	22	24	8.3	39	5.1	54	3.7
10	20	25	8.0	40	5.0	55	3.7
11	18	26	7.7	41	4.9	56	3.6
12	16	27	7.4	42	4.8	57	3.5
13	15	28	7.1	43	4.6	58	3.5
14	14	29	6.9	44	4.5	59	3.4
15	13	30	6.6	45	4.4	60	3.3

6. Lange's second method has been advantageously modified by Dr. Alfred L. Carroll as follows. Half an ounce of lime-water is placed in the test-tube, instead of baryta-water. Instead of the inexact rubber-ball aspiration, he provides a water-jar graduated at the side, with tubes arranged in the ordinary well-known way for producing aspiration of air through the test-tube. Cloudiness produced by aspiration of eight cubic inches indicates the presence of eighteen parts of carbonic acid in ten thousand; by twelve cubic inches, of twelve parts; by twenty-four cubic inches, of six parts.

The practice of testing the air of school-rooms is strongly recommended to all teachers, especially principals. In default of more exact apparatus, let a ten-ounce bottle be filled with the air of the room by a syringe or bellows; let an ounce of lime-water be added and well shaken: if there is little or no turbidity, the air is fairly good. Air previously contained in the bellows or syringe must be evacuated before using.

DRAINAGE AND SEWERAGE.

Effects of Air- and Water-Poisoning.—The drainage of a building should be strictly scrutinized when there is a suspicion that the origin of a "filth-disease" can be traced to the premises. This applies especially to outbreaks of diphtheria or other sore throat, pneumonia, dysentery or diarrhœa, typhoid fever, scarlet fever, or measles.

Foul smells may cause frequent annoyance, or even headache and sickness, for years before an outbreak of positive disease comes. The tendency of polluted air is to lower the general vitality. Air from sewers is "sewer-gas," and contains a variety of more or less injurious gaseous substances. The term "mephitic poisoning" may be used to denote their bad effects.

In an extreme degree the effluvia of drains and privies are rapidly fatal. In a school at Clapham, England, "the clearing out of a privy produced in twenty-three children violent vomiting and purging, headache and great prostration, and convulsive twitchings of the muscles. Two died in twenty-four hours."[1]

"When the air of sewers penetrates into houses, and especially into the bedrooms, it certainly causes a greatly-impaired state of health, especially in children. They lose appetite, become pale and languid, and suffer from diarrhœa; older persons suffer from headaches, malaise, and feverishness; there is often some degree of anæmia, and it is clear that the process of aëration of the blood is not perfectly carried on. In some cases I have known decided febrile attacks lasting three or four days, and attended with great headache and anorexia."

"The air of sewers passing into houses aggravates most decidedly the severity of all the exanthemata, erysipelas, hospital gangrene, and puerperal fever; and it has probably an injurious effect on all diseases." (Parkes.)

It seems probable that the origin of typhoid fever generally depends on the drinking of water contaminated by drainage; yet there is reason to believe that drain-effluvia may cause the disease. "In a case mentioned to me by a friend, an outbreak of enteric fever in a training-school was localized in certain parts of the school (whereas the drinking-water was common to all), and was traced to imperfection of traps in those parts of the house which were affected. In this case the drains led down to a large tank at some distance and at a much lower level, and the smell of the effluvia was so slight that at first it was not believed that the drains could be out of order." (Parkes.)

"A marked illustration of disease due to polluted air, when the drinking-water was pure, occurred in the school in this State, in 1864, where fifty-one out of seventy-seven young ladies in the institution were attacked with typhoid fever, of whom thirteen died; three servants also died of the fever. The vaults of the privies were shallow, filled to overflowing, and emitted a very offensive odor, which at times pervaded the whole building. The kitchen-drain discharged its contents on the surface of the ground, and a few rods from the school there was a foul barn-yard." The school referred to was the Maplewood Institute at Pittsfield. The statement is quoted from a circular of the Massachusetts State Board of Health for 1879.

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[1] Parkes's Hygiene, American edition, 1884, p. 146.

The way in which typhoid fever may be caused by polluted drinking-water is well seen in the following case, given by Dr. John L. Leconte:[1]

The water-supply of a large and prominent boarding-school for girls was obtained from a river, and stored in two cisterns under-ground. The cisterns were built of heavy wooden curbs, with a timber floor, in which it became necessary temporarily to make holes, afterwards plugged up. The cisterns had brick cemented bottoms and linings, but the plugs projected inwardly through this masonry. The plugs were removed a year later, without the knowledge of the authorities, so that the ground-water had free access to the interior. Eighteen months later, this mistake (which by itself might have caused no harm) was supplemented by placing privy vaults outside of the building, one of which was placed eight or twelve feet from the cisterns. The vaults were of the supposed "tight" kind, with nine-inch brick walls and bottom, heavily and carefully cemented, and arched over.

In three years from this, typhoid fever broke out. Under medical direction, water taken directly from the river was substituted for the cistern-water, and the last case of the disease occurred ten days after this precaution was adopted. As an evidence that the use of the cistern-water was the sole cause, it is stated that, although numerous cases occurred among the children and several among the teachers, not one occurred among the servants. The latter drank only tea and coffee, and very rarely used water, while the children (as usual) drank it freely at all times. Among seven absolute water-drinkers six were attacked by typhoid.

Dysentery and Diarrhœa.—There is sometimes difficulty in strictly separating these complaints; they have, moreover, in some degree a common origin, and are spread by the fæces of patients infecting the air. More directly to the point is the case mentioned by Clouston, where it seemed to be proved that dysentery was produced in an (insane) asylum by the exhalations from sewage which was spread over the ground (a stiff brick clay subsoil) about three hundred yards from the asylum. "The case seems a very convincing one, as the possibility of the action of other causes (impure water, bad food, etc.) was excluded." (Parkes.)

Diphtheria.—At Groveton, New Hampshire, an epidemic of diphtheria occurred in which the centre of infection was the school-house. Twenty-two cases broke out among the scholars in thirty-six hours, appearing at once in widely-separated places; one hundred and fourteen cases in all, with fourteen deaths. There were several circumstances which combined to make the school-house dangerous to health. A brook had been dammed by the boys so that in rainy weather it ran under the school-house, leaving at other times a stagnant pool. There was a boggy meadow near by, polluted by privies which had not been cleaned for two years. The refuse of a saw-mill and tannery was thrown into a mill-pond twenty rods distant; the water was drawn down to repair the dam, causing an intolerable stench; the outbreak of diphtheria followed, succeeded by typhoid fever; when the pond was kept full, the disease disappeared.[1]

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[1] Philadelphia Medical Times, May 29, 1875.

PLAN OF DRAINAGE AND SEWERAGE.—Before planning the arrangement of plumbing-fixtures in the house, we must settle two points,—the drainage and other measures necessary for keeping the cellar dry, and the place and manner of discharging the waste- and soil-water of the house.

The lot should be graded so as to carry rain-water away from the house and to some point of discharge.

As a general rule, it is well to surround the foundations with a trench extending below the foundations and filled for a foot or two with loose stone, in which a line of drain-tile is laid, which is led to a proper place for discharging the water. If necessary, drain-tile is also laid in the cellar floor; the joints are not sealed, but are wrapped with tarred paper or cotton cloth, and the trenches are then filled with sand or broken stone. The tiles discharge the cellar-water into a deep masonry trap filled with coarse sand or gravel; thence, in a city, the water will pass to a sewer, but requires a running-trap to keep sewer-air from entering the cellar; a vent is placed on the inside of the latter trap, leading above-ground.

To keep out dampness, there may be a dry area outside the cellar wall, or the wall may be double, or be provided with a damp-proof course of asphalt or slate just above the ground-line.

The cellar floor may consist of a quarter-inch layer of asphalt or concrete, with a finishing layer of the best Portland cement. Six inches of well-rammed clay (Waring) may serve to render the floor damp-proof.

The disposal of waste- and soil-water presents a serious difficulty when there is no sewer. This is not uncommon in prosperous villages which have aqueducts but are not closely enough built to make sewers seem a necessity. The danger is that the soil will then become water-soaked with the discharges from water-closets and sinks; and the emptying of the combined discharge from a large school must be regarded with suspicion. A tight vault or cesspool would soon fill: hence the practice of building vaults with loose-jointed walls, or earthen floors, expressly to let the fluids leak out. There is a common notion that such fluids are rendered harmless by the soil. It is impossible to say how far they may convey a noxious influence, or whether, "as a rule," fifty, or one hundred, or two hundred feet is a safe distance from wells. We ought to see that the vault is at least one hundred feet from the nearest building.

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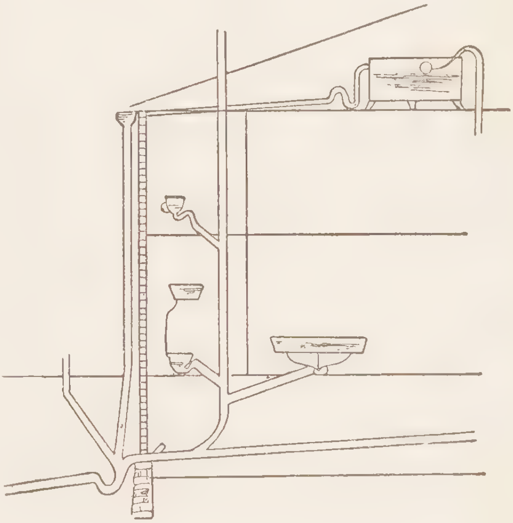
[1] Connecticut State Board of Health, Second Report, p. 48.

There is an abundance of a deposit of gravel and sand spread over large parts of the Northern States, which is a good soil for natural drainage. But in the case of a rocky ridge covered thinly with this soil, or of a hill which is rock at its north end and gravel at its south, there is a risk of sewage flowing into seams of the rock and finding its way under buildings. A recent epidemic of diphtheria in a large school at Lansingburgh, New York, seems to have originated in that way from privies placed on each side of the house, some twenty feet distant. A foul smell had long been complained of in the rooms over the cellar.

An intercepting basin of *tight* masonry is sometimes interposed to catch the solids and let fluids pass on to a cesspool. In one school where this is done, the liquids are discharged by a flush-tank through a system of drain-tiles underneath the front yard,—a grassy plat of five thousand feet,—with not only no offence, but with great improvement of the appearance of the lawn. There is no freezing in winter, though some of the tiles are within four inches of the surface.

Another plan is to discharge into a large body of dry earth in a covered tank, the fluids soaking through and passing to a meadow. The earth is frequently turned over with forks.

Fig. 20.



The following is a list of requirements for the drainage-pipes of a house (compare Fig. 20).

1. Outside drain of vitrified pipe; cement joints with gasket of oakum or puddled clay. When there is liability to settle, or when trees or wells are near, iron pipes with tight joints. Iron pipe where it passes through the house wall and for five or ten feet beyond. Not to run under any cellar.

2. Soil-pipe, or foul-pipe within the house, always of cast iron (some use wrought iron), four inches in diameter, carried by preference along the cellar wall, in sight; the perpendicular part rises without changing direction or size to a point two feet above the roof (not near a chimney or a window), where it may be left open, or capped so as to give least interference with draught. It should be extra heavy if the body of water is large. The inside is coated with coal-tar or enamelled. (The Bower-Barff process may be recommended.) The joints of lead.

3. The prolongation above the roof is for the purpose of securing a

continuous upward passage of fresh air, which disinfects the interior. The air enters by a vent just inside of the main trap, outside of the cellar.

4. *Traps.*—One between the house-system and the sewer, outside of all; and one at each water-closet, latrine, urinal, or basin. None between those points. The outer trap is a simple U-shaped bend (running trap). It can be placed at the bottom of a man-hole for convenient access; the rain-water may discharge into it, and the vent is close to it. Traps under fixtures can be "ventilated" to prevent siphoning, but the expense and complication attending this kind of work are so great that it is best to use a trap which resists siphoning.

Back-pressure of gas from sewers is obviated—1, by the outer trap; 2, by free escape up the vent and at the roof-end of the soil-pipe.

5. *Waste-Pipe.*—This is an upright pipe in all respects like the soil-pipe, but receives only water from basins; it is carried through the roof. (Not given in plan.)

No waste-water from baths, basins, etc., should empty into a water-closet trap. Each basin or group of basins should have its own direct discharge into soil- or waste-pipe.

6. Leaders (or rain-water pipes) may discharge into the main trap or otherwise. If they discharge beyond the trap into the drain, they need a trap. No leader should be used as a soil-pipe, and no soil-pipe should be used as a leader.

7. Cistern-overflows must not communicate with the drainage-system, nor with any place where the air is foul.

8. The entire system of discharge-pipe should be tested before connecting it with the fixtures, by plugging the lower end and filling the pipes.

All bends to have large curves; joints at acute angles.

No work to be covered in until approved by the inspector. It must be exposed to view as far as possible, for ready repair. If covered, the cover is of wood, easily removed.

FIXTURES.—Every school ought to have the means for washing face and hands: at least one stand on each floor or one for every two rooms. Plain porcelain-lined iron, marble-topped basins, etc.; they should not be boxed in.

Country schools should provide clean pails with covers. (Special cleansing is appropriate for drinking-vessels if any child has had diphtheria.)

If there is a water-supply, the choice lies between water-closets, latrines, and flush-tanks. Each has its merits. Neither will run by itself; each will become offensive in proportion as it is neglected or is kept unventilated.

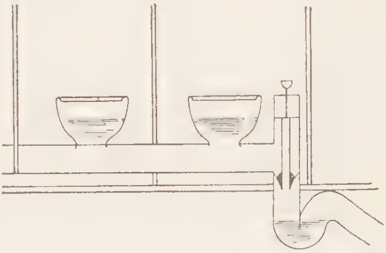
One remark applies to all: they must be constructed with the greatest simplicity, and expressly arranged for ease of cleaning. Wooden boxing about closet-seats is wholly needless, and the enclosed space is sure to be foul. The needful wooden seats, etc., should be fixed so that they can be easily removed with a screw-driver. Wooden floors should be well laid and thoroughly oiled; asphalt is better; partitions should be varnished or painted, seats the same if thought fit.

Water-closets are made in so many forms that it is useless to enumerate even the chief sorts. The worst form is the pan-closet, though often preferred on account of cheapness; it is complicated, easily put out of order, and accumulates internally a fund of ancient filth. Another bad kind is a hopper which dribbles and never carries its load through the trap.

There are many excellent but expensive closets with valves and plungers, made of earthenware, and suited to private houses. The best for schools is a short hopper closet, which contains no parts to get out of order (an oval basin with rounded rim, with water discharging under the rim downward all around). The only objection is that it sometimes requires a trifle of washing. Any closet chosen must have a sudden and copious flush of water. The supply should come from a tank, not directly from water-pipes. Noiseless action is desirable in certain situations.

A "latrine" is a long cast-iron trough, lined with enamel or not, and made deep enough to hold a few inches of water. A wooden seat is fitted to it. Some patterns are absolutely plain; others are a sort of compound of water-closet and latrine, as in Fig. 21. This class of apparatus is emptied very rapidly by lifting a plug at one end, made hollow to let off superfluous water.

Fig. 21.



Closets should be placed on every floor for teachers, or for larger girls.

"Flush-tanks" are latrines of cemented mason-work made with a rounded bottom and a grade down to the plug-end. If well made and kept clean, they serve a good purpose. They ought to be discharged once or twice a day, and then swabbed or broomed and hosed out. The inside may be coated with coal-tar. These are about the cheapest arrangement that can be recommended. They can be placed in the yard; they do not freeze in the short period of a school-session. If it is very cold, the water must be shut off in the cellar and the supply-pipe emptied while the tank is not in use.

Urinals are the most troublesome things about the house, but they can be so kept, even in the basement, as to give no offence. It is not necessary to play a hose over everything six times a day. Let the whole construction be very simple; a back and a foot-piece of slate, with or without a trough, are the essentials. The slate must be oiled before it is set up. The trough may be simply a hollow in the stone floor at the junction with the back. The apparatus for making a sheet of water flow over it is hard to keep in order; the more important thing is the daily service of the janitor, who washes it with a cloth, applying soap and hot water every few days as needed (afterwards going over it with a rag damped with kerosene, as I have seen done).

The floor of urinals should be of impervious material,—asphalt or slate, never brick or cement,—and should slope towards the outlet, so that a drench with the hose can be given daily with little trouble. The raised platform often interferes with this work.

Wooden urinals may be kept in tolerable order for out-doors by frequent painting of every part, but wood ought not to be used if slate can be afforded. Cast-iron urinals rust, and throw off the enamel, and the surface is not capable of being cleaned. Zinc and galvanized iron perish readily.

A foul urinal is intolerable. It is best to place these conveniences and water-closets outside in a protected shed with sheltered access, warmed by hot pipes if necessary. There is generally some slight fault to find if they are in the basement.

Good lighting is desirable, and ventilation by a heated flue is necessary if the basement is the place used. If there is a fall in the ground, the basement may stand free in the rear and enjoy the advantage of full-sized windows, which is much to be desired.

Supervision by the master is necessary, for the sake of good order and to keep the janitor to his work. Teachers and monitors should be close at hand in recess-times.

Automatic arrangements are common for giving a flush whenever a person sits down; or they may be arranged to discharge all at once, at desired intervals, by connection with an automatic flush-tank.

The country privy hardly needs description. If the friends of youth have any faith in precaution,—if they think that any measures are needed, or are likely to avail, in checking youthful immorality,—here is a good place for them to begin work.

Of all departments of school-hygiene this one most needs the personal control of a persevering man, either the teacher or some active neighbor. The authorities must first put things to rights, and then establish some understanding about inspection; for if a lady teacher declines to consider this a part of her duty, it can hardly be required of her.

Without revolutionizing matters, we should insist on good repair; good light; separate houses for the two sexes, with a high fence separating the paths (if there is one house in two compartments, separate recesses may be given); a solid path that can be shovelled clear of snow; the distance of say fifty feet from the house; a solid paved surface over which earth is spread, with subsequent frequent sprinkling of dry earth, and frequent (weekly or monthly) removal.

Finely-powdered dry earth is one of the best agents known for deodorizing refuse. The matters only require to be kept covered; earth has the power of absorbing and holding the foul gases. It should be used dry, and the bin or barrel for storing it should be sheltered. Ashes, or the scrapings of country roads, are good. A layer of from two to four inches is laid on the floor of the pit; the amount required afterwards is a pint for every time it is used.

The system is readily applied to the use of buckets or pails, as shown in Figs. 22, 23, from Massachusetts Board of Health Reports, 1876, 1883.

Fig. 22.

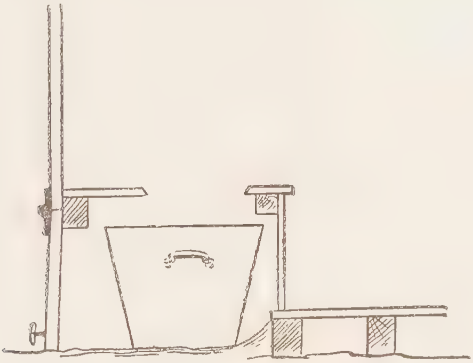


Fig. 23.

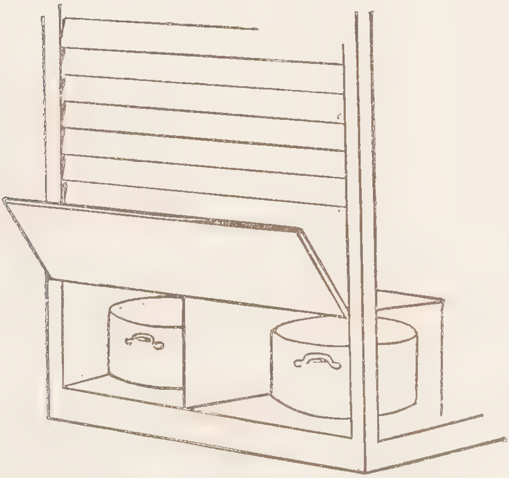


Fig. 24 shows a temporary arrangement, which might become permanent, described by E. S. Philbrick. The trench under the seats is about three or four feet wide, and six inches deep, with sloping sides. It is laid with coal-tar or asphalt and gravel well rolled, or with hydraulic cement and gravel smoothly trowelled with strong cement-mortar after hardening. The margin is higher in front. There is a flap-door behind. If carefully attended and cleansed weekly, it is a very satisfactory arrangement for using the earth system, "but otherwise it soon becomes a terrible nuisance."

A movable trough (Fig. 25) may be made of stout plank with calked seams, coated inside and out with coal-tar. It is on runners, and has a hook or ring to drag it with. It is two feet wide, and long enough to fill the space. It is treated with earth as before described, and removed from time to time, and the contents spaded into the ground or otherwise disposed of.

Fig. 24.

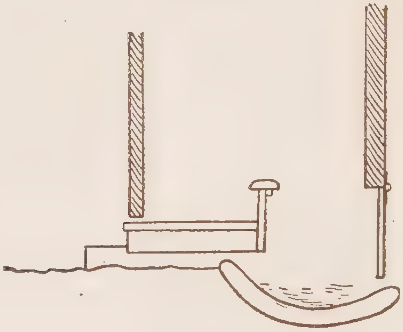
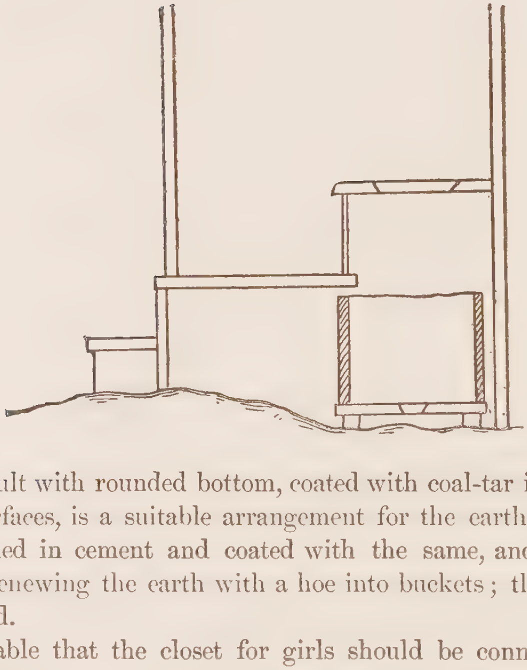


Fig. 25.



A brick vault with rounded bottom, coated with coal-tar inside and over all exposed surfaces, is a suitable arrangement for the earth system. The bottom is bedded in cement and coated with the same, and is built conveniently for renewing the earth with a hoe into buckets; that is, it is left open at one end.

It is desirable that the closet for girls should be connected with the house. The dread of exposure to the weather often prevents delicate persons from going out when nature demands it: there is no doubt at all that this circumstance gives rise to much ill health from constipation and retention of urine. The plan just described has the advantage of relieving this difficulty. The shed in question may be placed within four feet of the house, and the space between may consist of an enclosed anteroom, with a small window at each side, constantly open to give ventilation, and screened with blinds.

CONTAGIOUS DISEASES IN SCHOOLS.

The diseases against the spread of which by schools it has been thought necessary to legislate are (in this country) chiefly small-pox, diphtheria, scarlet fever, measles.

Whooping-cough, chicken-pox, mumps, German measles, are generally neglected in these precautions. Skin-diseases, as itch, ringworm, and vermin, are of some consequence, and yet, as involving no risk to life, they may be passed over by the legislator and left to the care of charity. The same has to be said of the far more important disease, contagious ophthalmia.

There is abundant evidence of the facility with which these disorders are spread by means of schools. This being popularly known, the first step used to be to propose to close the schools. Probably this has a good effect. But the real point to be aimed at, for the good of a community, is outside of schools, and consists in *isolating the patients.* When patients are also pupils, the function of the school as a co-worker with sanitary authority begins.

Towns, cities, and villages should have local Boards of Health, with power to make regulations to prevent the spread of contagious disease by preventing persons from attending school, also to suspend the use of the buildings and rooms when judged to be dangerous to the public health.

It may sometimes be desirable to waive this right in favor of Boards of School Control, but the superior right should belong to the Board of Health. Courtesy should not interfere with independent action in emergencies. A school, for example, might be closed by the health authorities before the school authorities could have time to act.

State boards should lead the local boards, and show their line of action. They might settle for practical ends the mooted questions of the time of safe return, the way of disinfecting, etc.

GENERAL REGULATIONS FOR PREVENTING THE SPREAD OF CONTAGIOUS DISEASES IN SCHOOLS.—1. Persons affected with diphtheria, measles, scarlet fever, or small-pox (or varioloid) must be excluded from the schools until official permission is given by the Board of Health for their readmission.

2. Persons living in the family or house where such a case occurs are also excluded until similar permission is given.

3. This permission is not to be given until sufficient time has elapsed since the occurrence of the last case to insure safety, nor until the premises have been disinfected under the direction of the Board of Health.

4. If a child suffering from one of the above diseases attends school, the premises of the school must be disinfected under the direction of the Board of Health before they are used again.

5. Physicians, teachers, school-officers, and school-children, knowing of such cases of disease, should at once report them to the Board of Health.

PERIOD WHEN RETURN TO SCHOOL IS SAFE.

	Diphtheria.	Small-Pox.	Scarlatina.	Measles.	Typhus.	Whooping-Cough.
J. Lewis Smith . .	Three or four weeks after symptoms have abated, person disinfected, no redness of fauces, or coryza.	At least eight weeks.	Eight weeks, complete desquamation, disinfection, cleansing.	Very volatile; safe in two weeks after convalescing.		Not determined; probably when no longer spasmodic.
Austin Flint . . .		Danger not extinct till all traces of cutaneous affection have disappeared and everything has been removed from surfaces of body by repeated baths or ablutions.				
A. Clark		Till healing is complete and cicatrices fully healed.	May be six weeks.	Probably a week at least after desquamation.	Short interval.	
F. Minot	Four weeks from the beginning of the last case in the family.					
Anna Lukens . . .	After disappearance of membrane.	Desquamation and disinfection.	Desquamation and disinfection.			When no longer spasmodic.
F. A. Burrall . . .			Desquamation and disinfection of person and property.	(Other diseases on same principles.)		
A. Jacobi	Last trace of inflammation or ulceration secondary to or attending the diphtheritic process; time uncertain.	Desquamation.	Desquamation. Forty days sufficient.	Eighteen days.		When no longer spasmodic.

6. The Board should also notify the school-authorities of such cases.

7. Notice must be sent to the family by the school-authorities, acting conjointly with the Board of Health.

It is hard to say with certainty how soon a patient may safely return to school. The variation in opinion among masters in medicine is shown by the table on the opposite page, taken from answers returned in correspondence with the New York State Board of Health.

The Medical Officers of Schools Association of England, in a code adopted January, 1885, name the following periods after which pupils who have had diseases may safely return to school.

Scarlet fever, not less than six weeks from date of rash, *if* desquamation have completely ceased and there be no appearance of sore throat.

Measles, not less than three weeks, *if* all desquamation and cough have ceased.

German measles (rötheln, or epidemic roseola), in two or three weeks, the exact time depending on the nature of the attack.

Small-pox and chicken-pox, when every scab has fallen off.

Mumps, four weeks from commencement, *if* all swelling have subsided.

Whooping-cough, after six weeks from commencement of whooping, provided the characteristic spasmodic cough and the whooping have ceased, or earlier *if* all cough have completely passed away.

Diphtheria, not less than three weeks, when convalescence is completed, —there being no longer any form of sore throat, nor any kind of discharge from the throat, nose, eyes, ears, etc., and no albuminuria.

Ophthalmia, until there has been a complete absence of discharge for at least one month, or until the inner surfaces of the eyelids are found on inspection to be quite free from granulations.

SMALL-POX.—In the Seventh Report of the Illinois State Board of Health there is an illustration of the way in which public schools may be made a vehicle for sanitation upon a large scale. In November, 1881, the Board ordered the vaccination of all public-school children. When the order went into effect (January 1, 1882), nearly sixty-nine per cent., or over four hundred and ninety thousand, of the enrolled scholars in Illinois were either unvaccinated or were susceptible to contagion through neglect to re-vaccinate at the proper time. By March 1, 1882, there was less than six per cent. of unprotected and susceptible remaining among those in attendance; the frequency of small-pox and varioloid was lessened more than one-third among school-children, and the mortality was reduced from sixteen and one-fifth to three and one-third per cent. During the four years 1880–83 the deaths among unvaccinated school-children were forty-eight per cent., while among the vaccinated they were nine-tenths of one per cent.

Under the statutes of Illinois it is the right and the duty of the State Board to make all rules and regulations which they deem necessary to preserve the public health. "Such rules and regulations when promulgated have the force and authority of law, and are to be enforced, if necessary, by the entire power, including school-officers, etc., of the State." This quotation is taken from an opinion of the Attorney-General of the State.

The school-directors are the immediate source of authority for the action of the teachers, under this law. Teachers should have been revaccinated within four years in any case.

The law of Massachusetts represents a different type of practice, providing that parents and guardians shall cause their children to be vaccinated under the age of two years, and revaccinated when the selectmen or mayor and aldermen require it (not less than five years having elapsed since previous vaccination): it leaves the enforcement of the law in the hands of these officials. In individual towns and cities the matter is left (as regards scholars) in the hands of the school-authorities, and with very varying results as regards enforcement, though in some cases strict account is taken.

Regulations for Vaccination.—1. Every child entering the public schools must show a certificate from some reputable physician, giving name, age, residence, approximate date of vaccination, date of examination, result of examination; the last two to be of the physician's own knowledge.

2. The fact of vaccination must be entered on the school-record, and on lists for promotion or transfer.

3. The school-authorities shall annually report the number of those not protected to the State Superintendent of Education.

4. School-authorities may order the exclusion of non-protected persons, at sufficient notice, where they think the measure required for the public health.

5. Revaccination at the age of fifteen may be required under similar circumstances.

6. Those unable to pay should be furnished with free vaccination by the school-authorities.

7. A physician's certificate of protection by a previous attack of small-pox is equivalent to a certificate of vaccination.

SCARLET FEVER.—This disease is one of the most destructive. It caused, by the United States censuses of 1850, 1860, and 1870, the proportions of one-thirty-fourth, one-fifteenth, and one-twenty-fourth of all the deaths. In England and Wales it causes, on an average, one-twenty-fifth of all deaths. It is very contagious. It often leaves behind it very serious injuries, even after apparent recovery. To keep children from having it is a parent's duty, if possible. An instance of what the government may do in the way of checking it is probably furnished by the following account.

The Boston Board of Health in 1877 established a regulation requiring children from infected houses to be kept out of public schools, and requiring physicians to report their cases of scarlet fever. Since that time the number of deaths from scarlatina has varied from year to year in the most irregular way. But, taking years by groups, it appears that the relative number of deaths has much diminished, as is shown by the following table:

Periods.	Average Population.	Average Deaths by Scarlatina per Annum.	Ratio of these Deaths to Population.
1861–67	189,000	172	1 in 1100
1868–76	295,000	323	1 in 913
1877–85	375,000	115	1 in 3261

Diphtheria.—Children at school may take diphtheria from one another in a variety of ways,—by using the same cup to drink from, by the practice of putting pencils and marbles to their mouths regardless of whose mouths they may have visited previously, by turning pages of books with wet fingers.[1]

"Diphtheria may be diffused by the exhalations of the sick, by the air surrounding them, or directly by the exudation, communicated in the act of kissing, coughing, spitting, sneezing, or by the infected articles used, as towels, napkins, handkerchiefs, etc. The poison clings with great tenacity to certain places, rooms, and houses, where it may occasion cases after the lapse of months."[2]

These opinions are quoted to illustrate the variety of the dangers that attend school-intercourse with an infected person. They are also pertinent in view of the doubt entertained by some as to the contagiousness of the disease,—a doubt honestly held.

In epidemics of diphtheria all cases of sore throat must be looked on with suspicion, more particularly if the children are feverish and depressed. Teachers should take note of this.

Contagious Ophthalmia is of frequent occurrence in children's asylums, and occasionally in primary schools. It frequently causes blindness or great injury to sight. Its existence among the inmates of an institution as an epidemic is due to carelessness about admissions, to overcrowding, poor food, and other causes of enfeebled health. One of the chief ways in which it spreads is by the children's washing together in the same water and using the same towel.

Funerals are a fruitful source of contagion. It may not be amiss to say that funerals *must not be held in the school-house*, as seems to be a custom in some places.

Children who have been exposed to any contagion may be ordered to remain out of school for a limited time, in the judgment of the Board.

Closing school is a measure that seems needless in a place where rules about exclusion are well enforced. It seems to have a beneficial effect sometimes, as the prohibition of public meetings does.

It is doubtful if contagious fever is often carried by library-books; or, rather, the known cases must be very rare.

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[1] Michigan Board of Health.

[2] Report of Metropolitan Board of Health, New York, 1873, p. 584.

In boarding-schools there ought to be a sick-room in the upper part of the house or in an isolated place. A pupil attacked with contagious disease should be at once isolated and all his effects disinfected. Communication of all sorts is to be cut off between pupil and comrades, and great care taken about food, clothes, and all things that come from the chamber. The parents are to be notified. If a considerable number of cases occur, or the disease is malignant, the parents of all pupils should be notified, that they may remove their children if they choose.

The school should have its own medical attendant, who is to take all steps necessary in epidemics.

It may be well to subject pupils to a delay, if when school opens it is found that they have been exposed to some contagious disease. This may prevent an outbreak in the school. The period required may be as follows, dating from the day of exposure:

Diphtheria,	12 days.	Chicken-pox,	18 days.
Scarlatina,	14 “	Small-pox,	18 “
Measles,	16 “	Mumps,	24 “
German measles,	6 “	Whooping-cough,	21 “

Boarding-schools ought to have stated reports made upon their sanitary condition: local or State Boards of Health might properly undertake the work. It is a matter which will repay investigation.

CONSTRUCTION OF CHILDREN'S HOSPITALS, ETC.

By LINDLEY JOHNSON.

THE study of the diseases of children has occupied so conspicuous a place within the past few years that the advantages of special institutions for their treatment are conceded by all.

Without discussing the history of hospital construction, I shall briefly consider a few points connected with the general design which should be the features recommending all plans where excellence in construction and utility are sought for. From these points the plan should develop (1) a well-selected site; (2) properly proportioned and isolated wards; (3) central administrative department; (4) detached kitchen and water-closet buildings, unless the kitchen be on the top floor; (5) careful study of heating and ventilation combined; (6) sanitary properties, including water-supply and disposal of all waste or soil; (7) fire-proof or slow-burning construction, except in cottage work.

I shall refer more particularly to the planning and construction of hospitals, as offering a better subject for the treatment of these more important features; these remarks, with the modifications suiting the requirements, may be equally applied to the construction of asylums, nurseries, etc.

SITE.

The location of a hospital, whether city or suburban, should be determined upon only after a most exhaustive examination of the immediate site, its surroundings and its climate.

CITY.—One would likely imagine that a hospital would be best situated in the centre of the most densely populated districts; yet on sanitary grounds this is not to be recommended.

It should be located far from the influence of neighboring nuisances. It must be easy of access. It is best situated upon high ground, to facilitate surface-drainage. It should be surrounded by an open clear space, to secure a permanent and free flow of pure air and sunlight, with an exposure towards the south. The grounds about the building should be tastefully laid out by a landscape gardener, for the double purpose of purifying the air and affording a cheerful outlook for the patients. A corner lot naturally offers the greatest chance for air and light.

The nature of the subsoil should be carefully determined, as the footings of the walls should bear upon a good bed of gravel, sand, or rock; clay is bad as a bottom, and may cause damp walls above, resulting often in serious trouble. Where the masonry of outer walls is laid in ordinary lime mortar in place of cement, the evils of a clay soil cannot be overrated. I have examined walls laid under such conditions, and have seen a complete disintegration of the mortar to a considerable distance above the first-floor joists. The interstices occasioned by the loosening of the mortar afford a convenient retreat for insects of all kinds, and admit dampness and wet from without.

In making a selection of a site for a large city or in a manufacturing town, I would give preference to a site in the south or west end, where the summer breezes are less likely to be contaminated by the foul air of neglected roofs and imperfect sewers.

COUNTRY.—If a site be selected in the country, it should have ample free space on all sides. There should be no marshy grounds within a reasonable distance. An abundance of absolutely pure water must be one condition governing the choice of a site, and it will often prove a serious question. If the supply be drawn from wells, there are two points of importance to be determined: first, that the water be of the proper quality and not contaminated by any surface drains or wastes; secondly, that the supply be sufficient to meet the demands made upon it in the autumn following a dry season; a practical test by the application of a steam-pump is the surest means of ascertaining the latter point. Where running water can be utilized as a motive power, I would recommend the use of a wheel or ram for forcing the water to the tanks. This will give a steady flow, changing the water in the tanks continuously, thereby reducing the temperature and increasing the chances of a pure supply; besides which it affords an excellent opportunity to construct a cold vault for the keeping of milk, butter, etc., of great value to most country hospitals.

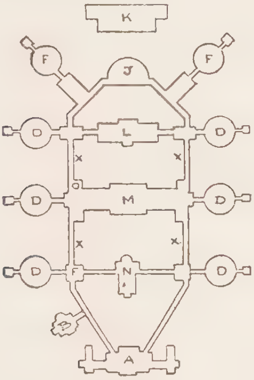
The location should be elevated, but easy of access by means of carriage. A site to the south or west of a large city is preferable to one to the north or east. The local climate should be healthy, the soil dry and porous.

The Chirurgical Society of Paris, after an exhaustive study of the subject, determined "that only small hospitals for urgent cases and those required for clinical instruction should exist within the city limits, and that not only would the salubrity of the larger hospitals be improved by their extra-urban position, but also their construction would be rendered more economical, by means of the reduced cost of land."

PLAN.

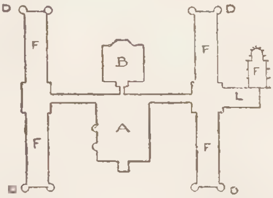
In both this country and in Europe the "pavilion style" of hospital, whether applied to large or small buildings, has been generally recognized as the one embracing the greatest advantages. By "pavilion style" are meant detached wards. The pavilions may be constructed several stories in height and of various dimensions, but under every condition the ward must be taken as the *unit of construction.* The heating and ventilation, the cubic contents, and the general dimensions throughout, to be satisfactory, should be studied and developed with reference to this unit. This should be the more carefully observed in the studying of children's hospitals, since children are more susceptible to inequalities of temperature. (Figs. 1, 2, 3, 4, and 5.) In no other plan does the location or shape of the ward allow

FIG. 1.



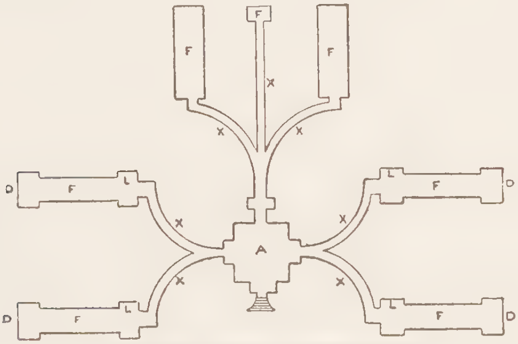
Antwerp Civil Hospital (Belgium).

FIG. 2.



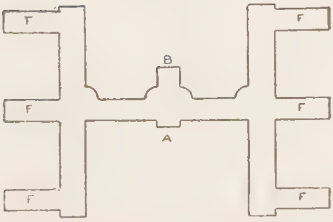
Norfolk and Norwich Hospital (England).

FIG. 3.



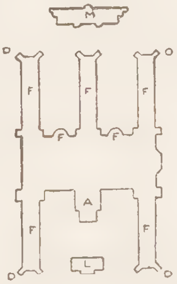
Boston Free Hospital (Massachusetts).

FIG. 4.



Bradford Hospital (England).

FIG. 5.



Leeds General Infirmary (England).

BLOCK-PLANS OF LEADING PAVILION HOSPITALS, SHOWING VARIOUS TREATMENTS OF THE WARD.

of so perfect a circulation of air and light with the same isolation from the administrative block, lessening thereby the danger of hospital diseases.

WARD.—Whether the pavilion, corridor, or block system be adopted, the ward should be studied as an independent feature. The rectangular ward has most to recommend it as regards shape.

The area must be in proportion to the floor-space allotted to each patient, varying, of course, with the climate and the object of the hospital. Among the best-known hospitals the allowance per bed of floor-space varies from about seventy to one hundred and thirty-eight square feet. The new Hôtel-Dieu allows about one hundred and seven square feet, and the results have been very satisfactory. In fever hospitals, or in wards for bad surgical cases, a floor-space of two hundred square feet per bed may be required. The proportion of floor-space and cubic contents will be regulated with reference to the shape of the ward, the system of heating and ventilation, and the condition of the patients.

The length of a ward, to obtain the greatest economy in service and convenience, must not exceed four times its width. The width must not exceed thirty feet,—from twenty-five to twenty-eight feet is preferable, suiting the conditions. The height of wards must be determined by the required cubic contents, the system of heating and ventilation, and the climate. Under ordinary conditions in this climate fifteen or sixteen feet is proper.

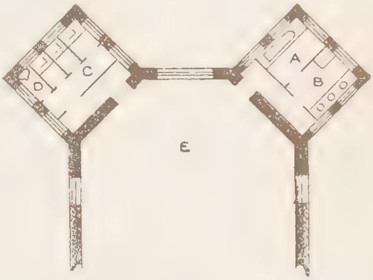
There must be windows opposite one another in the two long walls, allowing of one bed between every two windows. The end away from the administrative quarters must have windows opening onto a veranda or clear, and corner doors opening into passages, with cross-ventilation, connecting at one corner with water-closet-urinals, and slop-hopper, and at the other corner with bath, sink, and basins. The doors leading into the ward are best double, that one-half may be bolted for ordinary use. A transom above is desirable. The windows should run up close to the ceiling and extend down within three and a half feet of the floor. They should be lifting-sash, with hinged transom above, and the frames constructed to admit of double sash in winter.

The wards are generally thought best situated with the longitudinal axe running north and south; but there are those who take exception to this, and argue that experience has proved that with the longitudinal axe running east and west the summer temperature is reduced materially, whilst the good results obtained by exposure to the winter sun are not lessened. Naturally, these conditions will be governed more or less by the latitude in which we build. Care should be taken in locating the work to run the wards so that the two longer walls shall have a daily exposure to the sun. This should be particularly observed in hospitals for women or children. The wards should be parallel, or nearly so, and the distance apart should not be less than twice the height of the pavilion.

Large wards are generally more healthy and comparatively less expensive than the smaller ones. About thirty beds is considered the most satisfactory number in every respect for general hospitals. Each pavilion should consist of two stories, with a basement below. There is often necessity for the construction of pavilions of greater height than two stories, more particularly in large cities, where the ground cannot always be secured for extending horizontally, and there may be economy in building to a height of four or five stories. In the country or at the sea-shore, where small cottage hospitals are desired, it will be found often best and cheapest to construct them of but one story.

ADMINISTRATION DEPARTMENT.—The central block should contain all the requirements of the administration department, such as nurses' quarters, committee-room, operating-room, single wards where it is not possible to isolate them, an easy staircase to upper floors, kitchen and scullery, etc., —which are best detached, or *located in the top floor,*—a good lift service for *food and linen,* a dispensary, water-closet and baths for the nurses, patient's room, consulting-room, etc. The engine-room, laundry, and dead-house are best detached and distant. The sick-wards must be accessible, yet well separated. The main entrance, hall, stairway, and cross-corridors must be well lighted.

FIG. 6.



Faulty isolation of ward annex.

This block may be three or four stories in height; and I would strongly recommend putting the kitchen department on the top floor, freeing the lower floors from any possibility of annoyance therefrom.

FIG. 7.

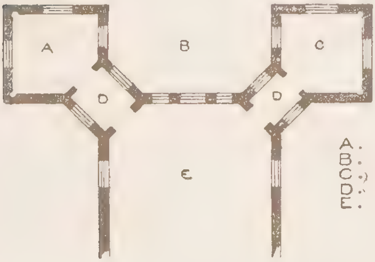
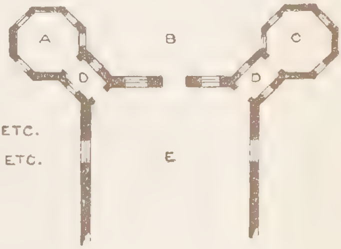


FIG. 8.



Proper isolation of ward annex.

The location and isolation of the ward (Figs. 6, 7, and 8), water-closet, and baths are sufficiently explained in the cuts of ward ends. Their construction and details will be explained under the heading of plumbing, etc.

HEATING AND VENTILATION.—The heating and ventilation of a general hospital offer the most difficult problem to be solved,—a point, too, of equal importance with the plumbing and drainage. They are inseparable from a satisfactory result. Whether we introduce a system of hot air, hot water, or steam, it must form a part of and assist in the ventilation.

The more common forms of heating are by (1) open fireplaces and stoves, (2) porcelain stoves (much used in the north of Europe), (3) the calorifere in use in France and Southern Europe, (4) the hot-air furnace, (5) hot-water circulation, direct and indirect, (6) steam heating, direct and indirect.

For small cottage hospitals, etc., it is doubtful whether other than open fires, stoves, or possibly a single portable heater, could be used, on account of the expense of both the original cost and the maintenance. When they are used, however, every care should be taken to introduce an ample supply of fresh air, that it may come in contact with the radiating surfaces before it enters the ward, and be extracted as soon as it has performed its duty. There are various forms of stoves manufactured to meet this need, the principle of which is to run the smoke in a pipe inside of another flue extended vertically to the top of the building, and connected horizontally with ducts led to registers at or near the floor, from which the foul air is withdrawn. With an open ceiling, ceiling-ventilation may also be introduced, and for warm weather it will be most desirable.

For general hospital heating we must, however, enlarge our plant, and either steam or hot water seems to meet with the greatest favor. The two systems are similar, and both have much to recommend them.

The first cost of a hot-water plant is somewhat greater than that of steam. The consumption of coal is less. The great advantage in hot water over steam is that a moderate fire will produce a circulation and a moderate heat, and the heat is retained longer after the fire is drawn.

The advantage in steam is that the temperature is more quickly raised, and where *direct-indirect* radiation is necessary the result is better, I believe. There is also with steam a power that may be used for other purposes : it, however, requires more careful supervision than other forms of heating. A combination of direct and indirect heating is more desirable in any event, the direct radiators being located in exposed positions and the indirect heat introduced into the wards, etc.

After determining upon a system of heating, we have to consider its introduction, circulation, and exit.

The fresh-air-supply must be taken from above, conducted in a cold flue of ample size, and run in a duct to the heater. From the heater it is run in terra-cotta pipes or a galvanized iron box horizontally from the boiler to each end of the basement, giving off branches which are run to the base of all hot-air flues, where a bunch of radiators are located, enclosed within a galvanized iron box. This warm air rises in the flue provided for it to the hot-air register, where it finds its way into the ward above the line of the head in the outer walls. These flues and registers must be large enough to allow of a ready flow of warmed air without the inconvenience of a rush. Upon the north and west walls larger heat-flues may be introduced to advantage. The problem of heating and ventilation to secure satisfactory results is such a complex one, involving such a thorough familiarity with the laws of mechanics, that the responsibility of its proper solution should be placed upon an expert, and the plans modified to meet his requirements. It is of equal importance with the question of plumbing and drainage, and likely to prove more difficult of solution.

The outlets for foul air should be just off the floor, and of carefully-studied dimensions. They are run in flues to the basement floor, connected with a horizontal duct and led to a large vertical heated flue, by means of which the foul air finds its escape above the building. Where the engine-house is located *outside*, the foul air is best run to it and the smoke-flue utilized. A fan may be required for the extraction of the foul air. Exhaust registers should be provided also near the ceiling, for summer ventilation. Where direct radiation is used, the radiators may generally be placed against the outer walls, immediately below the windows.

PLUMBING, DRAINAGE, ETC.—The ward water-closets, urinals, sink, slop-hopper, tub, and basins should be collected in extensions at the extreme outer corner of the wards and separated from the wards by a narrow passage having a cross-draught. This should under no circumstances be omitted. These annexes should have windows in three walls at least. The floor should be tiled and all the fixtures exposed.

Sanitary appliances and plumbers' supplies are being so constantly improved that what one may recommend to-day is likely to be discarded to-morrow. The entire question of plumbing and drainage, more particularly the disposal of sewage, is unsolved and unsatisfactory, and I believe a few years will introduce radical changes which must prove beneficial. No one with the proper sense of smell or taste will hesitate to doubt the benefit of having a lot of soil-wells about the property, trusting to water-seals to exclude from his house the deadly gases generated, which often have no means of escape except through these very traps. Then the question of water-supply must be considered in connection with the soil. It may be taken from a well three hundred feet deep and at the same distance from the nearest soil-well, and yet it may drain the soil-well of every drop of liquid soil. Where we are enabled to enjoy the privileges of a city sewer, we often congratulate ourselves, but without reason. It needs but the simplest examination of our surroundings to realize what fearful disadvantage we live under when we make that fatal sewer connection and incur the responsibilities that must follow. We have, in addition to the danger incurred by running our soil-pipes and trusting to traps, our wastes from all sinks, etc., which must find the same outlet and increase thereby the number of possible escape-passages for sewer gas, from either a defective trap, evaporated seal, or siphonage.

But a far greater and more general evil in connection with the sewer is the contamination of the fresh-air supply. When in-doors we practically live upon the air drawn from either the front or rear pavement, possibly within eight feet of a sewer inlet that may be rank with poison: there are certainly within a stone's throw half a dozen such inlets, which are bound under certain conditions to rob us of anything like pure air. But, not content with this form of impurity, we run this air direct to our heaters, develop all the impurities it contains, and then distribute it lavishly throughout our houses, imagining we have done our duty. With our present system of city sewage it should be made a criminal offence to take the fresh-air-supply except from the top of the building, whence an extra brick flue should carry it to the heater.

The water-closets should be siphon closets, with seat-vent and flushing-tanks. The tub may be iron enamelled, the slop-hopper of porcelain. The soil-pipe must be extra heavy iron, with the joints calked and leaded, and extended full size above the roof. It should run down and be extended through the basement wall ten feet in the direction of the well, where it may connect with a heavy glazed and vitrified terra-cotta pipe run to the well. This pipe should have a running trap close to the well, with a fresh-air inlet on the house side of the trap. All waste from sinks, tubs, etc., should run into a grease-trap built of brick and cement, and the overflow run into a separate well. Anti-siphon pipes should be provided for all fixtures. The seat-vent from the water-closet should run into a warm flue. In city work we naturally connect with the sewer, and have to trust to a system of traps too apt to be defective from one cause or another.

CONSTRUCTION.—The usual form of construction not only in hospitals, etc., but in nearly every class of building, is so radically defective that I wish to call particular attention to it. I refer particularly to the construction of the floors.

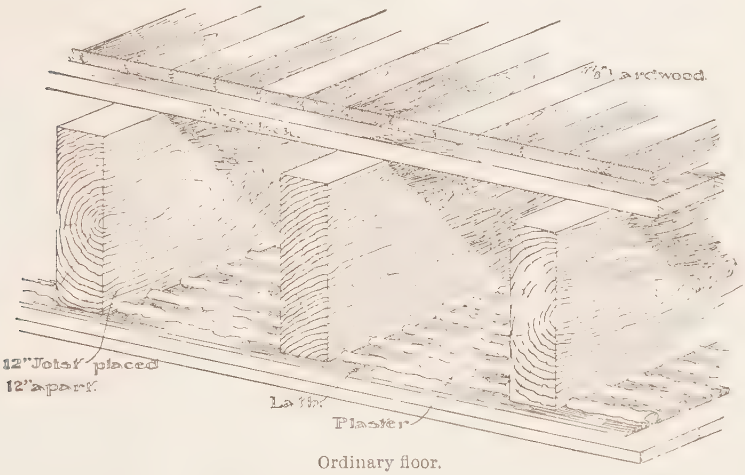
I will take for example the floors in a pavilion, say thirty feet by one hundred and twenty feet, of two stories in height above a basement. Where a fire-proof construction is not used, the joists are likely three by twelve inches, spaced about twelve inches apart, sealed below with lath and plaster, and covered above with one or two thicknesses of floor-boards. Here we have in the three floors a sealed space of over nine thousand cubic feet, to collect all manner of impurities. If well constructed, it is practically air-tight, causing dry-rot in the timbers.

The construction of the roof offers generally the same objection. This is not an imaginary danger, as the statistics will show where the origin of fires has been investigated; and this cause can doubtless be extended to such fires as have left too little to investigate.

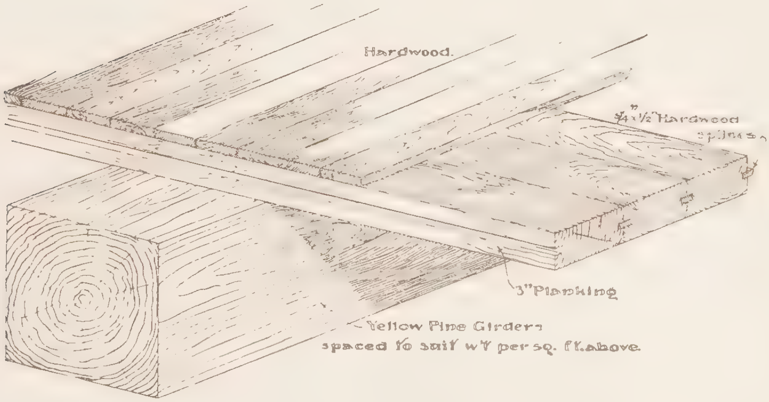
Why not employ mill construction? The cost is greater, but by no means proportionately to the additional safety secured, particularly in those buildings where children or the sick are sheltered. By referring to the accompanying sketches of the two floors (Fig. 9), the ordinary and the mill construction, a fair comparison may be made of their relative advantages.

The spacing of the girders can be reduced or increased, suiting the floor-weight allowed for. The entire building so constructed would not offer a space of six cubic inches not open to the air and an examination, reducing thereby the danger of disease, vermin, and fire. Where mill construction is used, the wood should not be filled, oiled, or varnished until all moisture has evaporated.

Fig. 9.



Ordinary floor.


Hardwood.
¾" × ½" Hardwood splines.
3" Planking
Yellow Pine Girders
spaced to suit w't per sq. ft. above.

Mill-constructed floor.

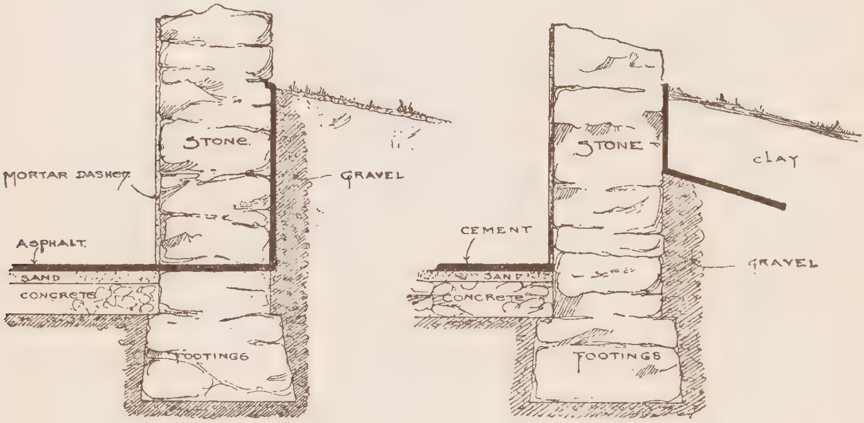
The outer walls should be constructed of stone or brick : the latter is preferable. Hollow walls I should recommend as a precaution against dampness, and to dispense with furring strips, though I should also advise asphalting the floor of the basement and running a layer of asphalt up and through the basement-wall. The trenches should be filled in with a gravel or sandy soil that will not retain moisture. (Fig. 10.)

The inner partitions are best built of hollow bricks : they are light, fire-proof, strong, and require no lath for plastering.

The main stairs should be constructed of stone and iron and made fire-proof. In the ward floors I should advise narrow white-oak boards, beeswaxed and polished.

The treatment of walls and ceilings has ever been a source of trouble and dispute. I believe a soapstone finish the best, rendering the wall hard and practically impervious to the germs of disease. This I think preferable to the construction in use in many German hospitals, where the inner linings of walls are torn out about once a year and renewed, necessitating the disuse of the ward,—a good feature in itself, but a great inconvenience and expense,—and creating the circulation of a great amount of dirt and impurities.

FIG. 10.



ASYLUMS, SPECIAL HOSPITALS, ETC.

Country sites for asylums are always preferable to city sites, since they afford an opportunity for surrounding the buildings with grounds where the inmates may find work.

The main features which I have alluded to in connection with the construction of country hospitals may be equally applied in the construction of asylums.

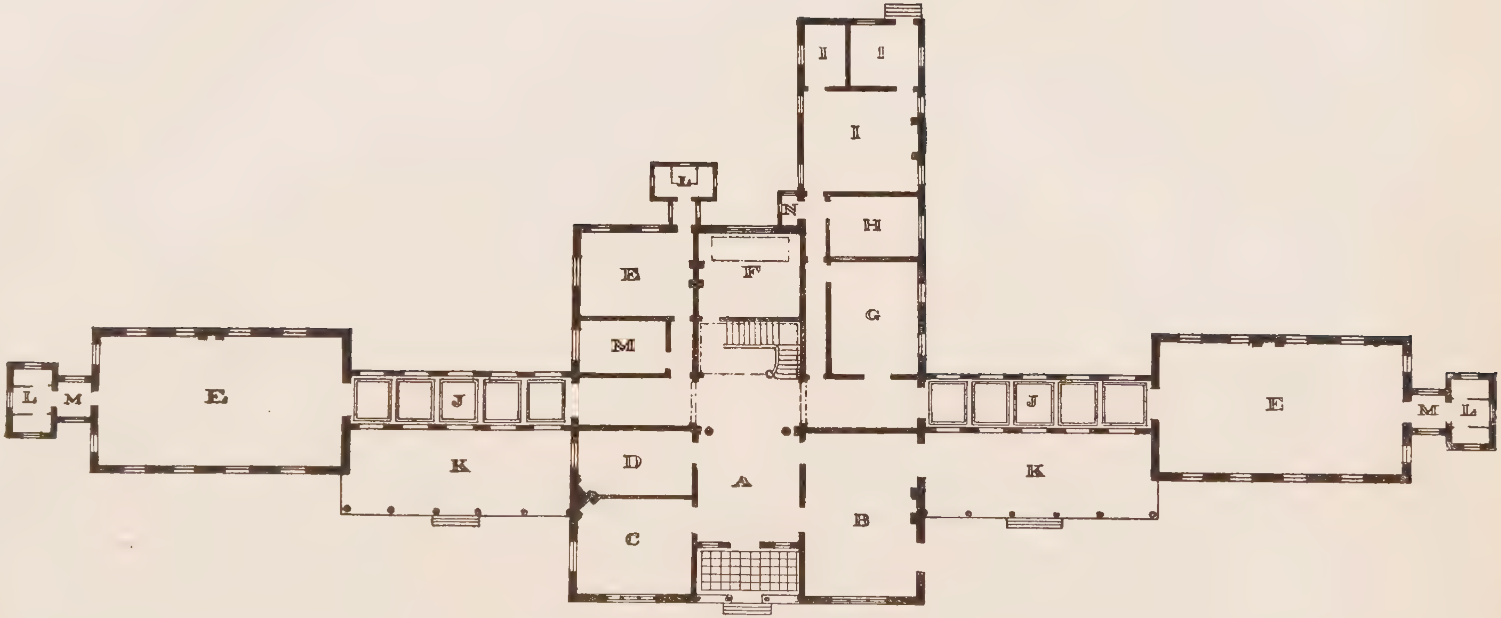
There is not the same necessity, however, for the isolation of wards or ward-annexes that exists in the former. Separate and single wards are, nevertheless, indispensable for violent cases, and the walls in such cases must be padded. All ward windows must be securely guarded with iron sashes or bars. The ward guardian should have a day-room commanding the ward over which he has supervision.

CONVALESCENT HOSPITALS.—There is a general want of hospitals or homes where patients may be removed from a general hospital before being in condition to return home, where often the poorer classes are subject to the drawbacks of defective sanitary construction, poor food, and insufficient heating.

This should differ chiefly from the general hospital in its increased facilities for the recreation of the patients. Enclosed promenades, larger day-rooms, etc., should be provided, and for the general interior arrangements a more home-like effect should be studied than is common to the

PLATE I.

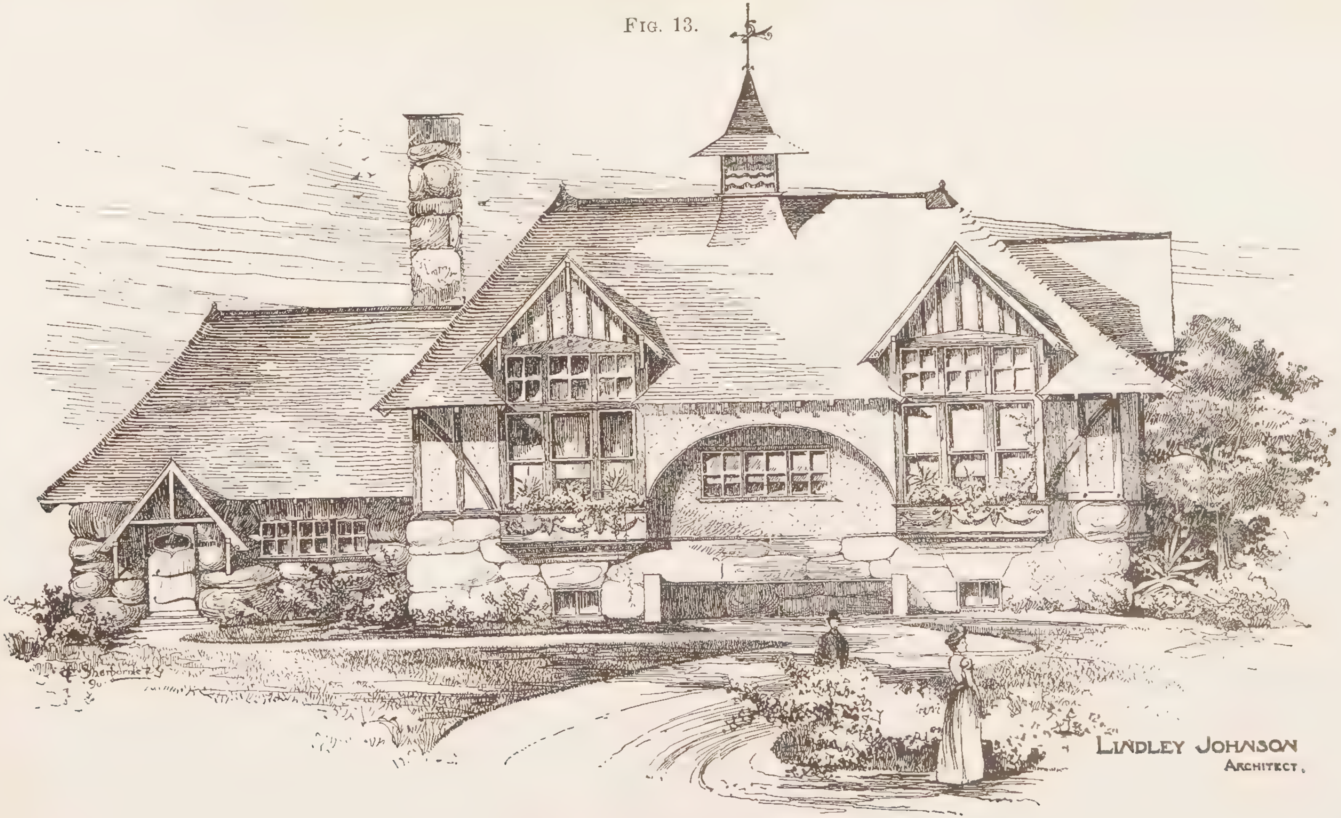
FIG. 11.



PLAN: FIRST FLOOR. A TWENTY-BED HOSPITAL.

PLATE II.

Fig. 13.



Sketch: Small Country Hospital.

general hospital. The building represented in the accompanying sketch (Plate I., Fig. 11) is suited to the accommodation of about twenty beds, unless the upper floor be required for additional small or single wards. A common day-room is sufficient, and it must have a cheerful outlook. A small dispensary should be connected with the building, particularly if in the country. There is no objection to the construction of two-story pavilions if additional beds be required.

Probably the best buildings of the class are those about Paris, notably at Vincennes.

Bienfaisance.—A most admirable type of building for charitable purposes is the Établissement de Bienfaisance, inaugurated in France by Dr. Gibert, and very ably illustrated and described in a publication of Dr. A. Foville, published in Paris, 1888, "Les Nouvelles Institutions de Bienfaisance." These institutions were first inaugurated in 1875, since when others have been founded, extending their charity to a wider field. They now afford free treatment and free remedies for all children who are in condition to go to and from the institution. They furnish to those food, medicines, baths of every description, massage treatment, gymnastic exercises, and in fact all that is likely to assist in the development of crippled body,—physical or mental; and they have done an incalculable amount of good to the children of the poor, who would be denied in their homes the necessary food and medicines and who could not afford to give up their time entirely to hospital treatment,—just that class who are ill, yet about, and too poor to seek advice. In some of these institutions the treatment and medicines are furnished free, while in others a nominal charge is made. The medicines are generally supplied the superintendent at a reduced rate, and medical assistance is often volunteered.

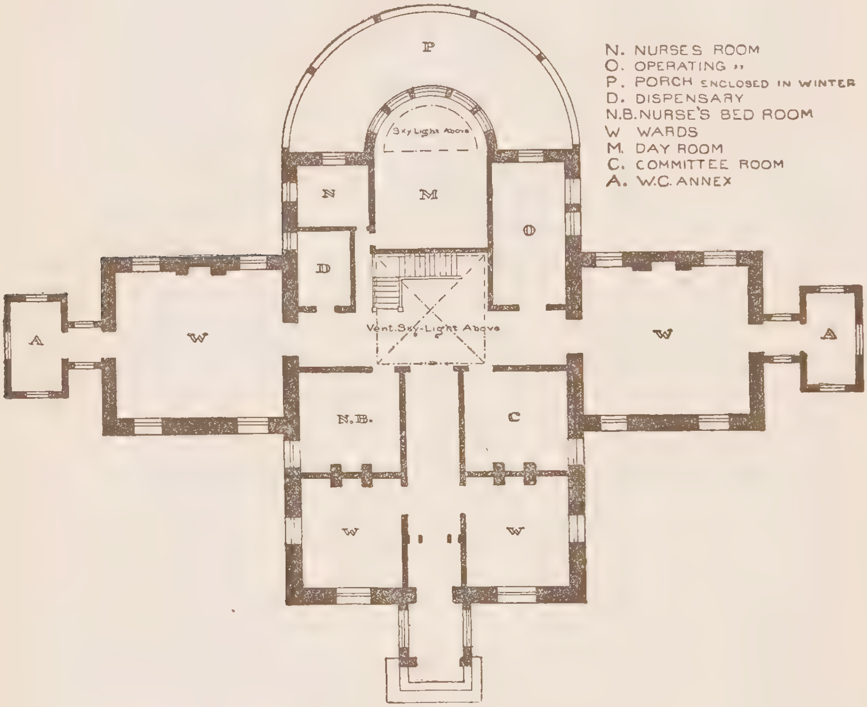
Cottage Hospitals.—Cottage hospitals, whether for the shore or the mountains, offer the architect somewhat more latitude for the development of individual taste without in any way sacrificing the features to be most considered. (Fig. 12.)

They are often built of frame or half timber with shingled roofs, where a good combination of colors and materials will lend to the structure a picturesque effect. (Plate II., Fig. 13.) Porches or balconies must be provided, and projecting bays and gables can be introduced, helping the elevation with bold shadows and good sky-lines.

Being constructed often without cellars, open fireplaces are indispensable, enabling a feature to be made of the chimneys. Mill construction is not likely to be considered in their design, owing to the additional cost and the light walls, which would scarcely justify such concentrated weights. The plans should contain single and double wards, and wards where eight or ten could be treated. They may be heated by stoves, in which case an abundant flow of fresh air must be introduced around the radiating surfaces. The smoke should be carried off in an iron pipe enclosed in a large brick flue, into which the foul-air-duct must empty.

At the sea-shore the drainage and the water-supply are likely to offer many drawbacks. Where no sewerage system is in use, we must resort either to a well-discharge, taking care to locate it as distant as possible from all supply-wells, or we must run it off to sea in iron pipes beyond low tide. In this event I should recommend seeking the advice of a local engineer.

FIG. 12.



COTTAGE HOSPITAL TWELVE BEDS.

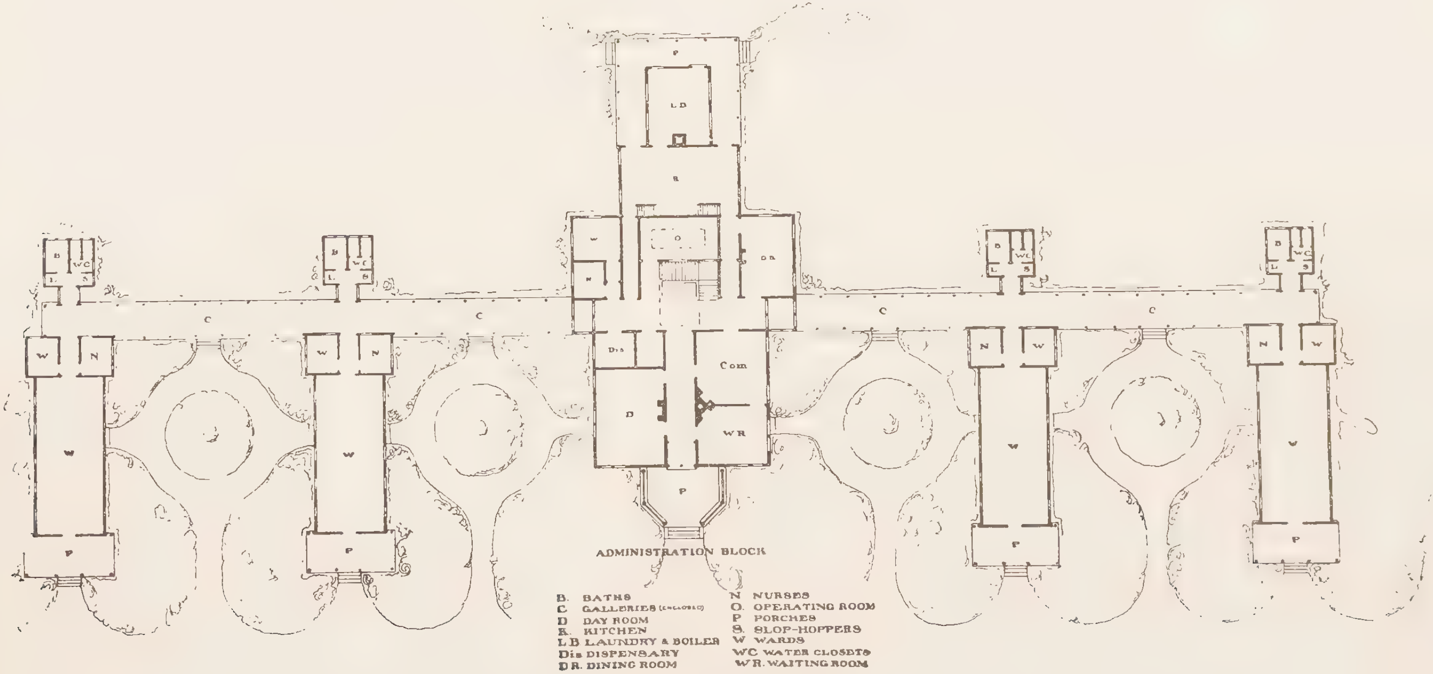
For the sea-shore a sea-exposure is so desirable that there seems to be but one practical general arrangement of the different cottages,—that is, that they should all have the same exposure, with the administration cottage in the centre. (Plate III., Fig. 14.) The most serious drawback I see to this is the difficulty of securing an economical and effective system of ventilation of closets. We must depend chiefly upon a natural ventilation, unless we go to the expense of connecting all the cottages with an underground duct led to a flue where artificial vent is produced.

Each set of fixtures may be ventilated by the introduction of a gas-jet at the base of a flue, but it is an imperfect form of ventilation.

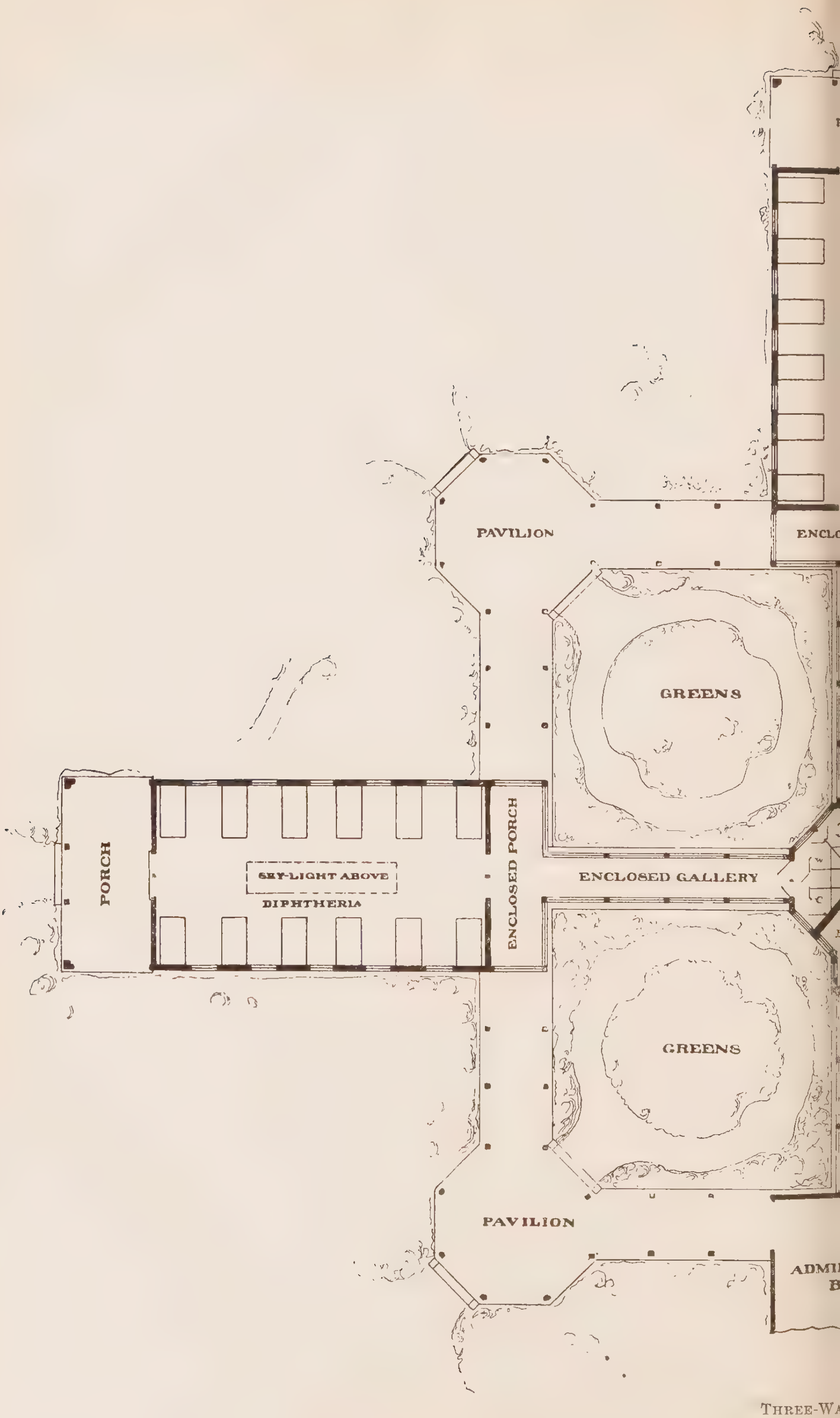
When the cottages are located around a common centre (Plate IV., Fig. 15), the closets may conveniently occupy the centre space, where a flue may be run up, carrying both the foul air and the smoke from boiler or kitchen building, which would occupy one axe. Either plan, however, will admit of a picturesque combination.

PLATE III.

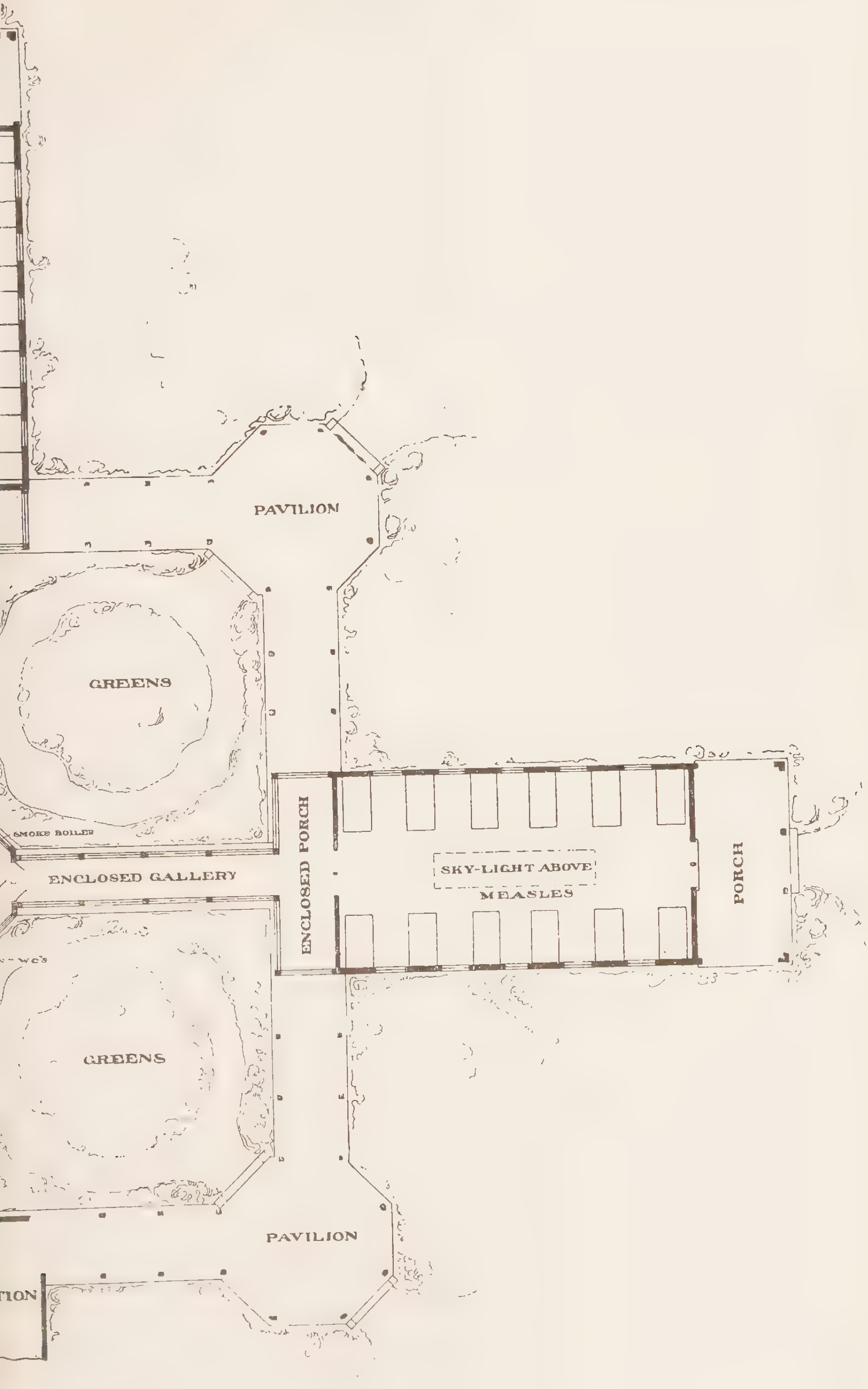
Fig. 14.



Plan: Country Hospital.


ENCLO
PAVILION
GREENS
PORCH
SKY-LIGHT ABOVE
DIPHTHERIA
ENCLOSED PORCH
ENCLOSED GALLERY
GREENS
PAVILION
ADMI
B

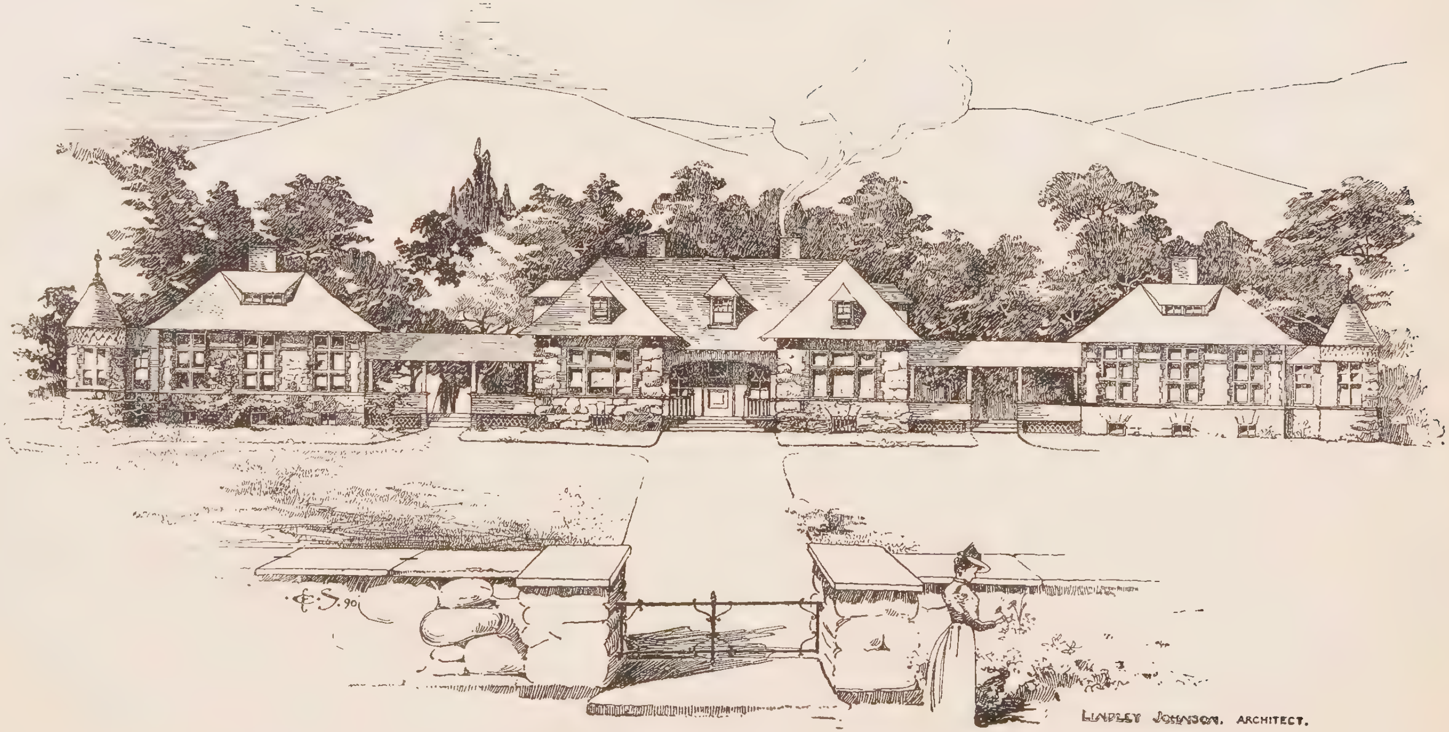
THREE-WA


PAVILION
GREENS
SMOKE BOILER
ENCLOSED GALLERY
ENCLOSED PORCH
SKY-LIGHT ABOVE
MEASLES
PORCH
wc's
GREENS
PAVILION
TION

TAL.

PLATE V.

FIG. 16.



SKETCH: SMALL COUNTRY HOSPITAL.

Where it is intended to provide separately for the treatment of different diseases, such as measles, diphtheria, scarlet fever, etc. (Plate V., Fig. 16), the construction of cottages around a common centre has much to recommend it. It insures a better separation, with an economy of service. In this case, however, I believe the water-closets, etc., are best located at the ends of each cottage, and the plat of ground lying between the cottages should be crossed with connecting, covered passage-ways that may be enclosed in winter.

The first floor of such cottages built without a cellar below must be kept up three feet above the ground, and extra precautions are required to ventilate this space and secure a dry floor. The walls should be back-plastered. The exterior face of walls may be boarded, shingled, or pebble-dashed, with cross-timbers showing.

The roofs are best covered with shingles. A cedar shingle exposed to the salt air will soon obtain a silvery effect which is very good.

The whole effect may be treated economically, and the result be creditable to the designer and an attraction to the neighborhood.

JUVENILE CRIME, AND PUBLIC METHODS OF PREVENTION AND RECLAMATION.

By J. PERCY KEATING, Esq.

The capacity to commit crime presupposes the consciousness of moral obligation. This sense, which we call conscience, is a primitive conception and prior to all reasoning and experience. The occasion of its exercise or application is man's conduct; and by conduct is meant not the blind obedience to organic propensity, but a course of action viewed through the power of abstraction as a means to an end. The capability of such application forms the measure of responsibility, and to such responsibility therefore are requisite first the concept itself, then the intellectual capacity which brings the particular act to the test of conscience, and lastly the perfect equipoise of the volitional faculty which permits of entire freedom of choice.

Now, the intellectual faculty, or power of forming abstract ideas, is of gradual development, dependent upon the organism and in particular its highest development the nervous system, which, possessing in the beginning a certain latent capacity, gradually develops in conformity with such capacity, assisted and directed by habitual exercise occasioned by the experience derived from the senses. It becomes, therefore, a question of importance in the domain of criminal jurisprudence to ascertain at what time of life the development has reached such a stage as to raise the presumption of criminal capacity.

The Romans, acting upon the ancient belief of philosophers that the human organism undergoes a complete change every seven years, marked the completion of the first of such periods as the age of discretion. Justinian fixed the age of puberty at fourteen years in males and twelve years in females; and in the matter of responsibility those who were nearer infancy than puberty, or below ten and a half in males and nine and a half in females, were deemed incapable of committing crime. With those who were nearer puberty than infancy there was a presumption of incapacity, subject to be rebutted by contrary proof, according to the maxim *malitia supplet ætatem;* and even where the presumption was overcome, a lighter punishment was inflicted than upon adults. The Saxons established twelve years as the age of possible discretion, and from this to fourteen responsibility depended upon natural capacity. By the common law of Germany fourteen is the age of discretion. By the Austrian code responsibility begins at ten, and an offence committed by one under fourteen is punished like an infraction of a police regulation. The age of responsibility begins in Spain at nine, but punishment is mitigated below the age of eighteen.[1]

The common law of England, as administered there and in the United States (except in such States as have altered the rule by statute), following the civil or Roman law, regards seven years as the age of discretion, below which the child is legally incapable of committing crime. Between seven and fourteen the presumption is in favor of innocence, which may be overcome by proof of capacity to understand the nature of the act. This capacity, as Blackstone says, "is not so much measured by years as by the strength of the delinquent's understanding, and is to be determined by the jury under the evidence in view of all the circumstances;" and it has been held in Massachusetts and South Carolina that if the capacity be shown by, or even inferred from, the circumstances, it is not necessary to show actual knowledge by the child of the unlawfulness of the act, outside the facts of the offence itself, for in such case it may be presumed. The exact criterion of legal capacity, however, has been of late years questioned by text-writers,[2] with the result, as it would seem, of affecting the general trend of modern decisions in favor of the child. For, while the conclusion to be deduced from the earlier cases establishes as the test the mere capability, as Lord Hale puts it, of discerning between good and evil, the later tendency would seem to be to require a knowledge or capacity to understand the act as a thing forbidden by the law under a penalty, and the ability of the child to regulate his conduct accordingly. As indicative of such a change it may be noted that the law of Texas, as interpreted by the courts of that State, requires proof of knowledge by the child of his legal responsibility, and the same is the rule in Kentucky and Alabama independent of statute; though, indeed, it has been held in Texas that the discernment required by the statute may be gathered from the circumstances of education, habits of life, general character, and oftentimes the circumstances connected with the offence charged.

The nature of the evidence necessary to overcome the presumption of innocence is likewise the subject of criticism. Thus, to the cases cited by Blackstone of convictions of children of ten, nine, and even eight years of age, for murder, the objection has been raised that the processes of the infant mind were judged by subsequent acts indicating a realization of the criminal nature of the act after the consequences were perceived, though such acts afford no logical inference that the child understood the nature of the crime at the moment of committing it. Despite these considerations, however, there is no reason why, even at this day, where the common law is in vogue and the presumption is overcome, a child of seven years of age should not be convicted of a capital offence and executed accordingly, unless, indeed, public opinion should in the particular case interfere to mitigate the rigor of a law that has outlived the age which gave it birth. It is believed that the youngest person ever executed for crime was a boy named Dean, between eight and nine years of age, who in 1629 was found guilty of burning two barns at Windsor.[1]

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[1] See 1 Green's Criminal Law Reports.

[2] See Austin's Jurisprudence, section 26.

Where the common law has been altered by statute in the different States of the Union, the age of discretion varies. Thus, in Illinois it is ten years; in New York, under the Penal Code of 1884, it is twelve; in Texas it is nine, and the presumption expires at thirteen.

With respect to the proof necessary to overcome the presumption of innocence, it is an old axiom that it varies in intensity with the tenderness of years; but this must be taken with the qualification that, as the lesser offences which usually come within the clause of *mala prohibita* do not so violently shock the moral sense and are not therefore so readily recognized as a violation of duty as are the higher offences, the proof of guilty knowledge should be well supported.

The presumption of innocence at common law between the ages of seven and fourteen was originally adopted *in favorem vitæ* in case of capital crimes only, but gradually became applicable to all felonies and misdemeanors for which an infant was punished. But it may be here noted that, while the presumption might be overcome, it did not always follow that the same punishment applied to infants as to adults; and herein the wisdom of our day is apparent in the distinction between the treatment of children and that of adults for crimes to which we shall presently have occasion to refer. If infancy is set up as a defence, it must be proved by competent evidence. Ordinarily the experience of medical experts is admissible to prove age, but it must be accompanied by facts on which the opinion is based, and it may be generally inferred from the circumstances.

As to a child's confession of his own guilt, while the utmost caution and circumspection are imposed in admitting it, it has none the less been held that he may be convicted on it. "The capacity to commit crime," says a learned English judge, "necessarily supposes the capacity to confess it." Therefore, in New Jersey in 1828 a boy of twelve years was convicted of murder on his own confession and was executed. It has been held, however, in the same State, that the mere naked confession unsupported by other evidence is not sufficient. Confessions, of course, may be excluded on the ground of improper inducement, such as threats, etc.; but an admonition or a warning need not be so construed. The command of a parent will not justify the criminal act of a child done in pursuance of it, unless discretion is wanting, or the child acted under restraint.

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[1] See Law Reports (N. S.), 5.

In the case of rape the common law presumes the infant under fourteen incapable of committing it. This presumption is founded on impotence rather than on want of discretion, and evidence is not admissible to rebut it. And so he cannot be convicted of an assault with intent to commit rape, though, if he aid in the commission of the offence and exhibit an intelligent evil purpose, he may be convicted as principal in the second degree. The presumption of impotence was established *in favorem vitæ*, rape being in early times, as it is now in many of our States, punishable with death. And therefore in Massachusetts, even though the presumption of impotence still exists, it is held that if there be proof of puberty the offender may be convicted of an assault with intent to commit rape, on the ground that the presumption should not be applied to an inferior offence the punishment of which is not death. But failing proof of puberty in such case, the offender can be convicted only of assault and battery.

In some of the United States, such as Ohio and New York, the rule of the common law that the presumption of impotence cannot be rebutted under fourteen has been repudiated, for the reason that the general conditions upon which it is based do not exist in this country, owing to the different circumstances of climate, mixture of races, and habits of life. And in North Carolina it has been held that even though the common-law rule obtains, and the offender cannot therefore be punished capitally, nevertheless where the intent is manifest he is subject to indictment and should be made an example of by the utmost punishment which the law permits.

As the law establishes the age below which the male is presumed to be incapable of rape, so it fixes the period below which the female is presumed to be incapable of consent. At common law the age of consent is twelve years. In England it is further provided by statute that between twelve and thirteen the consent of the female only reduces the man's crime from felony to misdemeanor. In our States the age varies. Thus, in Pennsylvania it is sixteen, but if the jury find the girl under that age consented, and is not a woman of good repute, the offence is fornication only, and the accused is acquitted of felonious rape.

But while the fact of the female being under the established age of consent fixes the crime in the adult, it does not, of course, overcome the presumption of impotence or want of discretion, as the case may be, if the offender be under the age of fourteen; and, therefore, in an English case where a girl of nine innocently consented to commerce with some boys, the court refused to sustain a conviction for assault, the presumption not being overcome. It is also held that the patient may be convicted of an unnatural crime though the agent be under fourteen.

An infant may appear in a criminal prosecution and defend himself in person or by attorney, and it is error to assign him a guardian as is done in civil cases. If an infant under seven is given in custody on charge of felony, an action for false imprisonment will lie; and where a minor is imprisoned under an illegal sentence the proper remedy is by habeas corpus.

PUBLIC METHODS OF PREVENTION AND RECLAMATION.

The treatment of the delinquent and dependent classes in general, with a view to the prevention of crime according to scientific principles, involves the question how far altered circumstances and conditions may influence character and conduct. This question, carried to its ultimate issue, involves the old controversy between determinism and free will; and, while such consideration is beyond the scope of our present inquiry, a brief reference to the attitude of scientific thought on the subject may not be out of place. Let it be understood, however, that, in discussing theories, any apparent inconsistency with revealed religion can only argue a defect in the theory, for it is the writer's humble belief that science is but the handmaid of religion, and that its final and culminating conclusions will be the vindication of revelation by the processes of human thought, so far, indeed, as the finite mind is permitted to contemplate eternal truth.

Natural science, having no means of ascertaining or explaining the essences of things, deals with phenomena alone. Within such limits mind is viewed as a force or principle of activity liberated in the exercise of the functions of the organism, and, as such, conditioned upon the proper adjustment of such organism. Now, this organism in its origin and development is subject to the universal physical laws of cause and effect, and herein the speculations of the evolutionist have traced, within a comparatively recent period, the general outlines of a most wondrous design. This theory no longer regards the human organism and its function, character, as the chance offspring of immediate circumstances, but as the product of an evolution controlled by a long line of circumstances extending from the present moment back through ascending generations to the very origin of the race; effecting modifications all along the line, which, becoming organic by the mysterious principle of differentiation and integration, are transmitted by descent. In other words, the law of evolution or reproduction, while preserving the type, is dependent on the environment for effecting, and on the heredity for transmitting, the modifications by which the species are distinguished.

Carried to its extreme, this theory regards every act, whether of the physical, the mental, or the moral order, as the inevitable consequence of such factors. Morality in its ordinary acceptance is a myth, crime being either the product of disease or the result of lack of development of the particular nerve-centre, since, as Maudsley says, man cannot evade the tyranny of his organization. Under such a view, will is but the aggregate of feelings and ideas at the time existing and predetermined by experiences, and the moral sense is converted into the altruistic tendency, a necessary outcome of the social state and condition of man's existence, in the absence of which the race would perish.

In the light of the more recent scientific theories, however, such conclusions would seem to be not altogether satisfactory. Beneath the physical organism and the forces which are liberated in the exercise of its functions, the distribution of which is from moment to moment caused by such predetermined organism and environing conditions, certain philosophers recognize as manifest an independent germ of personality or proper spontaneity which transcends the power of natural science to explain.[1] The Duke of Argyll, in his "Reign of Law," while claiming that man's will is subject to law in the sense that it must act only on motives, admits that among the motives presented he has a selecting power. "He can, as it were, stand out from among them,—look down from above them,—compare them among each other and bring them to the test of conscience" (p. 306). This freedom from compulsion it is, perhaps, which leads Prof. Fiske to assert[2] that the present scientific state of knowledge warrants a return to the theory of Plato, which views the soul as a spiritual substance incarnated in perishable forms of matter. Herbert Spencer, while adopting the deterministic view, recognizes, nevertheless, an unexplainable independent entity, when he says, "The aggregate of feelings and ideas constituting the mental I, have not in themselves the principle of cohesion holding them together as a whole, but the I which continuously survives as the subject of these changing states is that portion of the unknowable power which is statically conditioned in special nervous structures pervaded by a dynamically conditioned portion of the unknowable power called energy."[3] And Prof. Stokes, of the Royal Society, if we are to accept the current report of a lecture recently delivered by him, regards the process of thought and of life generally as the result of interaction between an individualized fundamental energy and the organism. This view would seem to accord with that of Mr. Spencer, though, of course, once such fundamental energy is admitted, there is no knowing how far it controls the development of the organization in its every aspect.

For the purposes of our inquiry, however, every school of thought would seem to occupy a common ground in admitting in the normal individual, first, the primitive conception we call conscience, however derived, and the presence of which is the basis of responsibility; and, second, the necessity of a strict regulation of conduct in obedience to its proper dictates as affecting either the individual himself or the society of which he forms part. But this conscience so recognized is reducible to one or two truths of a most general character, and, while these truths lie at the very root of all action and conduct, they form indeed but a small part of our moral states. Grafted upon them, as the result of experience and the means to the end which they point out, is the variable element into which enter all our ideas, judgments, habits, recollections, passions, sentiments, and prejudices; and, as this is the result of environment, so it may be controlled and directed. Herein, then, lies the value of education, that it offers an environment which will assist the healthy development of every order of man's nature,—the physical, for the purpose of establishing that equilibrium of physical force, centring in the nervous system, which promotes not only correct processes of thought, but also the fullest exercise of the inhibitory faculties commonly called strength of will; and the mental and moral, with a view to the free use and development of the power of right reason properly directed, especially in its application to conduct.

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[1] Ribot's Heredity, p. 342.

[2] Destiny of Man, p. 42.

[3] Principles of Psychology, p. 504.

These considerations as applied to the treatment of crime have contributed in revolutionizing the methods in vogue only a century since. For, while formerly the sole mode of repressing crime was by punishing the offender, our present system goes to the root of the evil by anticipating and preventing it. As applied to the young, such system lays claim to peculiar consideration, not only because the child is helpless and in no wise accountable (under any view) for the conditions and circumstances surrounding him, but because, not having attained the full growth to which his organism is susceptible, his development is the more subject to environing influences. As a necessary corollary to such conclusions, the State, in whom as custodian of public order the power to repress crime is lodged, has acquired a clearer knowledge of its own obligations in this respect, as extending even to the endowment of all its members with such qualifications as are commensurate with their civil duties, especially where those upon whom the same obligation primarily rests, through force of circumstances or neglect, fail to discharge it; and this, too, without in any manner trenching on the duties of the individual or the spirit of private benevolence upon which all work springing from the social relation must necessarily depend.

The subject, therefore, cannot be concluded even upon this cursory view without a passing reference to the reformatory and preventive measures of the period, though, amid the innumerable features which characterize public interest in the subject, we can do no more than glance at such as apply to the public institutions, considered apart from the legislation touching the condition and treatment of minors in private life.

And first in the order of importance we may note the separation of delinquent and dependent children from adult criminals by removing them from the contaminating influence of prisons and poor-houses, a change which at this day seems so obviously necessary that one cannot but wonder that it should have been of such recent origin. This step was followed by the separation of delinquent from dependent children, for the sake not only of preventing the moral contamination of the innocent by personal contact with the criminal, but also of preserving that self-respect which is apt to be obscured by the absence of any distinction between misfortune and crime. Reformatory work is likewise occasionally accompanied or preceded by certain correctional features intended to impress upon the child the seriousness of his offence. Hence the origin of State industrial schools, as distinguished from reformatories. But it must be here observed that below a certain age the offence partakes so little of the nature of crime, by reason of the immaturity of years, that no such distinction is applicable, and the industrial school is therefore a proper place for the culprit; while, on the other hand, vicious and incorrigible children, though unconvicted of any particular offence, may with better results be classed among subjects of reformatory work. And, inasmuch as the commission of the overt act is ordinarily only the natural result of evil courses, the true criterion to be followed in marking the separation would seem to be not so much the offence as the character of the individual.

The adoption of the indeterminate sentence, too, was a foregone conclusion when the real object of the treatment of juveniles came to be fairly understood. That a child should have the advantage of superior surroundings and opportunities for self-improvement only for such term as might be deemed commensurate with the seriousness of his offence is irrational. Hence in such States as have inaugurated a thorough system the term of control by the State authorities is generally extended to such period as offers some guarantee of future good conduct. In the matter of mere preventive work this period may extend from the earliest age after weaning until the attainment of capacity to earn a living,—usually about sixteen, though it may last till twenty-one. With respect to reformatory or correctional work, if any hard-and-fast line is at all practicable, the age of twelve is perhaps as low as can be fixed upon, having regard to the distinction. On the other hand, the age of sixteen has been thought to be the limit beyond which the influences of reformatory work so greatly decrease, and the liability of the culprit to exert a baneful influence on the younger inmates so increases, that in certain communities—England, for example—no youth is admitted to a reformatory above that age, though, of course, such limit of admission does not affect the period of his stay if admitted prior to that age. It has been suggested in this country that there is great need for a reformatory for juvenile offenders between sixteen and twenty-one, where a severer discipline would serve to deter and punish repeated offences; and the suggestion is certainly important when we consider the chances of reform even at such age which might be thrown away by contact with hardened criminals. The Reformatory at Huntingdon, Pennsylvania, supported by the State, is intended to supply this want. The control by the State, however, which attaches to minors under such conditions is not confined to institution life,—a point to which we shall presently advert.

The custody of the child by the State involves, of course, the forfeiture of the parent's right to the same. This point has been fully tested in the courts of this country, wherein the objection was formerly raised that to confine a child without trial and conviction is opposed to the Declaration of Rights. It was adjudged, however, that such right undoubtedly exists, on the ground that the period of infancy is a period of custody, and, where the parent fails in his custody to secure the welfare of the child, the State is in duty bound, in the same interest as well as for the protection of society, to substitute it.

But, while such right of the parent is forfeited, his obligations should not be thereby discharged, lest, indeed, he should profit by the State's assistance in ridding himself of the care of his offspring, and lose sight of his due share of responsibility for his offspring's misfortune or evil-doing. This principle is of the utmost importance, and indeed its strict enforcement may be regarded as one of the essential features of work of this character. The subject, it must be confessed, has not as yet received its due share of attention. In the United States it is for the most part overlooked. In England the parent is obliged under penalties to contribute to the support of the child while in the institution, or to undergo a certain amount of work or even punishment if the circumstances demand. The prescribed amount is five per cent. of the burden; but the returns show so small an average of contribution as to warrant the complaint that the law has not served the purpose in view, or that the parent's responsibility has not been sufficiently brought home to him.

Another great step which characterizes the methods of our day is the recognition of the value of private zeal as an aid and stimulus to the effective accomplishment of public work of this character, substituting as it does a pure motive and unflagging interest for the routine superintendence of paid officials. And this fact naturally leads to the question whether the State cannot do its part better, so far as institutions are concerned, by assisting institutions of private origin, than by establishing institutions of its own. It cannot, indeed, be denied that the latter, being necessarily of a non-sectarian character, must give less prominence than its importance demands to the religious feature of such work, which is so essential to its success. The objection has been advanced against such private institutions in this country that where paid *per capita* they are tempted, for the sake of the State contribution, to retain the child within the institution for a longer period than his interest and that of the State will warrant; and the sectarian institutions of New York are instanced, where the number of inmates has increased to an enormous extent. But, if the increase may be attributed to such causes, the defect would seem to lie in the absence of such State supervision as would secure the discharge or placing out of the child at as early a day as possible. The system works well in England, where the industrial and reformatory schools are for the most part of voluntary origin, and in many cases under sectarian rule, but where the most rigid system of State supervision and control acts as a safeguard against any such contingencies. The Royal Commission appointed in 1882 to examine into and report upon the system there in vogue strongly deprecates any effort to prevent religious sisterhoods and brotherhoods from carrying on such schools on an equality as regards government aid with the machinery of a voluntary committee and paid superintendence; and Mr. Tallack, in his excellent work "Penological and Preventive Principles" (London, 1889), remarks that "it may justly be deemed matter for grave reflection and surprise that comparatively so little prominence has been given to such systems of popular education as involve the training of children, both to self-supporting industry and amid those harmonious influences of piety which are fostered mainly by denominational schools, where the pupils are surrounded by arrangements and persons adapted simultaneously to guard their liberty of conscience and to prevent their faith being needlessly assailed by jarring criticisms or by infidelity or immoral contamination."

Whatever differences of opinion exist, however, as to denominational agencies, it cannot be denied that the management of public establishments of the kind by mere *elective* bodies is the least efficient, not only because of their usual lack of the proper spirit animating such work, but also because of the possible interference of political considerations in their selection, thereby involving constant change of persons and of policy.

In England after eighteen months' residence the child may, with his consent, be placed out on license with trustworthy persons, such license to be revocable by the managers in their discretion; and this leads to the consideration of one of the most important features of prevailing methods. The family group is the basis and the mainspring of man's affections and emotions. Hence the artificial environment whereby it is hoped to form character should imitate or provide the family life as far as practicable. Such conclusion has led to the adoption of what is known as the "cottage system" in institutional life, whereby the inmates are broken up into groups resembling as nearly as possible the family, and, what is even more important, to the "placing-out system," so called, whereby institution life is substituted as soon as may be by actual family life, accompanied, of course, by such continuing supervision as is made necessary in the absence of those parental ties which strangers can seldom supply.

It would occupy more than the allotted space to dwell on the special characteristics of each system. Suffice it to say that the "cottage system," as compared with the congregate systems in institutions, tends better to preserve the individuality of the child and permit of personal supervision over each one. Its possibilities may be illustrated in the words of Dugdale ("The Jukes") when he says, "It enables the managers, by careful selection of temperaments and dispositions which shall healthfully react on each other, to segregate those who suffer from the same deficiencies, so that the defects of one shall not become a demoralizing example to the rest, and to group such natures as present well-organized habits so as to become exemplars to those who lack those special habits, thus to consciously organize by artificial means an environment in which the subjects themselves will become instruments for each other's regeneration."

With regard to the "placing-out system," it must be observed that it does not wholly do away with institution life, the latter still holding its place in serving as a temporary shelter until homes can be provided, and also as a place of preparatory training under such discipline as may pave the way for the reception of the child in the well-ordered family to which he may be introduced. There are cases also where, by reason of unusually vicious temperament, the necessity for punishment presents itself in order to reduce the culprit to a sense of his crime, and where, therefore, it would be unsafe and injudicious to introduce him to the freedom of family life. But with these exceptions institution life is to be deprecated, in that it affords a false environment and fosters dependency, which is the reverse of what is intended in such work. As to the "placing-out system," the best method is that which provides for no compensation on either side, or, in other words, a system of adoption, as calling into play the affections upon which so much depends. Apprenticeship has been objected to, in that the children are not so prepared by previous discipline and education as to insure their contentment, and that the money-getting spirit may induce the persons to whom they are apprenticed to neglect their religious and secular education and even their comfort.

We have said, however, that State supervision is necessary to the success of such system, and in this respect the method in vogue in some of our States, notably Michigan, Massachusetts, and Rhode Island, is especially to be commended in many particulars as thorough and effective. While differing in detail, the general plan adopted in these States is much the same, and as an example we may cite the general points of the Michigan system. They include a State Board appointed by the Executive of the State, acting without compensation, having its proportionate representation of women, with a view to the interests of the female wards of the State. They visit the institutions and report to the Executive. There is besides a Board of Control of the public institutions, and also a State Agent in each county, whose province it is to seek suitable persons willing to adopt, and to keep acquainted with the condition and history of every child that has ever been taken under public charge. Where the child is accused of an offence, the County Agent (in Massachusetts, the State Visiting Agency) investigates the circumstances and advises the court, who thereupon either discharges the delinquent, or binds him out, or commits him to the care of a person desiring to adopt him, or commits him to the reformatory, as his discretion points out. In Massachusetts the child may still continue at home, but under the oversight of the State Agent, who undertakes, however, to bring him up again when needful, meanwhile to watch over him. This is what is known as the sentence of probation. The Agent periodically visits the child where bound out or adopted, and reports to the Board. The officers of the institution may also bind out or place a child for adoption, after first notifying the Agent, who files his report as to its advisability. In the case of dependent children between the ages of two and twelve years, they are placed in the State Public School, upon the order of the judge of probate, accompanied by a medical certificate as to health. The school is conducted on the family plan, with about thirty children in each cottage and a lady superintendent. Thence, after a few months' preparatory training, they are placed out in homes or on written contracts approved by the County Agent;

and, in addition to the inspection of the County Agent, the special travelling Agent of the school supervises their treatment. The contract provides for their treatment as members of the family and their education in the public schools, and for the payment of a specific sum for the child's benefit at majority. Several other States have adopted systems more or less similar to that above described.

A few words remain to be said on the system of education pursued in institutions of a reformatory and preventive character. This may be divided into the religious, the secular, and the mechanical.

The great importance of the first few will deny. De Tocqueville, the colaborer of De Metz in the establishment of the great Reformatory of Mettray in France, says no human power is comparable to religion in reforming criminals. We have already adverted to the question whether it cannot be better attended to in denominational institutions of private origin than by the State itself.

Nor will it be denied that the second is likewise essential to the formation of the intelligent citizen. I therefore, even where the training is in the line of agriculture, which, by reason of its being the calling of the vast majority, naturally occupies a large share of the education in all schools of this character, a certain time should be devoted to the elementary branches of an academic course.

With regard to the teaching of trades, some have questioned whether it be the duty of the State so to provide; but the day wherein such objections could prevail may be said to have passed away. As an educator, manual training, embodying the teaching of trades and technological and industrial instruction, is invaluable. "Physiologists," says Commissioner Dawson, in the general Report of the United States Bureau of Education of 1887, "have long been telling us that muscular exercise invigorates the brain; in addition to this important result which the exercise of the hand shares with all other bodily exercise, the advocates of manual training have urged its effect in quickening observation, in increasing the range and acuteness of the perceptive faculties, and in establishing an intimate familiarity between the mind and things." And it may be added that it promotes the habit of steady attention upon certain fixed lines of work, which, while exciting the child's interest, serves as a guard and restraint upon his conduct; and, not the least result, it affords an opening for a successful career, which is a great stimulus to good conduct. In this country, with the exception of Massachusetts, there are no laws requiring industrial and technical training, though in the majority of reformatory and preventive institutions trades are taught. A difficulty has been encountered in the opposition of trades-unions to the sale of articles manufactured in such institutions, which in some States has culminated in laws either prohibiting such sale, or checking it by compelling the branding of the articles so manufactured. To this extent the subject has assumed an economic aspect which must be determined in the end, like all class legislation, by the power, importance, and claim to consideration of the class sought to be benefited as compared with the rest of the community.

As to the character of the particular handicraft, a selection should be made of such as afford employment for large numbers in which there is a great and constant demand for skilled workmen, and in the selection of these the natural bent of the individual should be followed, as far as possible, while the sole object should, of course, be the benefit of the individual, to the exclusion of any consideration of profit to the institution itself. Hand-labor, too, is greatly preferred to machine-work. The contract system of labor in reformatories is almost universally condemned, both here and abroad. Girls should be taught the domestic arts, such as sewing, cooking, and the like, though with the gradual enlargement of woman's sphere of usefulness in business circles the range of occupations will naturally be extended from time to time.

In the matter of discipline, the infliction of corporal punishment in extreme cases is still permissible, but under such safeguards as render its abuse improbable, if not impossible. In the case of delinquents, a short term of solitary confinement has been found beneficial for the purpose of bringing them to a proper sense of remorse, and is in vogue in England. The culprit there may even be whipped, as an alternative, but not by a subordinate officer, and, as a safeguard against undue haste, passion, or mistake, a night must intervene between the offence and the punishment. The mode of emulation adopted in this country by the system of marks and credits for conduct, studies, and industry, as well as the system of shortening the term of detention by reason of good conduct, happily dispenses with corporal punishment to a large extent. In France the offender before being released is informed of the punishment which will be inflicted in case of a relapse.

In close connection with the question of discipline the introduction of female influence has been of great avail. The gentle ways of a woman often have more weight than the cold counsels of a man, and are especially beneficial in extending sympathy to the girls, arousing the sense of honor in the boys, and generally in creating and encouraging those finer instincts which contribute to elevate man's nature.

As the physical organization is so important a factor in all work of the kind, its development claims a large share of attention; therefore a due proportion of out-door exercise, proper distribution of hours of work and play, healthy diversions for the purpose of cultivating and encouraging the animal spirits, a careful supervision of the sleeping- and living-apartments with a view to proper drainage and ventilation, and the prevention of disease and the promotion of general health, all contribute to the success of institutions of this character, and likewise enter into the selection of proper homes for such as are recommitted to the influence of family life.

MEDICO-LEGAL TESTIMONY.

By JEROME WALKER, M.D.

Probably no class of medico-legal cases is of deeper concern to the physician, be he expert or general practitioner, than that in which the plaintiffs are children, and the complaints those of physical injury, rape or attempt at rape. Such cases at times are of extreme importance, because of the severe penalties that can be and often are inflicted on the supposed guilty ones, and because of the weight that is frequently attached to the physician's testimony and the consequent responsibility that rests upon him. To be largely instrumental in sending a human being to prison for ten, fifteen, or twenty years is a grave responsibility, and the more so when perhaps the result is due to a careless but confident physical examination, a misinterpretation of signs and symptoms, and an inexperience in the study of human nature. In addition, the physician deals with children who may not have clear ideas as to the difference between right and wrong, are keenly alive to real and fancied injuries, and are readily influenced by those of stronger mind, or for whom they have an attachment, to testify even against friends and kindred.

There are also those feelings of sympathy and pity for children which are inherent in most of us, and which will tend, unless we are very careful, to cloud our judgment. And this danger is imminent whether the children are plaintiffs or defendants.

To assist physicians who have had little or no experience in medico-legal cases such as have been referred to, is the object of this article.

Naturally, the subject of how to deal with children in such cases, and how to testify so that we can as physicians tell "the truth, the whole truth, and nothing but the truth," is to be considered under three headings: 1st. How to examine children orally. 2d. How to examine children physically. 3d. Medical testimony.

HOW TO EXAMINE CHILDREN ORALLY.

Mr. Wilkins, superintendent of the Brooklyn Society for the Prevention of Cruelty to Children, who has had a large experience in his line of work, states that "young children of four, five, or six years of age, or thereabouts, do not, as a rule, seem to have clear ideas as to right and wrong, and, if any one gains their confidence, such person can do almost anything with them." Older children, especially girls from twelve to fifteen or sixteen years of age, are more suspicious. They do not readily impart information or answer questions if they think that by so doing they criminate themselves or delay the execution of cherished plans.

To gain the confidence of all the children we deal with is of prime importance. But this confidence is to be received cautiously, for, eager to do what they can for us, they may too readily agree or disagree with lines of inquiry as their impulses tell them they can best please us. Unless we are careful, they will lead us astray: we arrive at conclusions which will not bear cross-examination, in court, by the opposite side, and the children imperceptibly drift into erroneous statements. To gain the confidence of a child, be first the child's friend, then you can assume the position of doctor and examiner. Among the lower classes threats and corporal punishments are common, and little children are often threatened with the visits of the doctor as a bugaboo. To appear to such children in the *rôle* of a doctor is therefore to inspire fear; and yet you must win their confidence. To obtain it, it may be necessary to see them on several different occasions. And though a stick of candy or something to eat may suggest itself to you to open the way, for a number of reasons, interesting the child in a doll, in a picture-book, or in its recital of events or description of things dear to its heart, will prove a better way. Some physicians, I am sure, never obtain a reliable history of a case from a child, because they are too dignified or in too great a hurry, or attach too little importance to becoming the child's friend.

To rely entirely or for the most part upon the testimony of deeply-interested, prejudiced, or angry friends of the child is certainly unwise. In fact, it is best to question the child and its friends apart from each other, as is done in the grand-jury room. The mere presence of a guardian will sometimes cause a young child unwittingly to prevaricate. A frown, a nod, a shake of the head or a finger, from such a source, will have much weight in fashioning the child's testimony. The statements of parents, especially, in regard to their children are to be cautiously received. As has been said, "parents can seldom be brought to see the corrupt morals of their own children; therefore few corrupt children are either admonished or punished by parents."

As children approach the period of puberty, boys frequently resort to practices which they consider manly or necessary, such as smoking, self-abuse, liquor-drinking, keeping late hours, overeating at restaurants, etc. Girls frequently become romancers, are carried away by a manly form, a pleasing face, and pleasant manners, and so are readily influenced by flattery and attentions. They are easily offended, imagine that they are slighted or abused, and are led into making insinuations or accusations for the sake of notoriety or to "get even" with some one. They acquire a knowledge, or rather an idea, of what intercourse, conception, and child-bearing are, and may, as boys do, acquire immoral habits. From talks with educators in public and private schools, with physicians and officers of societies having the care of children, and from my own experience in schools and charitable institutions, I must conclude that some children, carefully watched over in public and private schools or in families, may and do grow up differently from the boys and girls just mentioned. But the fact remains that there is an astonishingly large number of children, especially in our cities, who know or surmise more about sexual relations than people at large suppose possible. The examining physician must take all these facts into account, as he tries to unravel the tangled history of a case and to ascertain the motives for certain statements,—blackmailing schemes or injuries. While it is true that in court the physician testifies in the main to what he has seen, his opinions are much sought after, and he can give a reliable opinion only after a careful oral as well as physical examination.

In questioning children much tact is necessary. Pointed questions with many children frequently produce answers not at all appropriate or desired. If, for example, it is asked, "Did so and so hurt you? did he throw you down? did he call you such or such a name?" the child may answer "Yes" or "No" when the opposite would be the truthful answer. Or if the child is asked, "Do you have pain here?" touching some particular part of the body, the answer may be "Yes" when the pain, if any, is elsewhere.

In our experience, it is best to let the child give its own story in detail, if inclined to be communicative, and even to follow up side issues touched upon. If not so inclined, the child may be encouraged to tell its story in its own way. Such a story, so told, is effective with a jury. Coaching a child so as to have it tell a straight story upon the witness-stand is a risky proceeding. The very simplicity and sometimes incongruity of expression in a young child's language serve to stamp it as original, and have more than once tended to clear up the uncertain and disputed testimony of others. The child telling its own story will be found to be very positive on a few points, such as the pain caused, the amount of blood shed, the size of a bruised spot. Some of the points the examiner can ascertain for himself.

Much value, of course, is attached to the child's description of sensations experienced. Unless the child has been really injured or has suffered pain, it is likely to use the common remark, "it hurt." But this is not a sufficiently comprehensive answer for a medical examiner, and, if persisted in, is of itself suspicious. A girl of thirteen years accused her father of incest, and repeatedly said that "it hurt her when he did it." On close questioning, she could not say whether he had really touched her or not. In answer to the question, "Did he move his body up and down?" she said, "No, not at all." The physical examination made soon after by a physician detected no abrasion nor any evidence of rape. In fact, there was so much sensitiveness of the parts that a gentle touch of the fingers caused much discomfort. The father was exonerated. If he had really attempted intercourse, the girl would probably have described more graphically the sensation or the pain than by the words "it hurt." At least, this is generally the case. The exceptions are when the children are imbeciles or idiots, or have been drugged, or have hardened the skin and mucous membrane of their privates by self-abuse, or have been accustomed to be used by others for various immoral purposes. On the other hand, sexual intercourse or the handling of the privates, when normally sensitive, may increase the irritability of the parts very rapidly, so that when the physician makes a physical examination they may be found very red and sensitive. Yet the statement holds true in regard to the repeated use of such a general expression as "it hurt me."

Suggestive questions, such as, "Are you not *sure* that all that you have told happened on Monday instead of Tuesday?" "Did he not really say such and such things?" are questions that, if asked at all, should be left till after the child has told its own story in its own way. This form of questioning, the lawyers on the opposite side, in a bland and insinuating way, are apt to use with children to break down their testimony. The medical examiner may, in like manner, do the same. As the child tells its story, note discrepancies and the positive statements, and, generally after the story is told, cautiously ask your pointed and suggestive questions. Make a written memorandum of the salient points of the oral examination and the time at which it was had, for memory is liable to fail and important points to be forgotten. Sometimes such a memorandum is useful to have on the witness-stand. Compare the results of the oral examination with those of the physical one, and upon the conclusions base the testimony.

HOW TO EXAMINE CHILDREN PHYSICALLY.

Probably most children dread a physical examination, in part because of an innate modesty and partly for fear of being hurt; yet some do not seem to mind it, and others appear to desire it. The medical examiner is naturally influenced, in his views as to the tendencies of the child, by the way in which it acts as he proceeds with his examination. But physicans who have examined a number of prostitutes have learned that a certain proportion, on account of an inherent modesty, dread physical examinations. So it is with some children who are conversant with and participants in immoral acts. The modest bearing is therefore not always an indication of good morals. On the other hand, neither is willingness on the part of a child to submit to a physical examination a proof of immodesty or immorality, for there are children who freely talk of their ailments, real or fancied, and who believe that a physician has the right to examine them if he or the parents think such an examination necessary. Such children are precocious, mature early, and have lived the most of their lives among adults who themselves are outspoken even upon private matters.

A physical examination of shrinking, nervous, irritable children is unsatisfactory unless preceded by a careful oral examination. Before examination of the genitals with the fingers, speculum, or other instrument, an inspection should be made of the entire body or of parts of the body, as the case warrants, noting accumulations of dirt, blood, etc., bruised spots, their size, location, whether recent or not, whether associated with the imprint of fingers, a cord, or any other foreign body. Stains upon the skin or clothing, also swellings, congested spots, and abrasions of the skin or mucous membrane, should be examined by the eye and magnifying glass. Stains should also be looked at with the microscope. In the inspection of the genitals the questions arise, are the parts intact? are they in their proper relation to one another? do they appear as if tampered with by thread- or pin-worms, by hands or other instruments? and what are the quantity and general appearance of discharges, if any be present?

An open condition of the vulva, but especially of the vagina, generally indicates repeated handling of the parts, by the child or another, or the introduction of some foreign body. In our experience, in a girl who has not been tampered with, the two sides of the vulva are very nearly approximated even when the legs are separated. An open condition associated with redness, abrasion, and a discharge is very suggestive of rape or an attempt at rape. Yet this condition is sometimes found in cases of severe leucorrhœa or with a gonorrhœa not obtained by sexual intercourse. There is a popular opinion that examiners find upon young children criminally assaulted evidences of severe laceration. Such laceration is the exception. Men generally criminally assault children, I believe, for self-abuse, and so, placing the penis between their legs or the cheeks of the nates, may leave no trace behind but abnormal redness and some excoriation. Fortunately, in some States of the Union an entrance into the genitals, however slight, constitutes rape. For this reason, also, a grave responsibility rests upon the medical examiner as an interpreter of signs and symptoms.

As to the local appearances in cases of suspected rape, Vogel truthfully says, "The funnel-shaped condition and marked tumidity of the external genitals, so urgently insisted upon in works on medical jurisprudence as symptoms of rape having been committed, can only be of value after frequent repetitions of the act, which make the condition well marked. No permanent alteration of form, not even any decided contusion or tumefaction, can ever originate from the simple contact of the glans penis with the hymen."

A physical examination should be made as soon as possible after the supposed injury is inflicted, for the recuperative power of children is generally so rapid that proofs of local injury may disappear even within twenty-four or forty-eight hours after its occurrence. One physician, therefore, examining a child soon after it is injured, may find what another examiner, later on, does not find.

No one adverse physical condition, if found, is sufficient to form a judgment upon.—Spermatazoa upon the genitals of a girl do not of themselves positively prove to an expert medical examiner that rape has been attempted or committed, for his knowledge of depraved human nature is such that he knows that some fiend, man or woman, may have placed semen there to cast suspicion on some particular person. Works on medical jurisprudence relate acts which are equally bad. Not only must the examiner compare the results of his oral examination with those of his physical one, but he must ever bear in mind the injuries that children may do to themselves either for their own gratification or to cast reproach upon others. The records of societies for the prevention of cruelty to children present numbers of such cases.

After the examiner has inspected the parts said to be injured, he should make a careful manual examination, and use with caution a speculum or such other instrument as may be deemed necessary, and at times the microscope for the detection of spermatozoa, etc. If he is not cautious, he may readily produce congestions, if not abrasions. If he cannot show in court that he was cautious, any evidence of injury found by an examiner on the opposite side of the case may be ascribed to the first examiner's rough handling. It is important to note whether the sensitiveness of a part touched is really as great as the child says it is or believes it is. One must discount much of the extravagant language used by many children, as, for example, "It hurts awful," "Oh, it's dreadful!" "Why, it's terrible!" etc., when the truth is that the pain or sensitiveness is slight, and would not be noticed by some children. Calling off the attention of the child to some article in the room, or interesting it in conversation, the physician may meantime touch a supposed sensitive spot and find its real condition. Cocaine is sometimes of great assistance in manual or instrumental examination.

Condition of the Genitals in Girls.—Five signs of virginity are ordinarily given by authorities,—viz.: 1. An intact hymen, crescentic, circular, or vertical in shape. 2. An absence of the carunculæ myrtiformes, the remains of a broken hymen. Not infrequently warts and vegetations are mistaken for them. 3. Entire fourchette, fossa navicularis, and posterior commissure. 4. A narrow and somewhat rugose vagina. 5. Integrity of the perineum. In times past, the greatest value has been attached to the absence, presence, or general condition of the hymen and carunculæ. But it is well known in these days that the presence of a hymen is not proof of virginity, nor its absence a proof that there has been sexual intercourse; for the hymen may be congenitally absent, or have been ruptured by handling, by clots of blood at menstrual epochs, or by the forced introduction of any one of various foreign bodies, or it may be so flexible and tough that intercourse can be effected without rupturing it. Laceration of the nymphæ, fourchette, or perineum can in general be effected only by violent contact. The hymen as an element in diagnosis has been overrated, because it has been supposed that it was fragile and would easily tear when connection was attempted. But the hymen differs in different individuals as to its strength and location. In very young children it is frequently musculo-membranous and depressed into the lumen of the vagina. As the child grows older it is, as a rule, more nearly on a level with the opening of the vagina and is thinner and more strictly membranous. In a record by the writer[1] of twenty-one cases of assault and rape the hymen was plainly perceptible in eleven children. In three instances it was very pliable, in one its free border was thick, and in all the septum was strong. In six cases it was hard to decide whether there was a hymen. In four cases it was absent. Some writers contend that it is never absent,—that it is merely not seen. In this connection the following letter from Dr. A. Jacobi is of value:

"There are good authors who hold that the absence of the hymen is not proven, but I have met with such absence, and agree perfectly with Prof. Hennig, who makes the statement that it occurs.

"But you know that sometimes, indeed often, its shape is by no means the funnel- or teat-like formation usually found in infants. Its development may be quite irregular, and sometimes in cases of vaginal catarrh, which is so very frequent in infants and children from a large number of causes, the hymen appears to exist only as a few prominences, which resemble granulations more than a normal organ. Rape *may* have been committed against an infant *though the hymen be intact.* At that early age the hymen is involved more interiorly than at an advanced age. *But* rape which sufficed to rupture the hymen in an infant must have been committed with so much violence as to result in very serious lacerations of perineum and vagina. (But *laceration of hymen* and *absence of hymen* are two different things altogether.) An absence of hymen, one day after the alleged outrage, with a slight swelling and vaginal catarrh and tumefaction of the labia, does not prove violence at all. It simply proves vaginal catarrh and consecutive swelling. The cases of that kind (that is, without absence of hymen) I have seen by the hundred, and you recall a great many yourself."

As has been already remarked, severe lacerations of the genitals are infrequently seen. Abrasions, congestions, swelling, and discharges are what may in the majority of cases be expected. If an abrasion be present, its location as to the median line of the body will give an idea of what position the assaulter was in when the attempt at connection was made. It is sometimes maintained that a man could not commit rape upon a child; but the fact is that the size of a man does not necessarily determine the size of his instrument, and we not infrequently meet with men of good size who possess small instruments, hardly above the average of those of good-sized boys. "In a medico-legal point of view," says Woodman and Tidy's "Forensic Medicine and Toxicology," "such a defence as the size of the penis is worthless, because, although the disproportion may prevent complete or perfect intercourse, it does not prevent the attempt, which is now justly held to be the essence of crime." According to Wharton and Stillé's "Medical Jurisprudence," while "a full and complete connection of an adult male with a child of twelve years is manifestly impossible, repeated efforts will produce such a dilatation of the parts as to render it finally practicable."

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[1] Archives of Pediatrics, May and June, 1886.

The Presence of Spermatozoa.—As far as I have been able to ascertain, in most cases of rape upon children semen is either not emitted, or, if it is, it is thrown upon the thighs and outside of the vulva, and has been ordinarily washed away before the examination. In the two or three instances where I suspected emission into the vagina, a microscopical examination of the fluid found in the vulva and vagina revealed nothing of moment. Probably a microscopical examination should always be made in cases of suspected rape, for spermatozoa might be found when we should least expect to find them. As to the examination of seminal stains upon cloth, I quote from a paper by Dr. F. M. Hamlin, of Auburn, New York, read before the American Society of Microscopists, at Chicago, in 1883:

"Having occasion, last January, to examine some seminal stains on cloth, I sought to avail myself of the experience of others. I found that all writers on medical jurisprudence and microscopy, including such names as those of Taylor, Beck, Beale, and Frey, to whom I had ready access, adopt and recommend the method of Dr. Koblanck, of Berlin, published in 1853.

"It is, briefly, as follows. Cut out the portion of cloth suspected. Place it in a watch-glass with a few drops of distilled water, let it soak for a few minutes (variously stated from two to ten), stir it about with a glass rod, and then squeeze out the water with the fingers. This squeezing may be done directly upon the slide or into the watch-glass, whence a portion may be taken up by a pipette and transferred to a slide.

"Following this plan with a piece of cloth known to be stained with semen, I obtained such poor results that I resolved to try some other method. Remembering how transparent a fine linen fabric appeared on a certain occasion when I was studying its fibre, I resolved to subject a portion of the cloth itself at once to the microscope. Taking a small piece of the linen and placing it upon a drop of water on a slide, I let it soak for a while, then put on a cover-glass and proceeded to examine it. Almost immediately I discovered a number of spermatozoa clinging to the fibres of the linen or lying in masses in the meshes. Encouraged by this success, I experimented with fabrics other than linen. In light-colored silk the spermatozoa were detected quite as easily as in linen. A firm piece of cotton sheeting proved refractory till I thought to unravel or fray out the ends, when I readily found the zoösperms adherent to the detached fibres.

"Having experimented with the fabrics commonly used for undergarments, I turned my attention to colored woollen goods. These were not, of course, sufficiently transparent to render the above plan practicable: so with a keen scalpel I shaved off a portion of the stained surface, which fell in a fine dust upon the slide. This was moistened, and, after soaking awhile, was examined. The spermatozoa were found even more readily than in the other experiments. . . .

"I therefore recommend the following procedures:

"1. If the stain to be examined is upon any thin cotton, linen, silk, or woollen fabric, cut out a piece about one-eighth inch square, lay it upon a slide previously moistened with a drop of water, and let it soak for half an hour or so, renewing the water from time to time as it evaporates. Then with a pair of needles unravel or fray out the threads at the corners, put on the glass cover, press it down firmly, and submit to the microscope.

"2. If the fabric is of such a thickness or nature that it cannot be examined as above, fold it through the centre of the stain, and with a sharp knife shave off the projecting edge thus made, catching upon a slide moistened with water the particles removed. After soaking a few minutes, —say five to ten,—the powdery mass will sink down through the water and rest upon the slide. The cover-glass may now be put on, and the preparation examined.

"The latter plan serves as well for hairs, but great caution must be observed in cutting them, lest the portions bearing the suspected deposit fly away and are lost.

"Whichever plan be appropriate, it is best first to moisten the slide with a drop of water. In the former case, by laying the cloth upon the water we get rid most easily of the air-bubbles, and in the latter the water preserves the powdery portions cut off from being lost, and they are not rolled to one side, as when the drop of water is subsequently applied.

"Should it be desired to preserve any of these preparations for production and examination in court, I have found that to hold down the cover-glass with a spring-clip, and run around it a circle of liquid marine glue, serves at least a temporary purpose.

"A piece of stained muslin lay nearly two months without protection upon my working-table. I then mounted a portion of it in water, as above described. It now, at the end of five months, shows the spermatozoa as well as ever. For permanent mounting I should suppose the addition of carbolic acid, chloral hydrate, or some such preservative would be of service. I have not found it necessary to use any dye or any solvent except water. A power of three hundred diameters is amply sufficient for these examinations.

"Concerning the durability of spermatozoa, Ritter asserts that he has discovered them after a period of four years. To show how, when dried, they will bear rough handling, I may add that I rolled and twisted between my fingers a stained piece of muslin till it was in the form of a string, unrolled and twisted it over again two or three times, using much force, and was yet able by my method to discover spermatozoa without much difficulty.

"I claim for my plan extreme simplicity, ease of execution, and the greatest degree of certainty, for piece after piece of the stained fabric can be put to the test, with the assurance that nothing in the process destroys the spermatozoa, and that they may be found if present."

The Distinction between Leucorrhœa and Gonorrhœa.—Vaginal catarrh is so common, as the result either of a scrofulous diathesis, of the irritation of worms, or of the presence of dirt and filth, especially among the very poor, that we can safely assume that most cases of running at the privates that present themselves to us are cases of leucorrhœa, unless we find that the alleged assailant has or has had gonorrhœa, and that the discharge does not readily succumb to treatment, but grows more profuse, or at least that the inflammation associated with it shows a tendency to extend inward. I do not know that there is any evidence of a diagnostic difference in the character of the discharge, either as seen by the unassisted eye or as viewed through a microscope. A severe benign inflammation, especially if the parts are not kept clean, may cause ulceration, and blood will mingle with the mucus and pus, as it sometimes does in gonorrhœa.

Says Dr. Alexander Russell Simpson, in Quain's "Dictionary of Medicine," "Unless a clear history of infection can be obtained, it is almost impossible to establish a distinction between a gonorrhœal disease and the simpler catarrhal leucorrhœa. In the former there is a notable tendency to spread to contiguous surfaces. In children suffering from the infectious discharge, traces of the injuries that are usually inflicted at the period of infection should be sought for."

Berkeley Hill writes, "The distinction between vaginitis from contagion and vaginitis from non-specific irritation is always difficult, and sometimes impossible, being mainly determined by collateral evidence. It generally has a contagious origin if there is pus in the urethra."

MEDICAL TESTIMONY.

The medical witness is ordinarily called upon to testify before a grand jury, in a police court, where there is sometimes a jury and sometimes not, or in one of the higher courts, where there is always a jury. In some cases he may have to testify in all three of the places. And in these days, when "interviewing" is a salient feature in the management of many newspapers, he may be called upon to give his opinions to the press. If after careful consideration he believes that it is wise so to do, he should write out what he has to say, or see that the interviewer writes exactly what he wants said. It is sometimes best to keep a copy of testimony so given. Otherwise the editor cutting down or rearranging "copy" handed in, or the reporter writing alone from memory, may change the drift of an interview.

In the grand-jury room the witness has but to tell what he knows, what he has found, and to answer such questions as may be put to him by the prosecuting or district attorney and any juryman, in as *simple, plain, untechnical language* as he can command. There is no necessity for his being disturbed in mind, as he will not be contradicted, and there is no opposing lawyer to attempt to muddle him with hypothetical and intricate questions. But it is necessary that he should have clearly in his mind dates of examination, exactly what he found, and how he arrived at his conclusions. His note-book should be in his pocket, that it may be produced in case an entry is called for. Plain, untechnical language is necessary, as the average grand jury is composed largely of plain men, with only a fair knowledge of science and rhetoric. Besides, there is no time for rhetorical or scientific display.

In the courts the witness must remember what he testified to before the grand jury. Discrepancies in statements are injurious alike to the reputation of the witness and to the cause of justice. In both the police and the higher courts the witness must expect occasionally to have to contend with lawyers who care nothing for his reputation as long as they think they see their way clear, by sarcasm, belittlement, plausible but specious questions, etc., to gain their end,—the success of their side. For this reason among others, and because an astute lawyer will—for the time being, at least—closely study the medical issues in the case, the medical witness should have his testimony well digested and be prepared to present it calmly, readily, plainly, and logically. It is pitiful, in a crowded court-room, before an intelligent jury, a number of lawyers, some of them learned, and the judge or judges on the bench, to see an educated physician of good repute stumble and make such involved statements, so full of technical terms, that they either are not understood or are laughed at. Too frequently are such sights witnessed. A tricky, ungentlemanly lawyer, seeing his opportunity, harasses the physician with long hypothetical questions, which he does not readily grasp, and which he answers almost at hap-hazard, because he is disgusted or is anxious to get through; or questions are put to him in such a way that if he answers them he is obliged simply to say "Yes" or "No," while by so answering he feels that he is not doing as his conscience tells him he should, but only as he thinks the law compels him to. The rights of the witness should be, and generally are, protected by the presiding judge; but witnesses seldom know that they have the right to appeal to the judge as to whether they shall answer such and such questions in the way in which some lawyers insist they shall be answered. In many courts much latitude is allowed as to the forms of questions, and some lawyers have come therefore to believe that a witness must always answer as the lawyer intends that he shall. The truth is that, if a question cannot be truthfully answered "Yes" or "No," it need not be answered, though the lawyer insists that it shall be. But it should be so answered if it can be done truthfully.

Again, the witness must remember that there is generally ample time for calm, deliberate answers, and that he has the right to insist that questions shall be repeated or put in such form that he can understand them. Straightforward, honest, well-digested testimony with no attempt at display has the most weight. Woe to the man who attempts a line of testimony for display or mere notoriety! Expert testimony should be given only by experts,—*i.e.*, persons who have qualified themselves by special study and long experience in specified departments of medical knowledge and work.

Special study does not make a specialist or an expert. An honest, well-grounded opinion from a general practitioner is sometimes of more avail than the opinion of some would-be experts. Declaring one's self an expert brings with the declaration an increased responsibility, an increased susceptibility to censure, contradiction, and entanglements in lawsuits.

With questions of law—legal name of crime, punishment, etc.—the medical witness has nothing to do. It is for him only to tell the truth about the case as he understands it from a medical stand-point, to have carefully studied his case, to have made his notes and brought them to court, to testify unostentatiously and quietly, to give mere opinions guardedly, and to remember his rights as a witness and his responsibility as a member of an honorable profession.

PART IV.

DISEASES OF THE NERVOUS SYSTEM.

GENERAL INTRODUCTION TO THE DIAGNOSIS OF DISEASES OF THE NERVOUS SYSTEM.

By ALLAN McLANE HAMILTON, M.D.

In our examination of children we are beset with certain difficulties which make the matter a much more difficult one than in the adult patient, for, with the very young especially, we are obliged to rely mainly upon objective symptoms, and are placed in the position, to some extent, of the veterinarian, who is forced to depend chiefly upon his own powers of observation.

In the undeveloped human being it is of absolute importance to take into account the value of expression, and realize just how much weight to attach to the child's powers of perception, comparison, and judgment, to its capacity for description, and to the state of its emotions and their connection with disturbed sensibility. Young children, like all other young animals, are largely emotional. In the estimation of sensory disturbances, particularly those of a hyperæsthetic nature, there is always great danger of exaggeration upon the part of the patient, and our efforts must be in the direction of localization and consistency of manifestation, rather than of degree; and for the appreciation of the latter we are to regard their general effect, and bring to our aid the average of previous experiences.

The diseases with dominant expression are, of course, objective, and are usually conspicuous enough whether the symptoms be convulsions or lesser hyperkineses, or paralyses with or without contractures. During very early life it is often difficult to make any diagnosis whatever of conditions which are to become chronic and conspicuous at a later period, and it is a fact with which we are impressed every day, that an intelligent history of the development of many nervous diseases of children is not to be obtained from the parents; in fact, the commencement of such maladies as pseudo-hypertrophic paralysis or infantile paralysis often cannot be determined accurately, if at all. The education of parents in regard to the development of their progeny is lamentably deficient, and they take little pains to gauge or compare the development of faculties in their own babies with those of other children, a certain kind of cleverness or aptness dwarfing all else.

Where objective appearances are those which alone guide the medical man, much may be learnt of inascertainable subjective suffering by the position of the patient, by its movements, and by the involuntary expressions which often afford a clue to the existence of painful disease. As examples, I may speak of the peculiar retraction of the head which is so characteristic of cerebro-spinal meningitis, or the fixation of the body which is an almost constant accompaniment of disease of the spinal envelopes or the vertebræ themselves. Who is not familiar with the lateral rolling of the head which is so common in certain acute organic cerebral affections of early childhood? There are other equally valuable indications of obscure disease the nature of which cannot be easily ascertained through any subjective complaints.

The hereditary nature of certain disturbances has come to be well recognized, so that family forms have been described by various observers, and in diagnosis the value of such an origin will be apparent. There are three forms of disease which are conspicuous in this connection. One of them is pseudo-hypertrophic paralysis, and in many instances there are at least two if not more cases in the same family, the female side being that on which the disease has been transmitted. This is Bramwell's experience, and that, I think, of other writers. In hereditary locomotor ataxia this influence, though rarer, is decidedly more conspicuous. Carré reports in one family that there were eighteen cases of the disease in three generations. In this connection we should never lose sight of the existence of syphilis, tuberculosis, and cancer in the family history, and their bearing upon diagnosis.

CRANIAL PECULIARITIES.

There are many peculiarities in the appearance of the head presented by young children who may later develop evidences of cerebral disease; and at a subsequent period, when they are expected to perform certain ordinary actions common to children in general, there will be found a loss of function, or, more exactly speaking, a non-development of function, which suggests some corresponding abnormality in the formation or growth of the brain. The external cranial conformation of such children is generally striking: there is a decided disproportion between the size of the head and that of the body. Sometimes, as the result of ventricular effusion, or an excessive peri-encephalic collection of fluid, we find a hydrocephalic enlargement, with pulsating or depressed fontanels; or, as an indication of various cerebral structural defects or deposits, there may result asymmetrical deformities. These latter, however, as a rule, are indicative of some inherited neurotic influence. Or a cranial asymmetry may correspond with the lesion in cases of hemiplegia spastica infantilis.

Certain young children, whose only defect seems to be a psychical one, present a peculiar flatness of the face, with patulous and prominent lips, large mouths, and small and widely-separated eyes, with irregular and unusual development of teeth. These subjects are often marasmic, weak, or rachitic, and ordinarily lie with limbs flexed, developing an amount of intelligence which is shown only in the simplest acts which concern their appetites. "They look listlessly out upon a blank world" (Down). In these it is not unusual to find some associated congenital defects, such as supplementary toes or fingers, webbing, or nævi. Such children are irritable, or else abnormally good-natured, sleeping most of the time, or at times they perform automatic movements of a rhythmical variety. It is advisable to take very accurate measurements, with lead wire, of the formation of the head and the size of the body.

SENSORY DISTURBANCES.

Points of Determination.—Tactile, analgesic, anæsthetic, hyperæsthetic, paræsthetic, disorderly, or subjective delayed perceptions; sensibility to heat and cold (determined by test-tubes filled with water at different temperatures). Location; in departures from normal integrity; appreciation of exerted power and its adjustment (muscular sensibility); appreciation of weights by means of the baræsthesiometer, the notched wheel, paper and metallic balls, etc. The posture of limbs, as in tabes; inharmonious relation between visual impressions and certain muscular actions; Duchenne's test, excessive innervation and over-supply of force for accomplishment of required act. Existence of delusions or hallucinations; moral changes; violence; destructive tendencies; periodicity; depression; exaltation; one-sided peculiarities; ecstasy; stupor; apathy; sexual perversion; morbid appetite. Ophthalmoscopic examination; light tests; tuning-fork and watch tests; tests for anosmia.

PAIN.—The greatest difficulty that we have to encounter in children is the estimation of pain, for not only are they apt to exaggerate, but they are unable to locate or describe their discomfort in a way that is at all satisfactory or convincing. West and other writers have expressed themselves very forcibly with regard to the significance of pain in the very young.

"In grown persons," says West, "there is a large class of ailments which consist of simple pain; they call the disorder neuralgia,—nerve-pain,—and mean thereby that such pain is independent of local disease: it is produced by the influence of malaria, it follows on loss of blood, or is associated with various altered states of the circulating fluid, or in other cases it is impossible to determine the exact cause to which it is due. Difficulties of its cure render it one of the opprobria of medicine, but it does not tend in any way to shorten life, and our patients at last find a sort of dreary satisfaction in the knowledge that the malady which renders existence so bitter is yet *only* neuralgia. In infancy and childhood, however, pain referred to any part signifies, almost without exception, that disease of some sort or other is going on there or near at hand. The tears so profusely shed do not prove that pain is the lot of the infant more than the grown person; but at one time cries are the only, as they long continue the most expressive, language. Hunger, sleeplessness, fatigue, discomfort of any sort, is expressed by cries; while the character of the cry goes far towards helping us to determine the nature of the suffering. But I have never in infancy known any instances of pain—severe, obstinate, recurrent—for which, sooner or later, a distinct local cause was not found; and even in later childhood the rarity of real neuralgia is extreme." The cry of the child who suffers from pain due to organic cerebral disease is short and piercing.

In the determination of sensory disturbances much care must be shown in the exhibition of tests, for it is impossible to gain from most children anything more than a guess when the æsthesiometer is applied in the manner directed by text-books generally. It is much better to employ what I may call the *direction test*, which implies the existence not only of tactile sensibility, but also of a more complex cerebral appreciation of direction and distance. After the patient's eyes are blindfolded and his fears are quieted, the finger of the observer may be passed upward or downward or transversely in different situations, the patient being required to describe the direction of the passage of the finger and the side of the body upon which contact has been made. It should be determined first whether capacity exists for perceiving impressions at all, the location, the sensibility to temperature, or to painful irritation, or to weight.

Pain referred to the head, and lasting any time, may be looked upon as a serious indication of organic disease of the brain, and Marshall Hall and many of the careful writers of the early part of this century have shown how important it is to recognize the existence of such pain, accompanied by frequent and causeless vomiting, as a pathognomonic symptom of tubercular meningitis. But, in fact, this symptom connected with delirium and coma is an indication of many of the coarse cerebral diseases of infancy.

Neuralgic pain is rare in infancy, or at least until after the sixth year, when headaches of a migrainous type are found commonly among overworked school-children, or as a sequel to some malarial or typhoidal fever. Trigeminal neuralgia is more common in girls, and is often associated with eczema. It is safe to assume, however, that severe head-pain in young children is an alarming symptom, especially if the pulse is irregular and feeble. The headache is usually general, or it may be confined to the frontal region. Intense headache, with associated photophobia, sensory hyperæsthesia, and constipation, is, as a rule, highly characteristic of meningitis.

The variety of headache of a lighter grade which appears in children after the third or fourth year is, to all intents and purposes, a hemicrania.

It is developed by fatigue or undue strain at a time when the brain is attaining its early development, and is aggravated by errors in diet and management. It is associated with gastric irritation, nausea, and loss of appetite, and usually begins in the morning. In many respects it resembles the ordinary sick-headache of the adult.

There are other forms of characteristic pain which require notice: the most important of these is that referred by the child to the knee, and which most often indicates some affection of the hip-joint which later develops itself. This knee-pain is sometimes very severe and intermittent, and the real disease is apt to be masked for a considerable time.

Certain pains of a lesser grade occasionally attract our attention, and among these I may mention the tenderness at the elbow and wrist-joint in certain cases of chorea which are not rheumatic and seem to bear no close connection with the movements. Neuritis, which is so common in the adult, is a rare disease in early life, although within the last year I have had two cases in young children where the pain was accurately localized, and in one, of idiopathic nature, there was double extensor paralysis of the forearms as a result. It is rare to find any of the ordinary forms of neuralgic pain of the trunk or extremities, though the contributors to Gerhardt mention a variety of cases, and parallels are found for almost every kind met with in adult life. Bouchut reports a case of cervico-brachial neuralgia in a girl of one year. Intercostal neuralgia, especially of the twelfth dorsal nerve, is occasionally encountered, and heed should be paid to the confusion of this neurosis with actual pulmonary or spinal disease, Eulenburg reporting no fewer than twenty-nine cases in children. Bohn reports a case of lumbo-crural neuralgia with zoster. The neuralgias of the lower extremities are most obscure, and, as a rule, are symptomatic of morbus coxarius or neuromimesis.

Disturbances of Hearing, except in their association, are not worthy of extended comment. Where a collection of acute cerebral symptoms depends upon aural disease, of course the necessity for making complete tests of the condition of the auditory apparatus will be seen at once.

There is a form of epilepsy which originates in otitis, and in which the attacks are always excited by irritation of the meatus or deeper parts. In such cases, not only should the aural disorder be investigated, but it is well to determine the degree of psychical disturbance which is commonly a feature of the condition. The acuteness of hearing should be estimated. In meningitis it is very decided, and is associated with various hyperæsthesia. In certain conditions of feeble-mindedness it is dull, or there may be absolute deafness. In the latter case there is often diminished sense of taste and smell.

Disturbances of Vision are rarely fully recognized until the child attains an age when it can communicate its loss. Certain idiots are blind from birth, and their eyes do not follow bright objects moved before them, or there is no discomfort produced by bright lights suddenly brought in front of the eyes. In such cases there will be inactivity of the pupils. In other cases the blindness may be simply psychical. The development of tuberculous or other brain tumors is very frequently symptomatized by blindness, and ophthalmoscopic examination reveals optic-nerve degeneration.

THE PSYCHOSES.

Almost all the mental diseases of adult life may be duplicated in childhood. In making this assertion we of course exclude the degenerative affections accompanied by coarse destruction. Emminghaus, in his admirable volume, describes the various forms of melancholia, mania, paranoia, acute dementia, transitory, periodical, and circular, as well as moral insanities, while no fewer than eleven pages are devoted to the hysterical disorders of childhood. Feeble-mindedness as the result of idiocy and imbecility is quite familiar, and is perhaps the most common form of infantile intellectual disturbance.

The diagnosis of insanity is far less easy in childhood than in adult life, and the observer is very apt to confuse the normal emotional and moral disturbance, which is simply considered as viciousness, with something much more grave. Among very young children it is a matter of considerable difficulty to discover the existence of delusions or hallucinations; in fact, hallucinations are far less common during the earlier years of life than at a subsequent period. The mania of childhood is rarely continuous, but during its existence is symptomatized by great violence, by destructive tendencies, and occasionally by attempts at suicide, although the latter are more characteristic of melancholia. It is well to estimate properly the character of certain acts of purposeless cruelty which often symptomatize the moral perversion which is connected with the various grades of mental disturbance. Some insane children, even though their surroundings be of the best kind, are very apt to inflict needless pain upon those about them, to torture animals, afterwards manifesting no remorse. Or occasionally among epileptics we find what may be regarded as a pyromania, though a pure spirit of mischief is the usual explanation. It is well to question these subjects closely, for it will often be found that they possess a fair amount of intellectual vigor, and that they are quite at a loss to explain their impulses or morbid tendencies. Instances of introspective insanity, especially at the time of puberty, are by no means uncommon, and occasionally forms of *folie du doute* are reported. Langdon Down speaks of a child whose mental peculiarity consisted in the fact that as he passed a dirty beggarman in the street he was afterwards afraid to go near his mother for fear of contaminating her, and he could not be persuaded to touch her at all. The melancholia of childhood is very apt to be symptomatized by religious delusions, and rarely by delusions of persecution and conspiracy; and this is notably true at puberty.

In our examination of patients with pubescent insanity, we are to observe the existence of a possible morbid vanity and boastfulness, perhaps connected with a certain hypochondriasis and state of self-accusation. We are to distinguish insanity from hysteria, which latter differs but little from that of adult life, though the convulsive variety is rare. We are to determine the existence of the latter by the exhibition of certain tests, such as the suggestion of punishment, or an appeal to the appetite of the subject; and in this connection I may speak of an instance of hysterical dysphagia which had lasted for several months and which was readily cured by the offer of a piece of plum-cake. The exhibition of the cautery or some such revulsive will often make the diagnosis perfectly clear. In these cases we must detect, if possible, the existence of hyperæsthesia or of nervous anorexia, and analyze the nervous derangements of respiration which may be present. Sometimes there is a motive for deception; and this should be detected if possible.

The intellectual condition of feeble-minded children is one of great interest. It is very often irregular and one-sided, there being perhaps a suspicious brightness and extreme development of some faculties, while others are correspondingly dull. Certain feeble-minded children have the power of improvisation, are fond of music, and show marvellous proficiency in arithmetic; intricate calculations are performed in an incredibly short space of time, and some subjects of this kind can multiply three figures by three figures as soon as written upon paper. In other ways they possess little judgment, are often regardless of the loss of friends, though ordinarily affectionate, are sly and very mischievous, and possess deficient morals. Some of them make little muscular exertion, and even when the time comes for them to walk they do not do so. There is usually a hypersecretion of saliva, so that they drool at the mouth. They examine objects presented to them in a peculiar way, reminding one of monkeys, and it is a common trick for them to feel the shape and nature of objects by the tongue (Down).

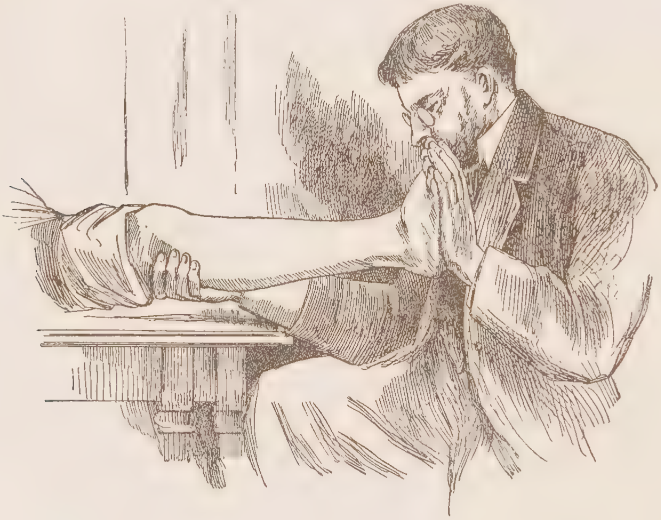
There is a rare form of infantile dementia of an acute character which should not be confused with congenital conditions of feeble-mindedness. The child, usually a female, is overtaxed during development, and presents a train of symptoms beginning in the debility which follows convalescence from acute disease, often typhoid or scarlatina, and ending in amentia. There are outbursts of fury, mental confusion, loss of memory and comprehension, and stupid silence. Cataleptic rigidity, anomalies of circulation, lowered temperature, salivation, and vacancy of expression occur at some time or other.

MOTOR DISTURBANCES.

Points of Determination.—The estimation of the degree of loss of power, —whether paresis or complete paralysis, whether the paralysis is idio-muscular or of central origin (to be determined by electricity); the association of atrophy with paralysis; the loss of special muscular function, or associated losses; the course of a progressive paralysis; the determination of retrogression; the mensuration of lost power (by means of the dynamometer, which is unreliable, and of value only in comparison); the appreciation of the course of spasm, whether local or general; fibrillary (idio-muscular can be evoked by slapping muscles), systematic spasm; the determination of incoördination, as seen in the gait or in the movements of the upper extremities (can be tested by making the patient perform acts of precision, or those requiring neat localization). Paradoxical movements; chin, wrist, abdominal, cremasteric (in boys after puberty) reflexes, knee-jerk, ankle-clonus; whether reflexes are increased, absent, or transferred.

The methods for ascertaining the condition of the reflexes differ in no way from those employed in adult life. It is always best to suspend the legs over the edge of a table or chair when the knee-jerk is to be produced, and in this position we may also determine the condition of the reflex in the tendo Achillis. (Fig. 1.) The child should be placed well on the table, so that there may be no general shock given to the thigh. The ankle-clonus may be evoked in the manner depicted in the cut. Sudden and forcible pressure should be made. Sometimes when the foot is first extended the subsequent flexion will give rise to the clonic spasms much more quickly.

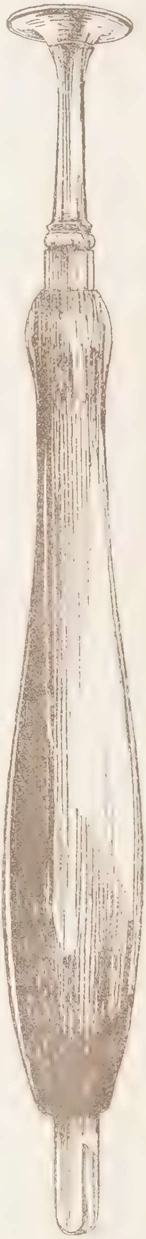
Fig. 1.



ELECTRICAL DIAGNOSIS.

For diagnostic purposes the medical man should be provided with a galvanic battery of at least thirty cells, and an induction apparatus. Pole-cords for either, composed of fine copper wire, are to be selected, in preference to the ordinary woven tinsel conductors usually supplied with the cheaper instruments. In addition to these, suitable electrodes (Fig. 2), one of which at least is to be provided with a key for opening and closing the current, will meet every need. It has been my custom for several years to use as simple an apparatus as possible, and I have therefore discarded the elaborate "table-boards" supplied with useless switches and interrupting apparatus, and I now employ for diagnostic purposes the galvanic battery and water rheostat and a galvanometer (that of Gaiffe being perhaps the best), while the simple milliampèremeter of Barrett of New York meets every indication, though the figures are somewhat difficult to read. The milliampèremeter is placed within the circuit, and we are enabled to read exactly the amount of resistance,—a much more exact method than the old one of calculation, in which the number of cells used was taken into consideration. The uncertainty of the old way can be readily imagined when we appreciate the fact that the electro-motive forces of no two cells or kinds of cell in use are exactly the same. The absolute measurement by the milliampèremeter is always fixed, no matter what the source of power may be.

FIG. 2.



In electrical diagnosis we are to estimate the quantitative reactions as well as the qualitative; and I refer my readers to any of the modern treatises upon electro-therapeutics for more extended information.[1] We are to find the condition of sensibility to both galvanic and induced currents, and to detect hyperæsthesia or anæsthesia; but the chief value of this agent in diagnosis is in the detection of impaired tone of nerves or the existence of muscular degeneration, to fix the degree of such changes and to trace their origin, and to detect the first indication of a return to the normal state. Then, too, the important question is determined whether a degenerative condition be a peripheral or an idio-muscular one, or whether it is due to a suspension or abolition of nerve-supply, or whether the distal muscular disorder is dependent upon some cerebral or spinal disease. These matters in special articles will be minutely gone into, and I may preface their discussion by a brief reference to the behavior of muscles when subjected to galvanic stimulation, at the risk of reiterating doctrines which may be considered trite.

In testing the integrity of a muscle, we are to observe the character and kind of contraction which follows the opening or closure of the current, which places it in the condition of "catelectrotonos" or "anelectrotonos," the terms referring to the state of contraction while influenced by the cathode or anode. A variety of abbreviations have come into use for the purpose of working out formulæ of muscular contraction, which are as follows: Ka = cathode, An = anode, S

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[1] See the admirable works of Amidon, Bartholow, or Birdsall.

(or Cl) = "closing," O = "opening," Te = tetanic contraction, Z = contraction, Z″ = a stronger contraction, z = a weak contraction. The cathodal pole produces in the normal state the most vigorous contraction in closure of the circuit, whilst the reverse is true of the anode. Most authors name three grades of excitability which is the result of a stimulation of motor nerves.

"In the *first grade* the weakest current which will cause a contraction is a cathodal closing contraction, KaSZ (Ka, cathode; S, Schliessung, closure; Z, Zuckung, contraction), and no contraction can be effected by the anode. In the *intermediate grade* the current is one in which the cathode causes stronger contraction on closure, but no contraction on opening, KaSZ′, whilst the anode causes slight contractions both on opening and closing, AnSz and AnOz. In the *highest grade* the current is one that causes a tetanic contraction on cathodal closing, and a feeble cathodal opening contraction, KaSTe, KaOz, whilst on anodal opening and closing decided contractions occur, AnOZ, AnSZ.

"Such are the normal formulæ to be obtained from nerves accessible for galvanic excitation. The same are true of the muscle, for, as has already been stated, the muscular contraction is the same whether the stimulation be *direct* or *indirect*. It is evident that the reactions to galvanic stimulation are largely affected by the current strength, and, as the formulæ are constant for the different degrees of excitation, we are provided with an exact method."[1]

No such rules govern us in regard to the excitability of the muscle to faradic stimulation. Its importance consists only in its presence or absence and in its relation to the state of galvanic excitability, and this may be studied to advantage in those interesting conditions in which the "reaction of degeneration" is found. It is well to keep accurate notes, and perhaps charts which indicate the condition of the muscles at various times.

Many of the paralyses of adult life occur in the child, although the pathological causation is, of course, different. Infantile hemiplegia, which is often due to mechanical pressure and generally to rupture of one of the meningeal arteries, is less rare than that produced by a lesion of the deeper cerebral substance, which is usually of slow appearance and dependent upon tumor. Such paralyses occur before birth, or even afterwards, and are those which result in general asymmetry, even though the muscular power be restored to a great extent in adult life. The hemiplegias are nearly always connected with rigidity and increased irritability of the reflexes,—in other words, they are spastic,—and if they be of very early origin there is almost sure to be some deformity. Of Gowers's eighty cases it was more common in girls, and in three-fifths of all the cases the onset was during the first two years of life. There is another form of hemiplegia which is quite apt to be associated with chorea and which may be of embolic origin. This is rarely seen before the seventh year, and is by no means always a serious or long-lived affair, especially if there be no large central lesion.

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[1] Benedikt, Handbuch der Elektrotherapie, p. 81, and translation in Bartholow's treatise.

In very young children paraplegia is something uncommon, if we except the variety described by Erb and known as spastic spinal paralysis, in which, added to the loss of power, there are adduction of the lower extremities, and more or less general spasm and voluntary movements. In such cases the reflexes are exaggerated, the sensibility is normal, and the child has ordinary control over his bowels and bladder.

Such a thing as ordinary transverse myelitis, except from injury or the compression resulting from Pott's disease, is comparatively rare, although we sometimes meet with it as a sequel of some of the fevers; it hardly ever occurs as an idiopathic condition.

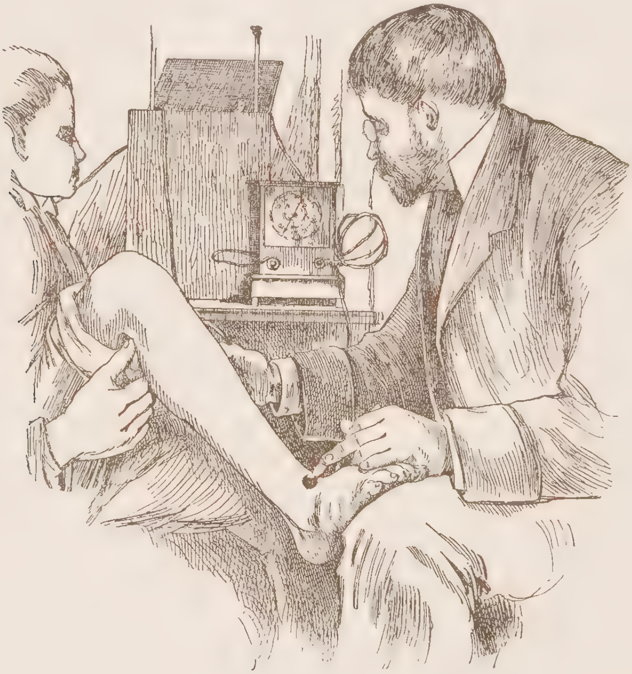
Undoubtedly the greatest number of infantile affections of an akinetic nature are those of polio-myelitis acuta, and we find this clearly shown by the books of public institutions and dispensaries. Such patients may present evidences of paresis and atrophy, the retrogressive history of the former being ascertainable from the parents. The atrophy is often extreme, and, if the case be at all confirmed, there will be an entire absence of electrical reactions. In these the knee-jerk is, as a rule, absent, and in this respect the diagnosis may be made from certain monoplegias or limited pareses of cerebral origin.

The diagnosis of wasting diseases from true infantile paralysis should not be difficult, because of the absence of paralysis, the susceptibility to electrical stimulation, and the general character of the atrophy, in the former.

Pseudo-hypertrophic paralysis of course presents the deformity so well known, in association with atrophy of parts once invaded, and by absence of the reflexes, while sensation is conserved. Here the chief paresis is seen in the quadriceps and the muscles of the back. Progressive muscular atrophy, so far as I know, is a disease unknown in childhood (except certain rare hereditary forms), and we may exclude this from consideration. The peripheral paralyses of childhood, though common, are ordinarily of different causation from those of adult life. They are usually the result of pressure, either from violence exhibited during delivery, or from injuries. Among these may be mentioned paralysis of the deltoid, or of the muscles of the upper extremity supplied especially by the musculo-spiral nerve. There are cases in which the dislocation of the humerus as a result of violence during delivery may give rise to nerve-pressure and a resulting loss of power which is often very intractable. Paralysis of the lower extremity from corresponding causes is quite rare.

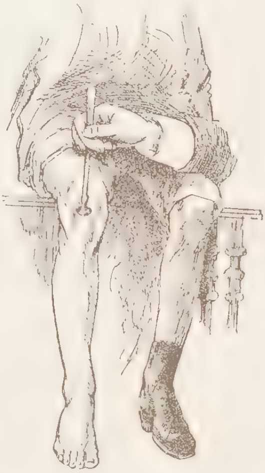
For the diagnosis of infantile paralyses of all kinds, we may avail ourselves of the same valuable agent that is of so much use in disorders of motility in the adult,—namely, electricity; but its employment is a much more difficult matter, especially in very young children, whose struggles and cries seriously interfere with any careful observations. It is well to have all preparations made before the apparatus is shown, and to be on the alert to watch the primary effect of electrical stimulation before the subject has actually experienced the pain of an application, and it is much better for some assistant to hold the child than for the mother or the friends to do so. The muscles should always be localized if possible, and one of the electrodes should have a small end, so that the current may be concentrated as much as possible. (Fig. 3.) With older children the examination is a matter of less difficulty.

Fig. 3.



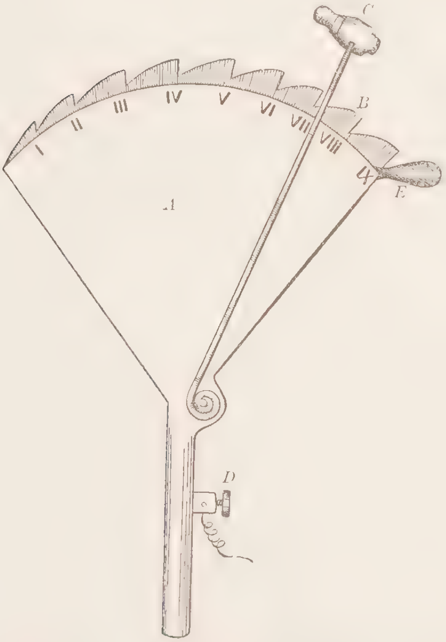
Too much importance cannot be attached to a careful consideration of all the slight actions of the child, and sometimes it manifests its weakness unconsciously. It is well to take moulds of muscular contour, not only for the purpose of diagnosis, but also to record the possible improvement that may follow treatment; fine lead wires may be applied to both sides and adjusted, and on their removal traces may be taken, which should be preserved for future reference. By this means muscular flatness and wasting are shown very beautifully. The length and amount of contracture of the extremities should be determined by accurate mensuration; the existence of atrophy and fibrillary contractions should be carefully noted, and in estimating the cause of contractures and joint-enlargements the possibility of their being hysterical should be taken into consideration,—for I am convinced that hysteria is much more common in children than is generally supposed.

Fig. 4.



In connection with akinesis it is always important to examine the reflexes, and it may be said that these are generally more active in children than in adults: to elicit proper responses we must provide ourselves with instruments of greater delicacy than those used for older persons. A small heavily-weighted percussion-hammer is to be preferred to one that is likely to jar the entire limb. (Fig. 4.) Ankle-clonus in children is perhaps of more importance than the increased knee-jerk. In acute polio-myelitis and pseudo-hypertrophic paralysis the reflexes are ordinarily lost, while in most of the other pareses they are increased. This is notably so in spastic spinal paralysis and the unsystematized forms, as well as hysteria. In connection with the akineses of the deuteropathic kind we usually observe very conspicuous circulatory disorders manifested in lowering of surface temperature and mottling which is increased by exposure to the air.

Fig. 5.



Brissaud's Apparatus for Registering the Knee-Jerk.—*A*, fan-shaped board; *B*, ratchet-catch holding arm; *C*, percussion-hammer; *D*, electrical communication with myographium; *E*, trigger.

The instrument of Brissaud (Fig. 5) enables the observer to measure accurately the force and the time of the knee-jerk, and the rate of transmission of the motor impulse. It consists of a fan-shaped board having at its inverted apex a spring with electrical communication with an electrical stylus and myographium. The spring superiorly terminates in a rod capped by a hammer. This rod can be detained at different points of a quadrant with ratchet notches, and may be released at the will of the operator by pressure upon a trigger. The force of the blow is indicated in pounds, and depends upon the tension of the spring. At the precise moment at which the blow is struck upon the patellar tendon the electric current is closed.

Disturbances of co-ordination are rare in children's diseases, except where they exist as a symptom of hereditary ataxia, or with cerebellar disease. The same rules that apply to the examination of adults may be followed here, for such tests rarely apply to subjectivity. Great care should be taken in distinguishing certain bad habits of movement from the actual loss of muscular sense or of co-ordination proper. The defective co-ordination in children of feeble mind is not, as a rule, due to any other defect than that of a purely mental nature. There are cases, however, of disseminated sclerosis in which in addition to the motor disturbances there is scanning speech.

Conditions of exalted motility, such as spasm and convulsions, are rather difficult of diagnosis, and especially the latter. The excitability of the young child, and its impressionability to all forms of peripheral excitement, make it liable during the early years of its life to convulsive and spasmodic seizures, which may be temporary, or may continue for some time, finally becoming epileptic. The duration of the tonic stage of infantile convulsions is a strong characteristic, and there is, as a rule, very little opportunity to localize in very early life the situation of the cerebral lesion. In those cases where the irritation of dentition or gastric disorder is the exciting cause, we find that the convulsions are repeated, and bear a certain relationship to the intensity and duration of the peripheral disorder, but clinically there is very little that is distinctive. The surface is usually pale, the pupils are dilated, and the muscular convulsions rather marked. It is important to distinguish the convulsions which are ordinarily designated as eclamptic from those which are symptomatic of true organic disease of the brain. The former are often easily explained by the cessation of the morbid condition after the removal of the cause. The profound disorders of early childhood, when such great demands are made, implies an unstable condition of the gray matter, with discharges upon the slightest provocation, and when the general constitutional condition is lowered in any way, or there is an unusual tax upon innervation, we find that a common expression of such demand is an epileptic paroxysm.

It is well to determine the connection of rickets, that familiar condition manifested by imperfect bony development. Gowers, in speaking of the connection of rickets with convulsions in children, says, "The essential element in rickets is defective development; the perversion of development that occurs (*e.g.*, in the bones) is secondary to, and consequent on, its defect. At the time at which this constitutional state chiefly occurs, structural development of the nervous system is complete. But it is probable that functional capacity is only fully developed after structural perfection, and the parts last developed may suffer from the general delay in development more than those parts that have been longer perfect and longer in full use. . . . It is certain that in rickets there is excessive activity of the centres of the brain and cord on which reflex spasm and convulsion depend. It is probable that the morbid tendency is exalted by an inherited neurotic disposition. We must except those forms of eclampsia that occur between the sixth and eighteenth months of life."

Gowers and others do not attach as much importance to irritation of the first dentition *per se* as to the rachitic state itself. It is undoubtedly true that in a debilitated child any peripheral irritation, such as that produced by the presence of intestinal worms or of indigestible food, may be alone sufficient to give rise to a temporary form of eclampsia.

We find that in children, especially those in the marasmic state, where there is excessive cerebral anæmia, convulsions may be produced.

It is important, as I have said, to make the distinction between these lighter grades of trouble and the epileptoid state when it owes its causation to some organic disease of the brain. In the latter instance the diagnosis may be positively made when the convulsions are unilateral, or when they are connected with other evidences of destruction of the nervous tissue, such as paralysis or psychical derangement. There are, however, a large number of cases of extensive brain-disease in which there is no evidence of one-sided seizure. There is, at first, more or less regularity in the occurrence of the fits, and after a time they acquire the periodical character of genuine epilepsy. In such cases it is well to make an ophthalmoscopic examination for the purpose of determining the possible existence of fundal lesions, and we should never neglect to ascertain, if possible, the existence of scars or other evidences of head-injury and the family history of predisposing conditions. Sometimes traces of congenital syphilis may be found which will make the accuracy of the diagnosis almost a certainty.

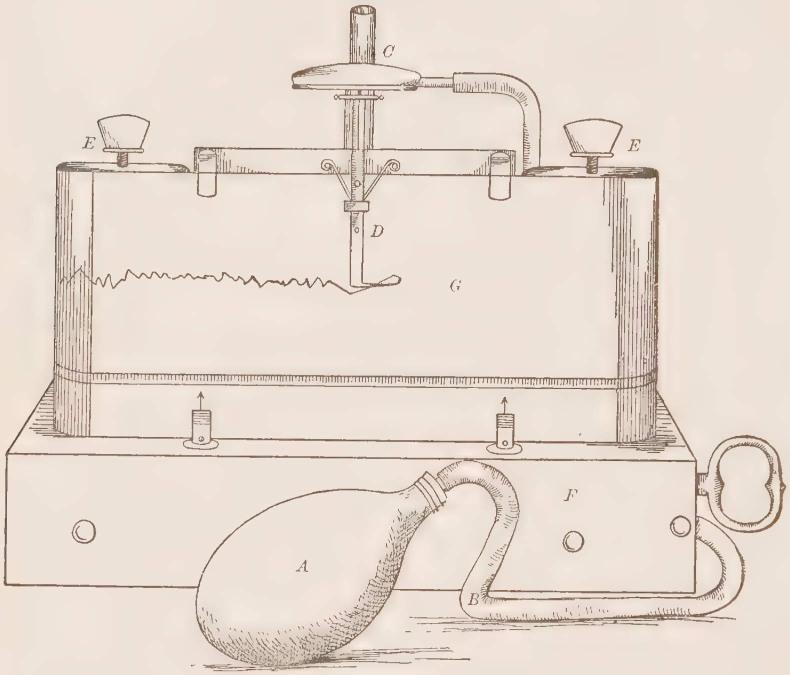
While tremor is almost unknown in early life, we find certain irregular jactatory movements which symptomatize the disease known as chorea, which is correspondingly rare in adult life. It is unnecessary to refer in detail to the diagnostic points of this most common of diseases. It behooves us to study the association of such movements with rheumatism and cardiac disturbances, the possible existence of overwork and malnutrition, and the climatic influences, bearing in mind that the disease makes its appearance in the spring, as a rule, and that it is not unusual for several relapses to occur. We must take into account the possible existence of an associated weakness of the right half of the body, which, however, must not be confounded with a genuine hemiplegia of cerebral origin.

For the estimation of tremor and its graphic registration I have devised an instrument which is useful as a clinical myographium. (Fig. 6.) The india-rubber bulb is held in the hand, or a corresponding elastic apparatus is applied to other parts. By compression of the air in a Marey tambour, even the most delicate movements are registered on a piece of paper which passes before a stylus.

The condition known as post-hemiplegic chorea is rather a disease of adult life, and the movements are finer, and in their clinical character more approach *tremor* than chorea proper.

Infantile tetanus, or tetanus neonatorum, must be differentiated from eclamptic conditions, which it resembles, though consciousness is preserved. We must also recognize the climatic influences, and bear in mind that it is essentially a disease of very early life.

FIG. 6.



THE AUTHOR'S DYNAMOGRAPH.—*A*, rubber ball; *B*, tube connecting with tambour; *C*, tambour; *D*, registering stylus; *E*, barrels carrying paper; *F*, box containing clock-work; *G*, paper with second-markings.

SPEECH-DISTURBANCES.

The speech-disturbances of childhood are of three kinds,—the *alalic*, which comprises the purely psychic disorders; the *paralytic* or ataxic, which originates in the disorder of the motor centres or co-ordinating apparatus; and the *dyslalic* (stuttering).

Aphasia in young children is a rare affection, and of course little or no success will follow our attempts to diagnose such conditions as alexia, for instance, unless the child be well on in years. The psychical difficulties are much more likely to be those dependent upon some mental deficiency, and are expressed in paraphrasia and echolalia, or a tardiness in interpreting symbols and applying them. We must bear in mind, in examination of these cases, that, unlike the adult aphasias, they occur with imperfect or limited development of faculty, and it is therefore impossible to apply the same diagnostic rules that we use in cases of adult speech-disturbance of cortical origin.

We should be careful to distinguish true cerebral defects from those which are the result of vicious training, where foolish mothers encourage their young children in the use of baby synonymes,—a habit which is sometimes kept up for several years. I have seen a child who was paraded before people and made to go through its performance of talking in a "language" it had formed, which was to all intents and purposes a genuine paraphrasia. This child had been encouraged to formulate a hybrid means of communication with another of the same family, and the new tongue was used for their little confidences, and in fact so constantly that it was several years before the child could learn to speak grammatical English. A remarkable case of this kind was reported some years ago by the late Dr. Edward Hun, of Albany.

In cases of true cerebral speech-defect we may sometimes find an associated peculiarity in handwriting, which was first pointed out by Ireland and others, and which has been described by the German writers under the name of *spiegelschrift*, or mirror-writing. I have met with two or three examples of this, in which the child not only invariably wrote backward, but made drawings reversing the relations of objects.

The ataxic disturbances of speech may be found as a result of sclerosis which sometimes follows scarlet fever, or in connection with aggravated chorea. In the latter case it is not uncommon to detect some explosive articulation, and possibly the disturbance of phonation which has been accounted for by laryngeal chorea.

A variety of speech-embarrassments which come under the head of paralalia (stammering) or dyslalia (stuttering) depend not only upon mechanical but often also upon mental defects. It is well to determine whether there be cleft palate, pharyngeal or nasal disease, enlargement of the tonsils, or an abbreviated frænum which gives rise to tongue-tie. The common forms are the following: *lalling*, where *r* is substituted for the consonant sounds; *lambdacismus*, where there is an inability to pronounce *l*; *rhinismus*, which gives rise to nasal intonation and is dependent upon some obstruction; *rhotacismus*, where the letter *r* is imperfectly pronounced, and, as Potter has pointed out, the use of the guttural *r*,—the former being produced by the vibration of the uvula, the passage being obstructed by the approximation of the back of the tongue to the soft palate, the latter by the vibration of the tip of the tongue against the hard palate; *stigmatismus* (lisping); *gammacismus*, where *t* and *d* are used instead of *k* and *g*, etc.

The importance of recognizing the condition of the organs in these familiar defects of speech cannot be over-estimated. Sometimes the tongue is hypertrophied in connection with other appearances suggestive of idiocy or cretinism. Again, we shall find paralysis of this member attended by difficulty of protrusion, or there may be some irregular or imperfect muscular contraction interfering with phonation which is the result of diphtheritic paralysis or some more obscure disorder.

The existence of mutism may depend upon absolute deafness, upon such mechanical defects of the speech-apparatus as I have described, or upon mental defects. According to Downs, thirty-six per cent. of feeble-minded children are dumb, thirty per cent. speak indistinctly, and not more than twenty-eight per cent. speak fluently.

VISUAL AND AUDITORY DEFECTS.

The same diagnostic rules which govern us in adult cases hold good with those under consideration. We are to remember, however, that in children there is apt to be more or less weakness or spasm of some muscles of the eyeball, which has no connection with central disease, and it is always best to wait in a case of long chronicity until the child attains an age when attempts at correction may be made by means of glasses, excluding, if possible, the particular symptom before we can attach any importance to its presence or association. A recent case which fell under my observation is an illustration of this. A child of two years presented a paralysis of the upper extremity which had been pronounced by one physician to be of central origin. After two careful examinations I came to the conclusion that it was a genuine peripheral paralysis due to pressure and injury received at its birth. Six months after this diagnosis the child presented an internal strabismus of the right eye, which somewhat staggered my belief, and I promptly sent it to an oculist, who considered it to be an extra-cerebral defect, but counselled the use of glasses at a subsequent period. The paralysis of the deltoid and upper-arm muscles disappeared in time, and the squint was cured by properly-applied prisms.

The existence of hypermetropia is so common in early life, and so often assigned as a cause of nervous disease of various kinds, that we must always make allowance for its presence. The pupils of children, so far as my experience goes, are perhaps much more active than those of the adult, and conditions which give rise to irritation of the sympathetic fibres with consequent dilatation are much more common than the reverse. In hydrocephalus, as a rule, we find at first pupillary dilatation, and afterwards contraction; and in the hydrocephaloid disease, and, in fact, in all other diseases of an anæmic nature, there is dilatation and very sluggish response.

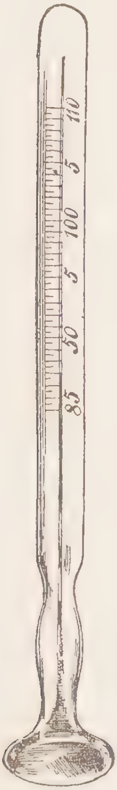
In certain spinal diseases of children, among which is the rare one hereditary ataxia, there is a loss of the pupillary reflex to light. Disturbances at the fundus are, as a rule, very pronounced and easily distinguished.

The careful diagnostician can hardly overlook the condition of the pelvic organs. The incontinence of urine occurring in convulsive diseases, as well as in the psychical disorder known as "night-terrors," and the actual mechanical and local interference with function in spinal disease, are equally worthy of recognition.

TEMPERATURE- AND TROPHIC DISTURBANCES.

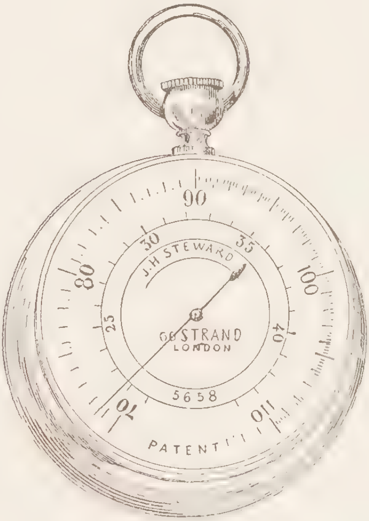
Variations in the surface temperature, especially in cases of acute nature, must be carefully determined. Thermal changes in certain chronic spinal troubles, such, for instance, as compression-myelitis from Pott's disease, must not be disregarded. We may avail ourselves of the special thermometer of Seguin (Fig. 7), or of that of Steward of London (Fig. 8), the latter being an easily-adjusted and thoroughly satisfactory instrument. It is necessary to make frequent examinations in a place free from draughts, and to leave the thermometer in contact with the surface at least fifteen minutes.

Fig. 7.



Seguin's thermometer.

Fig. 8.



Steward's surface thermometer.

Trophic disturbances of the skin, bullæ, bed-sores, glossy skin, pigmentary deposits, eczema, purpura, herpes, acne, changes in the nails and hair, thyroidal enlargements or atrophy, mucin deposits, true muscular atrophy, corneal changes or ulceration, must all be looked for in certain cases, and are often as important as more dominant conditions.

HYDROCEPHALUS:

DROPSY OF THE BRAIN, WATER ON THE BRAIN.

By FRANCIS T. MILES, M.D.

The essential feature of hydrocephalus is an abnormal accumulation of serous fluid within the cranial cavity. The location of the fluid may vary. It may occur between the dura mater and the arachnoid, the so-called arachnoid space, or fill the meshes of the pia mater external to the brain (external hydrocephalus), or occupy the ventricular cavity, or be found in both these latter localities at the same time. The effusion may result from various causes. It may accompany coarse disease of the brain (tumors, etc.). In atrophy of the brain it fills the space left vacant by the diminished volume of that organ, constituting the hydrocephalus *ex vacuo*. It occurs in enfeebled conditions of the system where there is arterial anæmia of the brain with venous congestion (the hydrocephaloids of Marshall Hall), and indeed wherever there is venous congestion of the brain from some impediment to the return of the blood, as in mechanical obstruction.

Again, intracranial effusion is found in conditions of the system which favor dropsical accumulations in other parts of the body, as after scarlet fever. And lastly, and most frequently (and these are the cases that most concern us at present), it is the result of inflammation of the pia mater and of the choroid plexus. In fact, whatever brings about intravascular pressure for a certain time may cause an intracranial effusion of serous fluid, although it seems necessary to assume in addition to such pressure some change in structure in the walls of the blood-vessels, since, from the numerous cases of cerebral hyperæmia that occur without effusion, we cannot look upon that factor as alone capable of producing it. As to how much a crippling of the normal power of absorption may conduce to the accumulation of the effused fluid, we know little or nothing.

It will be well for us now to consider what are the conditions in childhood which allow effusion from the intracranial vessels to take place so much more readily than in adult life. In the first place, we see that the cerebral circulation in young children takes place under circumstances favorable to the distention of the vessels under increased blood-pressure. Before the fontanels are closed it is obvious that they, by yielding under pressure, furnish a ready means for the increase of the intracranial space, and thus there is failure of counter-pressure and support to the distended vessels, allowing a further stretching of their walls. And even after the closure of the fontanels, the cranial bones of children, with their active nutritive changes consequent upon rapid growth, yield readily to pressure from within, so that they with comparative rapidity adapt themselves, by increased superficial extent, to the demand of the cranial contents for greater space. We may suppose also that the more succulent brain-substance of children allows of a ready absorption of its fluids before the advancing pressure of the distending blood-vessels, thus yielding them less support than in the adult brain. But probably the most potent factors in producing effusion within the cranial cavity of the child are the great thinness and distensibility of the vessels of the brain, and the readiness with which their structure becomes altered under abnormal blood-pressure. Moreover, when we consider the rapid development of the infant brain, we must suppose that there is a normal condition of the walls of its vessels permitting a correspondingly abundant transudation of the fluid parts of the blood. The fact, however, remains that effusion in abnormal amount takes place with great readiness from the vessels of the child's brain. It occurs probably more frequently than is generally supposed, but the more active absorption that goes on at that period of life restores the equilibrium before it is fully recognized.

It is usual to distinguish two forms of hydrocephalus,—acute and chronic.

ACUTE HYDROCEPHALUS: LEPTO-MENINGITIS INFANTUM.

Acute non-tubercular hydrocephalus is the result of an inflammatory condition of the pia mater running a more or less acute course, and terminating in effusion into the ventricles. We do not here, as in tubercular meningitis, have a specific exciting cause of the inflammation.

Meningitis, or, indeed, arterial hyperæmia however caused, if it continue a sufficient time, may terminate in such ventricular effusion, although it may be impossible to distinguish the moment when simple over-distention of the vessels assumes the conditions of an inflammation with abnormal effusion. As has been already said, we cannot suppose that simple active hyperæmia, the rapid passage of healthy blood through healthy vessels, even under high pressure, can cause an effusion which distends the ventricles, nor can it account for the failure of absorption which allows the fluid to remain there. We must assume some structural alteration in the vascular walls produced by an inflammatory process, which allows the fluid part of the blood to pass through them with abnormal facility while at the same time the process of absorption is crippled. The stoppage of the communications between the fourth ventricle and the subarachnoid space by lymph may assist in preventing the escape of fluid from the ventricles.

Lepto-meningitis giving rise to ventricular effusion occurs most frequently in children between the ages of one and five years. It is frequently seen in connection with febrile states (eruptive fevers, pneumonia, bronchitis) or with depressed conditions of the system, as after exhausting diarrhœa or whooping-cough, or when the system is reduced by defective nutrition, as in teething. In fact, any depression of the general health and strength conduces to it. Thus it is more common among the ill-conditioned and imperfectly-nourished children of the poor. It is obvious, moreover, that a lepto-meningitis may be the result of the extension of a neighboring inflammation, as is so often seen in meningitis following otitis interna.

The invasion of lepto-meningitis is generally abrupt. If the child is not suffering from some general disease, there may be only feverishness and a general disturbance of the health. On the other hand, there may be restlessness, symptoms of pain in the head, vomiting, wakefulness, or, again, sluggishness,—in fact, the symptoms generally attributed to cerebral hyperæmia. These brain-symptoms may disappear after a short time, and after an interval again return, to recede again, or finally to merge into the condition of pronounced lepto-meningitis; or a sudden convulsion may usher in an attack of the disease.

The symptoms of lepto-meningitis are those of acute cerebral hyperæmia, —restlessness, especially showing itself in uneasy movements of the head, which is rolled from side to side, sleeplessness, irritability, fretting, which shows itself in facial expression, headache, complained of in older children, corrugated brows, avoidance of bright light with a grimace. A flush confined to one cheek is often seen. Vomiting is a frequent symptom, and it is sometimes very persistent. The bowels, as a rule, are constipated. The pupils are generally contracted, and there may be occasional strabismus, which must not be confounded with the occasional crossing of the eyes seen in very young infants. The pulse, in my experience, is, as a rule, slower than natural, though this may alternate with a pulse of great rapidity. The Cheyne-Stokes respiration is very frequently seen, and, when marked, is a very significant symptom that the brain is implicated. At first the fontanel may be felt to pulsate strongly; at a later stage it is protuberant, and unyielding to pressure to a marked degree, but the pulsation has disappeared. Pressure upon it now causes uneasy movements in the little patient, though lying in a state of coma. A stiffness and tremor of one or more of the limbs is often marked, and is an important symptom, as indicating intracranial pressure. This stiffness may implicate the muscles of the spine and neck. As the ventricular effusion increases and more pressure is made upon the hemispheres, we may have eclamptic convulsions supervening. In one of these the child may die. In the advanced stage of the disease we may bring out the *tache méningitique*, which is done by drawing the finger-nail, the point of a pencil, or other blunt instrument, over the surface of the skin, the track of the instrument being immediately marked by a red line, resulting from the altered activity of the vaso-motor centres permitting an abnormal dilatation of the minute arteries. A state of coma and insensibility gradually comes on, and the child lies quiet, with only occasional movements of the face or limbs, which seem indications of pain, but are most probably merely reflex actions. This condition may continue for a long time, and there is often an appearance of improvement, which is very deceptive. This mitigation of symptoms probably results from the absorption of the interstitial fluid of the cerebral substance, thus temporarily relieving the nervous elements from pressure, or, in commonly-used terms, the brain accommodates itself to the pressure. And it may be that when the general intracranial pressure attains a certain degree the effusion into the ventricles is for a time restrained. The effusion into the ventricles, however, continues, and the final picture is that of profound insensibility, with a mere automatic continuance of the movements of respiration and the heart-beat. The face is placid and devoid of expression ; the limbs are generally flaccid, though sometimes rigid or contracted, often exhibiting great emaciation. The temperature of the surface and of the extremities keeps up until near the end, as does the strength of the pulse. At the last the heart begins to fail, and gradually ceases to beat. After the disease is established, and even after effusion into the ventricles has taken place, there may be an arrest of its progress towards a fatal termination, and recovery or chronic hydrocephalus may result. In many cases of recovery it would seem that the fluid in the ventricles is never wholly absorbed, giving an unnatural enlargement to the cranium in after-life.

The disease is very variable in duration, sometimes ending in a few days, and sometimes lasting for weeks, thus approaching the chronic condition. It is very important that the diagnosis be made between simple lepto-meningitis and tubercular meningitis, in view of the more favorable prognosis in the former disease. It is, however, difficult, and often is impossible. A careful investigation into the family history must be instituted, and a close examination of the patient, with the view of ascertaining if tuberculous deposits exist in any organ of the body. The more or less prolonged prodromal symptoms often seen in tubercular meningitis will give us an important clue to that disease, when they exist.

Pathological Anatomy.—The post-mortem appearances found in lepto-meningitis are, with the exception of the excess of fluid in the ventricles, somewhat negative. Evidence of the high degree of arterial hyperæmia which exists during life we do not find. This results partly from the compression of the brain and its membranes against the cranial walls, caused by the tension of the fluid in the ventricles, which obstructs the entrance of the blood into the minute vessels during the last moments of life, when the failing heart can no longer impel it with force, and partly because this tension exists after death (as is shown by the force with which the fluid spirts out of an opening made into the ventricles) and presses the blood out of the small vessels, leaving the brain blanched and apparently bloodless.

It is maintained by some that the post-mortem elastic pressure exerted upon the blood-vessels is contributed to by the absorption into the brain-substance after death of the fluid which, according to Hitzig, exists during life between the dura mater and pia mater, and thus the post-mortem volume of the brain is increased. While the vessels of the pia mater of the convexity of the brain present little or no indication of previous hyperæmia, those of the choroid plexus in the lateral ventricles are engorged, and sometimes present punctiform hemorrhages, evidences of their great distention during life, and of the prominent part they take in the production of the effusion. As a rule, neither upon the surface of the hemispheres nor on the pia of the base is there any evidence to the naked eye of pus, only the microscope shows that along the course of the vessels upon their borders in the perivascular space there is an accumulation of leucocytes that have made their way through the vascular walls. Sometimes the appearances are more indicative of the formation of pus. The fluid in the ventricles is albuminous, and rendered turbid to a greater or less degree by the leucocytes and pus-corpuscles it contains. The convolutions on the convexity of the hemispheres are flattened by the pressure from within, the sulci are narrowed and linear. The brain-substance is pale, and less succulent than normal. The rest of the body presents nothing noteworthy except the emaciation, which, if the disease has been long continued, may be very great, and is, in fact, somewhat characteristic of this affection.

Treatment.—In lepto-meningitis it is doubtful how far any treatment can be considered curative, but, inasmuch as a certain number of cases recover partially or completely, we should do all in our power to alleviate symptoms and place the patient in the best attitude, so to speak, to sustain the assaults of the disease. The great and well-known sympathy between the gastro-intestinal and the cerebral circulation inclines us to expect some result from acting on the former, and, in fact, benefit does seem to be derived from pretty freely opening the bowels in the early stage of the disease, and from relieving the obstinate constipation during its progress. For this purpose calomel appears to be one of the best drugs; but its continuance "in broken doses," with the view of obtaining its constitutional effects, I must deprecate as useless and tending to depress and weaken the patient. Iodide of potassium and iodide of sodium are remedies in constant use, and much relied on by some. To obtain any good effects from the iodides, they must be given in large and increasing doses, the amount to be limited only when they cause some disturbance of the stomach or of the system generally.

Local bloodletting, as by leeches to the mastoid processes or wet cups to the back of the neck, if resorted to in the onset of the disease affords relief to the symptoms of hyperæmia, although it is difficult to explain its action, which is probably through the vaso-motor nerves. Cold applied continuously to the head by means of the ice-bag has long received the sanction of most practitioners, and is always demanded by the laity. In many cases it is demanded of us to do something to calm the restlessness of the little patient, to ward off convulsions, or to mitigate their severity. Here I believe we have the most potent agents in the bromides and in hydrate of chloral. The bromides, moreover, probably tend to modify favorably the intracranial hyperæmia. They may be freely exhibited, care being taken to watch for the condition of bromism, which, if it is induced by the drug, may be confounded with the cerebral symptoms caused by the disease. Chloral used as a rectal injection is an excellent means for restraining convulsive attacks. In spite of the prejudice against the use of opiates in supposed congestion of the brain, there is no doubt of the advantage to be derived from them as a means of quieting great restlessness. A very important remedy is the warm bath frequently repeated while cold is applied to the head.

From derivatives, as blisters behind the ears, croton oil, etc., I have observed little or no advantage, while they almost certainly annoy and irritate the patient. Shaving the head and painting it with tincture of iodine, as is sometimes done, is as worthless as it is revoltingly disfiguring.

CHRONIC HYDROCEPHALUS.

Chronic hydrocephalus is the gradual accumulation of serous fluid within the cranium, sometimes occupying the space between the dura mater and pia mater (the so-called arachnoid cavity), sometimes the meshes of the pia mater (the so-called arachnoid space), but most frequently the cavities of the lateral ventricles, in which latter case it produces the most characteristic symptoms,—viz., enlargement of the head, alteration of the visage, etc. When the effused fluid is in the space between the dura mater and pia mater, or in the meshes of the pia mater, it is designated external hydrocephalus; when it is in the ventricles, it is called internal hydrocephalus. These forms may be combined, and the fluid be present between the dura mater and brain and also in the ventricles.

In the great majority of cases of chronic hydrocephalus, especially those resulting from ventricular effusion, the head is greatly increased in size, and shows abnormal conditions of the cranial bones and of the sutures. The hydrocephalic fluid may be identical with the cerebro-spinal fluid, or it may be turbid and contain albumen, sometimes to a high percentage. The presence of albumen beyond a very small amount furnishes the presumption that the effusion is the result of or accompanied with inflammatory action.

External Hydrocephalus may exist before birth (congenital), and, by increasing the size of the head, sometimes interferes with the delivery of the child. The enlargement of the head may go on after birth, but only exceptionally does it attain the dimensions commonly seen in internal hydrocephalus. In a few cases the effusion after reaching a certain amount has remained stationary, and the individual has lived to adult age.

It is difficult to reach a conclusion as to the causes of congenital external hydrocephalus in all cases. Sometimes it appears to be the sequel of an internal hydrocephalus, the fluid of which has broken through the walls of the distended ventricles into the subdural space. In such cases the brain, as would be expected, is found compressed and atrophied at the base of the cranium. Sometimes there is defective development of the brain, and fluid is found both within the ventricles and in the subdural space. Again, cases are recorded of a simple effusion in the subdural space, in which the brain was but moderately atrophied. It cannot be decided at present whether such cases are the result or not of an inflammation. External hydrocephalus occurs in children sometimes as the result of enfeebling diseases, as diarrhœa, rickets, congenital syphilis (?), etc. The amount of fluid in these cases is small, and its occurrence may be looked upon rather as a symptom of the general disease.

Another form of external hydrocephalus is much more frequent and its etiology better understood. It is the result of an inflammation of the dura mater, a pachymeningitis. As a result of the pachymeningitis, there is formed a sac of false membrane which is filled with fluid, sometimes serous, sometimes showing evidence of the inflammation which has accompanied its formation. In these cases, although the head does not attain the size usual in internal hydrocephalus, it presents the same general conditions. The sutures are widened, there is transparency of the cranial walls and fluctuation, and it is difficult, if not impossible, in some cases to make the diagnosis between it and internal hydrocephalus.

The form of external hydrocephalus which consists in the accumulation of fluid in the meshes of the pia mater demands but passing mention here. It is almost always a passive effusion filling up the space left by the shrunken brain,—a hydrocephalus *ex vacuo*. There is no sufficient evidence that an accumulation of serous fluid in the meshes of the pia mater ever causes injurious compression of the brain-substance.

CHRONIC INTERNAL HYDROCEPHALUS.—Accumulation of serous fluid in the ventricles is the form of hydrocephalus most commonly recognized. It may be congenital or acquired, the congenital form embracing the larger number of cases of hydrocephalus. The effusion may have advanced so far at the time of birth as to impede delivery and necessitate its evacuation, or, although a certain amount of fluid is present, the head may be of normal dimensions. After birth the accumulation goes on with varying degrees of rapidity in the first weeks or months of the child's life, and it may be so gradual as to render it impossible to say of a particular case whether the hydrocephalus is congenital or acquired.

Cases of internal hydrocephalus may be divided, according to their causation, into those in which there is simple accumulation of the cerebro-spinal fluid in the ventricles without inflammatory action, and those in which the effusion is the result of an inflammation of the ependyma of the ventricles and of the choroid plexuses. In these latter cases the accumulated fluid may indicate the inflammatory process to which it owes its origin, by a turbidity caused by the admixture of pus or blood, or, although the fluid is clear, by the increased amount of albumen it contains.

It is often impossible during the life of the patient to decide in which category the case must be placed, and even a post-mortem may not enable us to decide, since the disease may have a non-inflammatory beginning, but an inflammation may supervene which leaves more or less obvious traces. In cases of the first category (non-inflammatory) the cause of the accumulation is found most frequently in an abnormally diminished resistance of the cranial walls caused by malnutrition and imperfect development of the bones of the cranium. Under these conditions the blood-pressure within the vessels, not being sufficiently counteracted by counter-pressure from contiguous parts, we may conceive, causes an abnormal diffusion or filtration of the fluid part of the blood through the vascular walls. This will take place most readily at the choroid plexuses of the lateral ventricles, where it is supposed the blood-pressure can act to most advantage in the numerous convoluted vessels of those bodies. After the effusion has begun, every additional extension of the cranial walls serves to thin and weaken them and render them less able to resist the increasing accumulation of the fluid within; in other words, the cranial walls, from an abnormal condition, yield before the pressure which normally they are capable of sustaining. Under these conditions, if an additional intravascular strain is brought to bear upon the vessels, it is easy to see that effusion through their walls is readily and largely increased. Thus, in cases where the cranial bones present this abnormal condition of their nutrition, the convulsive expiratory efforts of whooping-cough, chronic bronchitis, or other lung-affections, which hinder the free return of blood from the brain, exercise a marked effect in increasing the hydrocephalic effusion. Here, as in acute hydrocephalus, the part that may be played by crippled and imperfect absorption cannot but suggest itself.

The disease which most frequently gives rise to what we may call the hydrocephalic conditions, which in fact may be considered the cause of hydrocephalus in many cases, is rachitis. We see in the head of the rachitic infant, with its prominent frontal and parietal protuberances and meagre visage, a strong resemblance to the hydrocephalic head, and it would seem that there may be sometimes a gradual merging, as it were, of the rachitic head into the hydrocephalic, without a sharp line of demarcation, the result of a gradual non-inflammatory accumulation of fluid in the ventricles. It is thought probable by some authors that the syphilitic taint may be the cause of non-inflammatory hydrocephalus, by injuriously affecting the osseous development and nutrition.

The second category—viz., chronic internal hydrocephalus of inflammatory origin—includes cases both congenital and acquired. As has been already said, it is extremely difficult to distinguish cases of chronic hydrocephalus which have an inflammatory origin from those which have not, and practically it is of little importance to do so. In congenital cases which are the result of inflammatory action it is not easy to give an adequate exciting cause for the inflammation. Some of the acquired cases, however, may be plainly traced to an acute lepto-meningitis which subsides leaving alterations which result in chronic internal hydrocephalus. Again, we see cases where the inflammation takes the chronic form from the beginning.

Pathological Anatomy.—The most obvious point in the pathological anatomy of hydrocephalus is the abnormal enlargement of the head, which is the consequence of the accumulation of fluid in the lateral ventricles. Only very exceptionally are hydrocephalic heads found smaller than normal. The size may reach enormous dimensions: cases in which the circumference reached fifty-two inches, and more, have been recorded. While the bones at the base of the cranium are generally less developed than normal, those constituting its vault and sides are greatly increased in superficial extent, and thinned, as it were stretched, to the extent of becoming diaphanous, yielding like parchment to pressure of the finger, or presenting unossified spaces within their circumference. Sometimes the flat bones of the cranium are made up of osseous islets connected together by membrane. Ossa triquetra are often developed. As a rare occurrence, however, the bones of the enlarged hydrocephalic head are greatly thickened. The fontanels and sutures are widely opened, causing the bones of the cranial vault to spread out from the base,—as Trousseau has expressed it, like the leaves of a flower. In cases of chronic hydrocephalus of inflammatory origin the pia mater at the base of the brain is often thickened and opaque, and the choroid plexuses show changes indicative of inflammatory action. The ependyma of the lateral ventricles, which in cases of hydrocephalus from defective resistance of the cranial walls is normal and transparent, in these inflammatory cases is thickened and granular, and may sometimes be stripped off in shreds. The hemispheres exhibit great alterations, while the cerebellum, pons, and medulla oblongata are very rarely affected, and are protected by the tentorium cerebelli from compression by the distended hemispheres. The lateral ventricles may be disturbed to a prodigious degree, their walls being thinned in proportion, so that we have the substance of the hemispheres represented only by a thin layer of nervous matter pressed against the vault and sides of the cranium, while the basal ganglia and peduncles are flattened out by the compression. The walls of the ventricles may be reduced to one-twelfth or one-twenty-fifth of an inch, or "no thicker than paper" (Dickinson), the convolutions and sulci being almost or quite obliterated, and sometimes it is impossible to distinguish the gray from the white matter. Such conditions must obviously produce structural changes in the nerve-substance, but of these we know little. The

Sylvian aqueduct may be widened and the hydrocephalic fluid found also in the distended fourth ventricle. The blindness so often seen is the effect of pressure upon the optic tracts, causing destructive changes in them, and white atrophy of the optic nerves. There may be found, besides, various defects of development of the brain, as absence of the fornix and corpus callosum.

Partial hydrocephalus—viz., when the excess of fluid is confined to one lateral ventricle, or even to one horn of the ventricle—is the result of local inflammatory action, and need not be considered here.

The hydrocephalic fluid varies, of course, with the size of the head, but also in proportion to the amount of compression and reduction in volume the brain has undergone. It may amount to many pints. It is sometimes clear and limpid, sometimes turbid, and sometimes it may be seen obviously to contain pus or to be tinged by extravasated blood. In the clear fluid of hydrocephalus from deficient resistance of the cranial walls (non-inflammatory hydrocephalus) there is found no more albumen than in the cerebrospinal fluid, which, in fact, it exactly resembles, but in those cases where inflammation has caused the effusion, or where an inflammation has supervened upon an originally non-inflammatory case, the specific gravity of the fluid is increased, and albumen is present in greater or less quantity. Huguenin says that if it is present in a greater proportion than two and a half per cent. it indicates an inflammatory action.

Symptoms.—The symptoms of ordinary chronic hydrocephalus stand in immediate and obvious relation to its pathological anatomy, as the enlarged head, the shrunken face, the staring eyes, the cerebral oppression and incompetence, and the nervous disorders. The head may reach such dimensions that it can no longer be supported by the muscles of the neck, and, as has been said, the body appears but an appendage to it. The distended fontanels and sutures together form an open membranous space between the cranial bones, that may reach between the separated halves of the frontal bones to the root of the nose. Percussion of the head elicits the sensation of fluctuation, and the thinned cranial walls and brain may allow a certain degree of translucence, as in hydrocele. The huge almost hairless head, marked with distended blue veins, contrasted with the small withered face, presents a striking and characteristic picture. The head is often covered with a profuse sweat. The orbital plates, depressed by the effusion downward to an obtuse angle with the body of the frontal bone, so alter the position of the eyes that the sclerotic is uncovered above, while the iris and pupil are half hidden by the lower lid, giving a ghastly stare from under the high arched eyebrows.

This position of the eyes is considered by some as pathognomonic of ventricular effusion in contradistinction to external hydrocephalus or cerebral hypertrophy, these latter conditions, it is believed, not causing the abnormal position of the orbital plates upon which the ocular phenomena depend. In some cases there is strabismus or nystagmus. Alterations in the fundus oculi have often been observed, and blindness or impairment of vision is common. It is obvious from the pathological anatomy of the disease that vision may be impaired in two ways, either by direct pressure on the optic tracts, or by the distention which the cortex undergoes impairing the functional activity of the visual centres. This will account for the fact that the alterations of the fundus are not always sufficient to account for the impairment of vision existing.

That the other special senses should be interfered with is most natural to suppose, and has been observed, but such investigations are exceedingly difficult in children. Hydrocephalic children are, with few exceptions, feeble-minded in various degrees, even to complete idiocy. This we would naturally expect from the stress of the disease being upon the brain, and it is, of course, in proportion to the damage the hemispheres have sustained that the intellect suffers. It is at the same time remarkable how tolerant the gray matter is of the distention and displacement, and how it continues to function even imperfectly under such altered conditions and impaired nutrition. In exceptional cases, where there has been an arrest of the disease, or where it has progressed very slowly, the brain may continue to develop, and the intellect, keeping pace, attain the average capacity. Cases have been recorded of even exceptional brightness.

The development of the body is retarded and imperfect; the limbs are small, wanting the plumpness and elastic tension of infancy, sometimes deformed. Not unfrequently there is tremor, with stiffness, or contraction, of one or more of the limbs, or rigidity of the neck and spine. Paralysis more or less extensive, monoplegia, hemiplegia, or paraplegia are not uncommonly among the symptoms. Convulsions, sometimes partial, sometimes general, frequently occur. The little patients often eat voraciously, although the digestion may be weak and vomiting frequent. In many cases of chronic hydrocephalus, when the observer's ear is applied to the head of the patient a sound or murmur synchronous with the ventricular systole, and of a blowing, cooing character, may be heard. Several explanations of it, none completely satisfactory, have been given. It would appear to be distinctive of chronic hydrocephalus, as opposed to cases of acute hydrocephalus with rapid effusion, and tension in the ventricles.

Prognosis.—The prognosis in chronic hydrocephalus is most unfavorable, especially when it is congenital, the child in such cases generally dying within the first few weeks after birth. Life may be prolonged, however, for months or years, while the disease gradually progresses, the head increasing in size, the senses becoming more and more impaired, the incoördination of movement and paralysis more marked, the convulsions more frequent, until death, sometimes suddenly occurring from an acute exacerbation of the disease, closes the scene. Cases, however, occur in which the effusion of fluid is arrested (by some process which we do not understand) suddenly or gradually, and the patient may attain adult age, or even, as has been recorded, old age. In some of these cases of arrest of the disease the thinning of the cranial bones gives place to great thickening from an abnormal osseous deposit. Cases have been recorded in which after closure of the sutures and fontanels a renewed development of the disease, probably of an inflammatory nature, has caused them to open again. Sometimes nature seems to make a conservative effort, as it were, and the fluid breaks its way externally through the orbit, the ear, or in most cases the nose. Some of these evacuations of the fluid have resulted in a cure. With the arrest of the effusion, however, we must not expect necessarily a further development of the brain, which may be more or less permanently damaged by the preceding condition. The large majority of hydrocephalics are idiotic, or exhibit but a childish intelligence, the few exceptions being those who reach an average mental capacity.

Diagnosis.—The form and size of the head of rachitic children at birth sometimes bear such a resemblance to the hydrocephalic head as to make a mistake in diagnosis possible. In those cases of hydrocephalus where the head is normal in size, the effused fluid having made place for itself at the expense of the brain, we have nothing but the symptoms on the part of the nervous system to guide us, and the diagnosis will be more or less difficult. Where there is marked enlargement of the head, the only affection with which hydrocephalus can be confounded is diffuse hypertrophy of the brain,—a very rare condition. Here, if the fontanels are open, fluctuation and a certain mobility of the cranial bones, or, with more certainty, cranial translucency, will be the distinguishing symptoms of hydrocephalus.

Treatment.—We cannot feel hopeful of the result of treatment in hydrocephalus. Internal remedies have been tried, with the view of promoting the absorption of the fluid, or of evacuating it by the emunctories of the body. Purgatives and diuretics of various kinds have been tried in vain, and iodine, iodide of potassium, and the mercurials, all have failed to restore the disturbed balance between effusion and absorption, or to influence favorably the diseased condition of the ventricular ependyma and choroid plexuses, on which the disease depends. Surgical aid has been called in with somewhat better success. It has been attempted to resist by mechanical compression the enlargement of the cranium, and so repress the effusion and promote absorption. The means employed is the careful and methodical application of strips of adhesive plaster over the entire cranium, caution being exercised lest the skin become excoriated, or, as has sometimes happened, gangrenous. It must be remembered that this treatment increases, at least at first, that compression of the brain which causes some of the worst symptoms of the disease, and again that there is a difference in tolerating it on the part of different patients. Although there has been great difference of opinion in estimating the result of this treatment, it must be admitted that many cases of improvement and some of cure have been recorded.

Evacuation of the fluid by means of puncture with the trocar or with the aspirator through the fontanel has been resorted to, but the fluid rapidly reaccumulates, even though external compression has been applied after the operation to the collapsed cranium. The more feasible plan seems to be to evacuate by repeated punctures small quantities of the fluid at a time, compression being carefully and continuously applied to the head. In some cases tincture of iodine has been injected into the distended ventricular cavities; but the results do not encourage us to repeat the procedure. The remarkable tolerance by the brain of such treatment is probably due to the thickened and altered condition of the ventricular ependyma, and its sluggish absorption. If cases of external hydrocephalus can be distinguished, they will from their nature present the most favorable opportunities for such injections.

Upon the whole, in the great majority of cases of chronic hydrocephalus, strict attention to the rules of health and diet, with the administration of cod-liver oil, and tonics when they are indicated, exhausts our resources, and sometimes these means effect much more than might have been *a priori* expected.

CEREBRAL PALSIES AND SUPPURATIVE MENINGITIS.

By LANDON CARTER GRAY, M.D.

CEREBRAL PALSIES OF CHILDHOOD.

Definition.—These palsies are of the nature of hemiplegia, double hemiplegia (or diplegia), or paraplegia, with spastic symptoms and cerebral defect, the atrophy of the paralyzed parts varying in degree, although usually slight, and not attended by any marked alterations in electrical reaction.

History.—Although Reil as far back as 1812, Cazauvielh in 1827, and Billard in 1828 had made some mention of the alterations in the infantile brain, it may be said that the first authors to describe porencephalic losses of cerebral substance were Breschet in 1831, Lallemand in 1834, Rokitansky in 1835, and Cruveilhier in 1849. But the first adequate description of cerebral palsies in children was given by Henoch in 1842, and this was followed by the excellent description of Little in 1853, by that of Turner in 1856, and by that of Von Heine in 1860. Heschl formulated the term "porencephaly" in 1859 to designate losses of cerebral substance. From that time to the present day the literature of the subject has been very abundant. Among the most notable of the earlier works is that of Leidesdorf and Stricker in 1865, narrating their experiments in the production of encephalitis in chickens, Tigges's on the production of encephalitis in rabbits, Virchow's in 1865 on congenital encephalitis of a peculiar nature, Cotard's *thèse de Paris* in 1868, Kundrat's great book on porencephaly in 1882, and Parrot's article in 1883 on steatosis. There is no doubt that Strümpell's article in 1884, describing his so-called polioencephalitis, reawakened interest in this subject and caused considerable addition to our knowledge of it. Among the later contributions the most valuable are those of Gandard, Jendrassík and Marie, McNutt, Wallenberg, Knapp, Audry, Gibney, and Gowers, whilst in the last two years Osler, of Baltimore, and Sachs and Peterson, of New York, have made valuable additions to our clinical knowledge of the subject. For other authors reference must be made to the appended bibliography.

Etiology.—The etiology is very obscure, although it is the custom to rank among the causes many attendant or preceding diseases, many circumstances of immediate environment, and many hereditary peculiarities. That these may all have some determining weight cannot, of course, be denied, but that they are the real causes of the disease cannot be affirmed, since they bear pretty much the same relationship to so many other diseases. Among such etiological factors have been mentioned marriages of consanguinity; syphilis and intemperance in the parents; difficult delivery; asphyxia of the new-born; cerebral traumata; infectious diseases, such as scarlet fever, diphtheria, pertussis, typhus, and variola; abnormal conditions of the mother during pregnancy; violent vomiting; defective nutrition.

Ages.—Of the one hundred and forty cases collected by Sachs and Peterson, one hundred and sixteen had their onset in the first three years of life, forty-nine being congenital. In one hundred and twenty cases of Osler's, one hundred and six began in the first three years of life, fifteen being congenital. The paraplegiæ are generally congenital, whilst the double hemiplegiæ (diplegiæ) usually commence as do the single hemiplegiæ.

Pathology and Pathological Anatomy.—Of the numerous diseases of the infantile brain those are well known that are due to a suppurative meningitis occurring from a distinct cause, cerebro-spinal meningitis of the sporadic or epidemic type, and cerebral meningitis of traumatic origin. Outside of these, however, the many pathological conditions affecting the fœtal and the infantile brain are involved in great obscurity. It is not strange that this should be so. Almost all these lesions are chronic, and autopsies are not had until a considerable time after the onset, so that the terminal conditions—the post-funereal ones, so to speak—are the only ones that can be studied, and a bewildering diversity of opinion necessarily obtains as to the exact nature and origin of the causative processes. Nor has the clinical expression of these intracranial processes thrown much light upon the subject. It must be remembered that the brain is a highly complex structure. In the child only gross impairment of motion, sight, and hearing can be observed, and even these only after a certain age. The ordinary forms of sensation, the group of symptoms included under the common name of aphasia, the so-called word-deafness, the fine muscular movements, the more delicate impairments of sight, such as hemianopsia, etc., the subjective sensations of vertigo, and the peculiar noises that are often of so much diagnostic importance in certain impairments of hearing, —all these are finer functions that cannot be tested in the child until it has attained nearly the intelligence of youth, and they are entirely beyond our recognition in the infant. Moreover, these old lesions that have begun in infancy or fœtal life do not cause the same localized impairment of function that they do in the adult brain. Hydrocephalus, hemorrhage, embolism, and thrombosis are described in this Cyclopædia by other authors. Aside from these, the following lesions have been observed in the brains of fœtuses and infants who had presented during life the clinical symptoms of the cerebral palsies:

Encephalitis and meningo-encephalitis;
The so-called polio-encephalitis;
The congenital encephalitis of Virchow;
Porencephaly;
Apoplexy of the new-born;
Thrombosis of the cerebral veins;
Atrophy and sclerosis.

Encephalitis and meningo-encephalitis generally begin in the pia and cortex, and proceed with cellular infiltration and sclerosis, minute hemorrhages, and eventually atrophy or, more rarely, hypertrophy of the affected convolutions. The large basal ganglia and even the spinal cord may be implicated simultaneously in this process, but this is to be distinguished from the secondary descending degeneration which may result from a lesion in the cortex. The onset of these processes has not been studied microscopically. The thick connective-tissue masses may vary in size from that of the cortical surface of the whole hemisphere to a mere cicatricial streak. In these sclerotic masses scanty remains of nerve-elements are found. The connective tissue may consist of a net-work, in the meshes of which are seen scanty openings for the vessels and granulated cells and nucleoli, or it may be very dense and the openings for the blood-vessels large and numerous. Because of a seeming relationship between the density of the connective tissue and the size of the vascular openings, as well as because Hayem has found the walls of the blood-vessels thickened, especially the adventitia, and because Marie has found these vascular walls in a condition of inflammatory infiltration with embryonic nuclei, it has been suggested that the vascular lesions were the initiative process; but there is nothing to show whether or not they are cause, effect, or part of a general underlying condition. In the hypertrophic form of sclerosis the cerebral substance is not infrequently dotted with ten or twelve masses varying in size from that of a bean to that of a ten-cent piece, round or streak-like, or of the consistence of india-rubber, principally in the cortex or in the central ganglia. From these masses the pia tears easily, but is adherent to the surrounding tissues, which are of a reddish color from a markedly vascular injection. These masses consist of thick connective tissue with spindle-cells, and pass into the normal tissue without sharp demarcation. A few atrophied ganglion-cells are found filled with pigment and granules.

In 1884, Strümpell advanced the theory that there was in children an acute infectious encephalitis of the motor convolutions, analogous to the acute myelitis of the anterior cornua of the spinal cord. This seems to me to have been one of the most flippant pathological suggestions ever made in medicine, advanced without the slightest proof on the part of its author; and as yet only one writer, Ranke, has adopted this view, and only one clinical observation, probably merely coincidental, not elucidated by an autopsy, has been cited in support of it. The avidity with which the suggestion has been seized for discussion and observation and the large literature which has grown up in refutation of it are pitiable illustrations of the paucity of our exact knowledge of the pathology of the fœtal and the infantile brain. Cotard and Gandard cite seven cases with the symptoms of Strümpell's so-called polio-encephalitis, in which the cortex was found to be normal, and in which the lesions were as follows: cyst in the corpus striatum, cyst in the frontal lobe beneath the cortex, sclerosis beneath one lateral ventricle, clot in the internal capsule, embolic softening of the internal capsule, embolic softening of the corpus striatum and surrounding tissue, hemorrhage into the thalamus opticus and corpus striatum. Besides this, Hóven records a cyst of the internal capsule, and in this case an especially careful examination was made of the cortex, which was found to be perfectly normal. Wallenberg also reports a cyst beneath the anterior corpora quadrigemina, implicating the crus cerebri and the lemniscus, and destroying completely one red nucleus. The title polio-encephalitis is the more unfortunate, as the same name has been very properly given by Wernicke to a thoroughly authenticated disease of the motor nuclei of the medulla oblongata, which are the true medullary analogues of the anterior cornua of the spinal cord.

In 1865, Virchow described what he termed congenital encephalitis, consisting of little yellowish masses in the white cerebral substance, and referable, he thought, to interstitial inflammation of the cerebral substance, the color coming from fatty granules. Although it has been maintained by Jastrowitz that fatty granules are physiological in fœtal brains, there yet seems reason to believe that Virchow's description applies to certain infrequent cases.

Heschl, in 1859, gave the name of porencephaly (πόρος, "a hole," and ἐγκέφαλον, "the brain") to certain exceedingly curious losses of cerebral substance in fœtal and infantile brains, varying in size from small cavities to an entire absence of both hemispheres. These cavities are generally full of liquid, and are traversed by filaments forming light and incomplete partitions. Substances resembling adipocere are sometimes found floating in them, or other substances of transparent, citron, yellowish, or brown color. These cavities may open into the arachnoid cavity, although they generally have a vascular membranous cover, the external face of which may be colored orange, yellow, or brownish. Ordinarily the pia is absent. Kundrat maintains that the convolutions bordering upon these cavities have a radiating form in the cases of pre-natal origin. The adjacent convolutions may be carpeted by a fine cerebral débris, or may undergo a gelatinous transformation. Sclerosis of the tissues bordering the cavity is very frequent, and these walls often have a rusty color, probably from hemorrhage. Near the lesion diminution or obstruction of an artery may be found. Instead of the cavities, however, there may be absolute loss of one hemisphere or both. The bones may be thinned or thickened, the skull may be hydrocephalic or microcephalic in shape, occasionally the front is very much flattened and slopes backward. Porencephaly is generally of fœtal origin, after this being most frequent in first infancy, occurring exceptionally afterwards. In fifty-seven cases analyzed by Audry, thirty-four were probably of fœtal origin, thirteen in the first two years of life, nine in the second infancy,—viz., three in the third year, one at three and one-third years, one at seven, one at nine and a half, one at ten, one at eleven, one at fourteen ; one case occurred in an adult. These singular losses of cerebral substance have been attributed to an arrest of development, extreme hydrocephalus, embolism or hemorrhage, encephalitis, and a profound cerebral anæmia. Kundrat endeavored to trace a constant relationship between vascular lesions and the porencephalic areas, but indubitable cases have been reported demonstrating that this view is not tenable. A peculiar gelatinous and cellular infiltration seems, in certain cases, to be one of the early stages of the process. The predisposing and exciting causes of the affection are but little known, although traumatism would seem to have been a distinct etiological factor in several instances.

The apoplexy of the new-born is regarded by Osler as one of the chief causes of the bilateral hemiplegia or paraplegia occurring at birth, and there can be no doubt that it is a very frequent condition in new-born children, especially, as Sarah J. McNutt has shown, in conjunction with abnormal labor, asphyxia, and convulsions.

Thrombosis of the cerebral veins is regarded as a frequent factor by Gowers.

It will thus be seen that the fœtal and the infantile brain behave in a different pathological manner from the adult brain. In the former we encounter much more frequently meningeal hemorrhages and acute forms of encephalitis and meningo-encephalitis, whilst the rapid losses of cerebral substance of porencephaly are practically unknown at later periods of life. Nor should we find it surprising that there is such easily effected retrogressive metamorphosis of the cerebral substance in the infant when we consider that the intracranial contents are the last portions of the nervous substance to develop and are very imperfectly developed during fœtal and infantile life, and that their great relative bulk, badly protected by the imperfectly-ossified cranium, renders them extremely liable to injury from without in the helpless fœtus and in the almost equally helpless infant. It is a singular fact, however, that most of the lesions are in the motor tract that extends from the motor convolutions to the muscles. The course of this motor tract is through the corona radiata to the internal capsule, thence through the pons to the region of the decussation, where it divides into two columns, the one going down upon the same side into the anterior pyramidal columns (columns of Türck), the other crossing to the opposite side to pass down through the lateral pyramidal column, thence into the anterior cornua, making direct connection with the great ganglion-cells from which arise the motor nerves that emerge along the anterior roots to terminate in the motor end-plates of the muscle. Although different convolutions are not infrequently the site of lesion, even as far back as the occipital lobe, the motor convolutions are pre-eminently affected. But lesions have also been found in the intracranial portion of this motor tract without affection of the convolutions: thus, as has already been stated, one of Gandard's cases had a clot in the internal capsule, with softening and ecchymosis of the Sylvian fossa, another had a focus of softening in the region of the internal capsule, and another a softening of the corpus striatum and the surrounding region; one of Wallenberg's cases had a hemorrhage in the right optic thalamus and corpus striatum, and another a cyst beneath the anterior corpora quadrigemina, implicating the pes and lemniscus and destroying fibres from the third pair and red nucleus; whilst Hóven's case had a cyst in the nucleus caudatus. These lesions act more disastrously upon the fœtal and the infantile brain than upon that of the adult, for the evolution of the former is seriously hampered. The condition of the spinal and peripheral portions of the motor tract has yet to be studied thoroughly, although in some cases of spastic paraplegia there is reason to believe that there is either a descending degeneration, or a lack of development of the intra-spinal motor strands. There can be no question that the motor tract is peculiarly liable to disease in children, and that the cerebral palsies are the earliest in point of time of the great series of lesions to which it is liable at this period of life. Dividing the motor tract into three portions, the intracranial, the spinal, and the peripheral, we may classify its lesions in this manner: cerebral, the cerebral palsies; spinal, myelitis of the anterior cornua (poliomyelitis anterior); peripheral, pseudo-hypertrophic paralysis; whilst progressive muscular atrophy is sometimes of spinal and sometimes of peripheral origin. The spinal and peripheral portions of the motor tract, however, are much more prone to chronic disease in children, almost the only exception being the acute onset of most cases of poliomyelitis anterior, whilst the intracranial portion of the motor tract is very liable to acute disease, the chronic lesions being rare. The cellular processes of the spinal lesions have been admirably studied, so that one of the most certain facts in pathology to-day is the disappearance or injury of the ganglion-cells of the anterior horn in poliomyelitis and in the spinal forms of progressive muscular atrophy. It is not as yet quite certain whether the muscular wasting of the peripheral forms of progressive muscular atrophy and the commingled wasting and hypertrophy of the pseudo-hypertrophies are due primarily to the affection of the muscle-cells, or are secondary to changes in the anterior cornua that have not thus far been detected by the means of microscopical preparation at present in use, although the evidence is as yet entirely in favor of the former view. But we are sadly in lack of knowledge as to the exact pathological nature of the lesions in the intracranial portion of the motor tract. Porencephaly, as we have seen, would seem to be a cellular process independent of preceding arterial impairment, inasmuch as Kundrat's opinion has been disproved that the porencephalic loss of substance is always in an arterial range. The fœtal and the infantile brain must be subject, therefore, to sudden cellular disintegrations, such as we see in the anterior horn of the spinal cord in poliomyelitis anterior, although on a much larger scale, and such as is not seen in the adult brain except in the rare cases of idiopathic softening that have been described by Wernicke. Arterial and capillary hemorrhage and arterial and venous thrombosis are also important factors, as we have seen, but we do not know what relationship they bear to the atrophies, the sclerosis, the porencephaly, or the cerebral porosity of Golgi.

Symptomatology.—In all cerebral diseases a sharp division must be made of the symptoms into those that are reflex and those that are direct or localizing. For instance, a hemorrhage may take place into the arm-centre of the cortex, at first causing unconsciousness or a convulsion, fever, headache, etc., and, when these have passed off, the paralysis of the arm may become apparent. The first class of symptoms are those which are due to shock of the whole cerebral substance from a sudden molecular change, whilst the latter are those which are due to actual destruction of a certain area of the brain-tissue. These considerations apply especially to the cerebral affections of children, who are more liable to reflex disturbance than adults.

The reflex symptoms, then, are convulsions, fever, delirium, hebetude or coma, and emesis.

The convulsions have nothing pathognomonic about them. They may be generalized, or they may implicate one member, the limbs on one side, or certain muscles alone. They may be tonic or clonic in character. It has been stated by a recent writer that loss of consciousness in a convulsion denotes a cortical lesion, whilst convulsion without loss of consciousness indicates a subcortical lesion. This rule may hold, to a certain extent, in adults, but not in children, who are not infrequently rendered unconscious by a poliomyelitic onset, or even by reflex causes.

The febrile movement is generally of a low type, rarely passing above 101° or 102° F. Not infrequently there is no history of fever whatsoever, so far as the statements of parents or relatives can be relied on.

Delirium, when present, is generally of a mild character, and of temporary duration, but it is often lacking.

Hebetude is generally present, and coma is sometimes observed, although both of these conditions, like fever, may be entirely absent.

Emesis is an infrequent symptom. There is generally an inability to take much food, but this is more the result of the hebetude or coma or the general malaise than of any nausea.

The localizing symptoms vary according to the portion of the brain that is affected. As yet, as has been stated, most of the cases have seemed to have a preponderance of motor symptoms, pointing to a lesion of the motor tract, but this seeming preponderance may be due to the considerations that have already been advanced. So far as we know them, however, the localizing symptoms are paralyses, contractures, exaggerated tendon reflexes, mental impairment, muscular wasting, mutism, speech-defects.

Classifying the cases by the motor symptoms, the paralyses may be arranged into the three following groups:

1. Hemiplegia;
2. Bilateral hemiplegia or diplegia;
3. Paraplegia.

In the hemiplegic cases the face is not always involved, and, when it is, the upper part of it is not affected. This form usually occurs in the first three years of life.

The bilateral hemiplegia or diplegia is usually congenital, from fœtal causes or brain-traumata in parturition. Gowers suggests for these cases the name of *birth-palsies.*

Paraplegia is usually congenital or begins in early infantile life.

In all these three forms contracture of the paralyzed muscles is usually present, the exceptions to this rule being infrequent. These contractures oppose a soft wax-like resistance to movements impressed upon the limb, yielding slowly, and gradually returning to their usual condition when the stretching hand is removed. The flexor muscles are usually more affected than the extensor. Not infrequently the small joints have a remarkable pliability, so that movements in any direction can be impressed upon the fingers, and they can be flexed, extended, abducted, and adducted with remarkable facility. If a limb is quickly and suddenly flexed, a lock-like movement will be felt in opposition momentarily by the hand of the person flexing. These contractures are the cause of various muscular deformities.

The tendon reflexes are generally exaggerated in the affected limbs, although only to a limited extent.

The mental impairment may vary greatly, from slight impairment to the most pronounced idiocy.

The muscular wasting is usually not active, as in poliomyelitis anterior, but would seem to be more from lack of development of the limb. Thus, in one little patient of mine the affected arm grows every year smaller relatively to its unaffected fellow, because the latter develops and the former does not.

Mutism and speech-defects are very common.

Sensation does not seem usually to be disturbed, at least in any marked degree; but of course it must be understood that it is simply impossible to test the sensations in infants and young children except in a very gross manner.

Vaso-motor disturbances are usual, such as lower temperature of the affected limb and defective vaso-motor circulation.

The electrical reactions are normal, except for some slight quantitative increase to faradism and occasionally to galvanism.

In the hemiplegic form Weir Mitchell has described, according to

Osler, post-hemiplegic tremor in one case affecting the entire arm, post-hemiplegic chorea (or hemiataxia) in twenty-four cases, mobile spasm and athetosis in six cases. In the bilateral hemiplegias so-called spastic chorea and double athetosis have been described.

In many of these unfortunate cases epilepsy occurs, there being sixty-two out of one hundred and forty cases collected by Sachs, or forty-four and three-tenths per cent. of all cases. Among the hemiplegias there were forty-one cases of general epilepsy,—nine of the Jacksonian type, and one case of petit mal,—in all about fifty per cent. In twenty-four cases of diplegia seven had general epilepsy (twenty-nine per cent.), one had Jacksonian epilepsy. In eleven cases of paraplegia four had general epilepsy (about thirty-six per cent.). This percentage of epilepsy differs but slightly from that given by Gandard, Wallenberg, and Osler.

Diagnosis.—The differential diagnosis is to be made from the following affections: cerebro-spinal meningitis of epidemic or sporadic origin; traumatic meningitis; suppurative meningitis; myelitis of the anterior cornua; transverse myelitis; tubercular meningitis; hydrocephalus; intracranial tumors; reflex hebetude, coma, or delirium.

In every instance a careful history should be obtained from some intelligent person who has been with the child at the onset of the disease, and the mother is almost invariably the best one for this purpose, unless some intelligent physician has recognized the disease early in its course. It is the lack of this precise history that makes the diagnosis of these cases so difficult.

Cerebro-spinal meningitis of epidemic or sporadic origin is characterized by retraction of the head, by the sporadic or epidemic prevalence of the disease, by its greater frequency in the years from one to fifteen, by a greater tendency to hebetude and coma, by a greater immunity from permanent mental impairment, and by the fact that the paralysis usually consists of one-sided and rarely of double hemiplegia or paraplegia.

Traumatic meningitis can only be diagnosed when the trauma has been known.

Suppurative meningitis can be recognized by the presence of suppuration in the ear, nasal cavities, orbit, or lung, or by some septic condition. Moreover, there is apt to be a peculiar remission in the symptoms, with fluctuating temperature, and chills or rigors.

Myelitis of the anterior horns is, as the name indicates, a sudden loss of the ganglion-cells in the anterior cornua of the spinal cord, and is characterized by a flaccid paralysis, with loss of tendon reflex, altered electrical reactions, and atrophy of certain muscular groups, usually in one limb. It is therefore monoplegic in its distribution, very seldom hemiplegic, and never doubly hemiplegic in the child. Cases that are characterized at the outset by convulsions, hebetude, coma, and fever, may cause some doubt about the diagnosis for three or four days or even for a week, but the distribution of the paralysis will settle the question.

Transverse myelitis is a rare affection in the child, but when it occurs it causes paraplegia, with vesical and rectal paralysis, perhaps bed-sore, whilst the upper extremities are usually unaffected, and there is no mental defect whatever.

Tubercular meningitis will be recognized by the chronicity of the cerebral symptoms, an acute onset being rare, and by the history of tuberculosis in other organs of the patient or in the family.

Hydrocephalus is so often associated with tubercular meningitis that the remarks just made will apply, except that the peculiar-shaped head is of itself almost diagnostic.

The differential diagnosis from intracranial tumor is often extremely difficult to make. In the latter, however, the tendency is to chronicity, to a greater localization of the symptoms, and to a greater frequency of neuro-retinitis and purposeless reflex so-called cerebral vomiting.

Hebetude or coma will not infrequently occur in children, seemingly of the most alarming nature, but yet will disappear in a day or two, being apparently a reflex of some indigestion or vaso-motor condition. Then, again, the acute febrile affections of children, such as the pulmonary, enteric, and miasmatic diseases and the exanthemata, will produce grave conditions of hebetude, coma, or delirium, and it will often be a matter of great nicety to determine how much is reflex from the primary disease, and how much may be due to an additional implication of the cerebrum or its membranes.

Prognosis.—The prognosis of these affections will depend, of course, upon the extent of the cerebral lesion, and the extent of the cerebral lesion will manifest itself in the paralysis of motion, in the contractures, in the exaggerated tendon reflexes, and in the intellectual changes. Every child, therefore, should be carefully examined, to ascertain the amount of damage done, as evidenced by these different symptoms. The paralyzed limbs should be tested in regard to their motion and sensation. The eye and the ear should be examined. The amount of contracture should be ascertained, and the tendon reflexes should be interrogated. The intelligence of the child should also be tested, and the seeming stupidity resulting from lack of mental training and from the isolated life of a paralytic, who has perhaps been petted and spoiled, should not be mistaken for the mental dulness caused by impairment or destruction of cerebral substance.

Treatment.—There is usually but little to be done in the treatment of these cases. In the cases of hemiplegia, especially those that come under treatment soon after onset, the faradic current is undoubtedly the most efficacious agent, and in several instances I have seen its use cause almost incredible improvement. In bilateral hemiplegia I have not seen anything like as good results, and in paraplegia it has been perfectly useless in my hands. Except in the aforesaid cases of hemiplegia of recent origin, I have not seen that massage is of any special use; but Osler speaks of persistent massage, with strong flexion and extension of the limbs, according to the recommendation of Weir Mitchell, as having been of great service. The spastic defects are usually beyond relief except by the apparatus and knife of the orthopædic surgeon. The mental defects, in proper cases, may be greatly helped by careful and systematic education. Operative interference with the brain in these cases is absurd, not only because of the impossibility in many instances of localizing the lesion, but also because in the vast majority of cases the lesion cannot be cured or aided in any way by surgical meddling.

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CEREBRAL MENINGITIS AND SUPPURATIVE MENINGITIS.

Different forms of cerebral meningitis have been considered in connection with hydrocephalus, tubercular meningitis, pachymeningitis, and the causative lesions of the cerebral palsies. Besides these the only meningitis of which we have any practical knowledge in children is suppurative meningitis.

Etiology.—Insolation, exposure to cold, infection, ear-disease, cerebral traumata, nasal disease, carbuncle, erysipelas, extension from other viscera, and purulent pleurisy are the best-known causes.

Insolation and exposure to cold have been known in some rare cases to set up purulent meningitis.

It is believed by some of the German authors that these cases may be infectious; but the proof is not very conclusive.

Of all the causes of suppurative meningitis, ear-disease is probably the most frequent. There are many intercommunications between the ear and the petrous portion of the temporal bone, by means of arteries, veins, and connective tissue, especially, as Seeligmüller suggests, by means of the connective tissue passing from the dura through the petro-squamous fissure to the mucous membrane of the tympanum and the mastoid cells and containing large branches of the middle meningeal vein and artery. The facial nerve is also an excellent highway for suppurative affections between the ear and the base of the cerebrum, as it is only separated by a very thin lamina of bone from the tympanic cavity, so that suppurative disease in the latter may affect the nerve in both its peripheral and its central tract.

Cerebral traumata may set up suppurative meningitis, and I am inclined to believe that this is a more frequent cause than is generally supposed. I have in many instances elicited a history of cerebral traumata that has been entirely overlooked by the family.

Any alteration in the nasal bones may extend to the brain through the cribriform plate of the ethmoid, and it is possibly in this way that the cases of meningitis arise that follow pertussis.

Suppurative meningitis has been known to follow erysipelas and carbuncle.

By extension from other viscera or passage of the infection through the lymphatics or blood-vessels are to be explained the cases of suppurative meningitis following croupous pneumonia, suppurative pleurisy, ulcerative endocarditis, pyæmia, septicæmia, acute articular rheumatism, small-pox, scarlatina, dysentery, and typhus.

Pathological Anatomy.—In the infectious cases meningitis is more of the vertex, whilst in those from aural and nasal disease it is mainly along the base and lateral aspects of the brain. In the slighter degrees the pia may be found to be congested, with slight streaks of purulent matter dotting it, but in a more marked degree there is a thorough infiltration of greater or less extent matting down the pia to the cortex, and varying in color from a cloudy yellow to a greenish yellow. Beneath these places of infiltration the convolutions will be found repressed, flattened, and ischæmic. The streaks of infiltration are greatest along the course of the blood-vessels. The infiltration generally passes along the choroid plexus into the ventricles, in which the amount of cerebro-spinal fluid is usually increased. In the chronic cases the membranes are tough, thick, adherent to one another and to the cortex, generally leading to atrophy of the latter. By microscopical examination various micro-organisms can be found in these cases, and these can be furthermore examined by cultures and inoculations, and by inoculations without culture. Netter has recently collected twenty-five cases of his own and forty-five observations of other authors. In his own cases six species of micro-organisms were found,—in eighteen pneumococci, in four a streptococcus pyogenes, in two a microbe resembling the intracellular diplococcus of Weichselbaum, in one a short bacillus of great mobility presenting most of the characteristics of the typhoid bacillus, in one a microbe very much like the pneumo-bacillus of Friedländer, and in one certain unknown bacilli that were very delicate and flexible. The observations in forty-five cases collected by Netter of other observers tallied in the main with his own. Netter is also inclined to believe that there is a difference in the exudation according to the micro-organism causing it. He states that the exudation in which the pneumococci are found is almost always very viscous and greenish, and that the meningitis frequently coincides with ulcerative endocarditis, although it would seem that the affection is relatively a benign one. In the cases in which the streptococcus was found the exudation was less adherent and was of a sero-purulent nature, while in the cases containing the bacillus of Friedländer the exudation was remarkably viscous and thick.

Symptomatology.—The symptoms of suppurative meningitis are delirium, hebetude or coma, fever, convulsions, muscular twitchings, headache, paralysis, and optic neuritis.

Delirium, hebetude or coma, muscular twitchings, and convulsions are general symptoms which are found in many cerebral affections, and have nothing pathognomonic about them in this special form of meningitis.

The fever generally ranges between 39.5° and 40° C.

Headache is usually a constant symptom, generally intermitting to some extent.

Paralysis is, as a rule, in the form of hemiplegia, although, of course, this may vary.

Optic neuritis is a symptom of considerable value when it is present, but it is not generally observed until the case has lasted for some time, and it is often absent. It indicates, however, simply an intracranial lesion, and tells nothing of the character of the lesion.

Retraction of the head is generally observed, and there may even be opisthotonus.

Lesser degrees of suppurative meningitis, especially in cases of ear-trouble, may commence insidiously with slight headache, sleeplessness, and heaviness, and pass gradually into the more pronounced symptoms of the disease. In other cases headache, some mental confusion, and dizziness may occur in children with suppurative ear-trouble, last for several days, and be entirely relieved by the discharge externally of pus from the diseased ear.

Diagnosis.—The diagnosis of a suppurative meningitis cannot be made by the cerebral symptoms alone, but must always depend upon the cerebral symptoms and the presence of those causes which are most likely to give rise to this variety of meningitis. When symptoms of meningitis follow trauma or insolation in a child, it is probable that the meningitis is of a suppurative variety. Careful examination should always be made, in any doubtful case, of the ears, the nostrils, the heart, and the lungs, and inquiry should also be made as to whether the child has had a recent pertussis, croupous pneumonia, purulent pleurisy, ulcerative endocarditis, pyæmia, acute articular rheumatism, small-pox, scarlatina, dysentery, or typhus. The differential diagnosis is to be made from cerebro-spinal meningitis, typhoid fever, and tubercular meningitis.

From cerebro-spinal meningitis the differentiation is usually made by the known epidemic or sporadic spread of the latter disease.

From tubercular meningitis the differentiation can be made by the absence of a tuberculous history in the child or in the family.

From typhoid fever the diagnosis may be sometimes extremely difficult, unless the cutaneous or enteric symptoms are present, or until sufficient time has elapsed to observe the range of temperature.

Prognosis.—The prognosis of the uncomplicated cases of suppurative meningitis is usually fair, but it is extremely uncertain in those cases which follow middle-ear or nasal disease, or when it comes by metastasis from other viscera. In such cases not only is the danger to life great, but there is also a still greater danger of some cerebral or motor defect being left behind.

Treatment.—The treatment of suppurative meningitis will vary greatly, according to the cause. If there be disease of the ear or its appendages, any pent-up pus should be promptly removed by operative procedure. If there be caries of the nasal bones that can be removed, this removal should be done promptly; but even then the treatment of the meningitis itself is a matter of great importance.

The fluid extract of ergot is an invaluable remedy, and should be given in doses of from five minims to a drachm every three or four hours.

If the meningitis is brought under treatment at an early period, a few large doses of quinine will often prove of service,—from two to ten grains, according to the age of the child. But I have never been able to satisfy myself that quinine was of any service after the early stages, except as a tonic.

The sulphide of calcium has sometimes seemed to me to be of use, in doses of from one-twentieth to one-tenth of a grain every hour for a day or two.

Cold applications to the head will give relief to the child and calm the restlessness, which latter may also be relieved, if necessary, by occasional doses of the bromides. Inunctions of mercury, usually of the unguentum hydrargyri, are time-honored, although I have never satisfied myself that they were of any especial use.

CEREBRAL ABSCESS.

Definition.—Cerebral abscess is the result of suppurative encephalitis.

Etiology.—The causes of abscesses are traumata, caries of the cranial bones, ear-disease, nasal disease, intracranial tumors, extension from other organs; and they may occur without known cause.

Traumata are insidious causes of cerebral abscess, because the abscess not infrequently follows the trauma after some length of time, and without the intervention of marked symptoms.

Caries of the cranial bones, especially of the petrous portion of the temporal bone, is a frequent cause, more especially in conjunction with ear-disease.

Nasal caries may cause abscess, and nasal polypi have penetrated the cranial cavity and caused disease of the frontal lobe.

When intracranial tumors cause abscess, the latter is always in the immediate neighborhood.

Cerebral abscess has been observed in conjunction with purulent lesions of other organs, such as bronchitis, pulmonary gangrene, empyema, ulcerative endocarditis, puerperal fever, typhus, variola, scarlatina, measles.

Symptomatology.—The symptoms of cerebral abscess are usually at first very vague. The child may complain and be fretful, manifesting a decided malaise, or there may be positive headache, sometimes very marked. These vague symptoms usually give place to delirium, which is slight and intermittent, or they may be succeeded by hebetude and coma. The temperature is somewhat elevated, generally ranging from 99° to 101° F., seldom above this; but in many cases there is no rise of temperature at all. Convulsions generally occur. Headache is almost invariable, with vertigo. Finally, paralysis, hebetude, and coma appear. The symptoms, in a word, are such as to indicate the presence of some slowly but steadily progressive cerebral lesion, and the individual symptoms will vary according to the portion of the intracranial organ affected.

Pathology.—Cerebral abscess is the result of a localized encephalitis tending to the formation of pus. It may be with or without a limiting membrane. It may, of course, cause secondary changes in the surrounding cerebral tissue. It is generally simple, except in the pyæmic cases.

Diagnosis.—The diagnosis of a cerebral abscess is made by the gradual onset of the symptoms, the variability of them, the presence of aural or nasal disease, or the history of some one of the suppurative or infectious affections enumerated of which cerebral abscess may be a sequel. The differential diagnosis will be from meningitis or cerebral tumor.

Tubercular meningitis is a chronic affection occurring in a child either personally afflicted with tuberculosis or hereditarily predisposed to it. Cerebro-spinal meningitis has not the insidious and gradual onset of a cerebral abscess, and in it the retraction of the head, the retracted abdomen, the *tache cérébrale*, and the known prevalence of the disease are factors that are absent in cerebral abscess. The diagnosis between cerebral abscess and suppurative meningitis is not always possible, as they are often present together and proceed from the same causes.

Cerebral tumors in the child are usually chronic, and in them there is often an optic neuritis, which is absent in the case of abscess.

In many instances it will be possible to make use of the cerebral thermometers to the use of which I called attention some twelve years ago. But it must be remembered that changes of from three to five degrees are necessary to indicate anything abnormal, and that the slight changes of axillary and rectal thermometry are useless.

Prognosis.—The prognosis of cerebral abscess is very grave. The tendency to recovery idiopathically is slight, and this should never be relied upon to the exclusion of an operation.

Treatment.—The treatment of a cerebral abscess should be by means of an operation as soon as the diagnosis is made. The localization of it is to be by anatomical data that are referred to elsewhere in this Cyclopædia. Although doubt has been expressed as to whether an abscess can be localized by localizing symptoms alone, I have made two successful diagnoses of abscess of the centrum ovale, and Von Bergmann has been still more fortunate in locating an abscess in the temporal lobe. Pain is seldom of localizing value, although in some instances, where the consciousness of the child is sufficiently retained, it may be found that percussion upon the skull will indicate the location by the pain that it gives; but I must confess that I should hesitate to follow this indication unless it coincided with other symptoms of localizing value. I have, however, known of this latter conjunction in several instances. Moreover, my clinical thermometers will often be found of great value in the localizing process. Operations have been done with great success for the relief of abscess, and every case should be operated upon. Von Bergmann's case of cerebral abscess was cured by an operation, although the patient had suffered for fifteen years from a purulent discharge from the right ear.

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MULTIPLE CEREBRO-SPINAL SCLEROSIS.

By WILLIAM BROADDUS PRITCHARD, M.D.

Synonymes.—Disseminated sclerosis, Insular sclerosis, Focal sclerosis, Charcot's disease; German, Herd-Sklerose; French, Sclérose en plaques disséminées-généralisées.

The sclerotic patches may be in the brain or in the cord or (as is usual) in both, giving origin to the unnecessary and cumbersome distinction of a cerebral, a spinal, and a cerebro-spinal type.

History.—To Cruveilhier[1] belongs the credit of having first called attention to the presence of pathological patches of sclerosed tissue disseminated here and there through the brain and spinal cord, though he does not appear to have regarded them as being diagnostic of any special symptom-group observed during life. Subsequently many facts of greater or less importance were added to our knowledge of the significance of these sclerotic patches or plaques by observers in both the French and the German school.[2] The disease, however, with which they were finally found to be essentially related as an integral part is peculiarly associated with the name of Charcot. It is to his genius that we owe the establishment of a symptom-group diagnostic of the affection. The fact that erratic or atypical cases have been observed in which some of the symptoms designated by Charcot as pathognomonic have been noted as absent, does not lessen in any appreciable degree the credit which must be conceded to him of having been the first (1864) to map out with any distinctness the clinical signs by which could be determined the presence of multiple sclerosis. Charcot, however, did not himself at first recognize the disease as existing before adult life, although in a later edition of his "Lessons" he calls attention to the fact, already noted by several observers, that the disease might occur in childhood or even in infancy. He quotes two examples of his own, as well as the now somewhat famous case of Hodemaker observed at Erb's clinic.[3] This case, that of a boy who developed the disease at seven years of age, dying at fourteen, is one of the earliest-recorded examples of the affection occurring in childhood.

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[1] Atlas de l'Anatomie.

[2] Carswell, Tuerck, Vulpian, and others.

[3] Deutsches Archiv für Klin. Med., 1879, Bd. xxiii.

Including the paper of Hodemaker, there have appeared up to the present time, so far as I have been able to determine from an extended though by no means exhaustive search through the literature of the subject, reports of more than fifty cases, ranging in age from fourteen months to fourteen and a half years,[1] published in the journals of France, Great Britain, and Germany. It is upon a *résumé* of the facts noted in this series of authentic cases that I shall base the following description of the disease as it occurs in childhood.[2]

Clinical History.—The disease may begin with symptoms strictly cerebral in origin, or they may be referable to the spinal cord only. Sooner or later, however, it becomes evident that the morbid process is not limited to either system, and, as a consequence, it is no longer considered either necessary or advisable to distinguish the two types. This is true of the disease as it occurs in adults, and the fact is still more conspicuous in children. The erratic tendency to select foci of degeneration here and there, at any part of the nervous axis, gives rise necessarily, and as might be expected, to much that is confusing in the symptomatology, and it may be safely stated that no single symptom has yet been observed which is pathognomonic. As described by Charcot, there are two modes of onset,—one slow and insidious, vertigo, headache, vague muscular weakness, with incoördination and tremor, being the symptoms first observed; in the other form, sudden in onset, the tremor, weakness, and ataxia date from a convulsion or apoplectiform seizure: subsequently, in both instances, ocular symptoms, such as third- and sixth-nerve paresis, optic-nerve atrophy, and nystagmus, defects of articulate speech, mental weakness, sensory disturbances, and contractures, occur, and the diagnosis is complete.

By far the most common mode of onset in children is the sudden or rapid form.[3] The child is noticed after a fall, a blow on the head, or shock from sudden fright, or perhaps without any apparent cause, to tremble. The tremor may succeed a convulsion, quite a common initial symptom of the disease in childhood. The gait is usually affected early also, the patient, if he has learned to walk, moving more clumsily or staggering. There may be coincidently a strabismus, or, if the child is old enough to notice it, a diplopia may be mentioned. Headache and vertiginous sensations are not infrequent, and would perhaps figure more prominently among the initial symptoms if the powers of observation and expression were less limited in early life. Nystagmus may be associated with tremor of the extremities as an early symptom, though, as a rule, it develops later in the disease. The same is true of speech-defects, unless there should be a primary focus of disease in the medulla. Mental weakness, contractures, muscular wasting, and actual paralysis of the extremities are late symptoms. Optic atrophy, not very frequently observed, may appear at any period, though usually at an advanced stage of the affection. The exaggeration of the reflexes, which depends upon the location of the lesion, is an early symptom in some cases, and may be associated with an ankle-clonus. The following cases are cited as typical in several respects of the disease as it affects children :

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[1] Pollok has published the results of an autopsy showing typical lesions which were congenital, Archiv für Psychiatrie und Nervenkrankheiten, Bd. xii. S. 157.

[2] This series includes, among others, nineteen cases tabulated by Unger ("Ueber Multip. enselformige Sklerose in Kind," 1887); Leube (one case), Deutsches Archiv für Klin. Med., 1870; Charcot (two cases), "Leçons sur les Maladies du Système Nerveux" (last edition); Stohr, Van Camp, and Rilliet and Barthez (one case each), included in Jaccoud's table: "Traité de Pathologie interne," vol. i. p. 207, 1877; Pelizaeus (one case), Archiv f. Psychiat. u. Nervenk., Bd. xvi. S. 698; Drummond (one case), London Lancet, 1887; Westphal (two cases), Charité Annalen, Bd. cxiii. S. 459, 1887; and twenty-one cases tabulated by Moncorvo, including four of his own, Jahrbuch f. Kinderheilk. u. Psychiat., Bd. xxviii. H. 2, 1888.

[3] The sudden onset may be relatively much less rare in the adult if, as Oppenheim has suggested, a large proportion of the adult cases date from childhood.

Case I.—(Reported by Wilson.)[1] Annie S. Parents healthy. Child born at full term; natural labor. Healthy until four months old, when it developed pertussis, lasting seven months. During pertussis the child had an attack of convulsions, followed by internal strabismus affecting the left eye, which remained three months. There was no return of convulsions, and the child made a complete recovery and remained well until one year old. At that time she was badly frightened by a dead goose thrown at her. The fright was followed by a convulsion, which left her quite feeble for some time. At the age of five years she was considered well and healthy. She attended school and learned readily, suffering from no illness until her seventh year, when she began to complain of dizzy attacks and diplopia. At this time the internal strabismus of the left eye was again noticed, though in six weeks, during which the child was under dispensary treatment, the strabismus disappeared and the general condition improved. Improvement continued about one month, when she developed a tremor of the lower limbs and began to lose control over them. Walking became more and more difficult, her legs giving way under her, the gait staggering like that of one intoxicated, until she finally lost all control over her lower limbs, becoming unable to walk at all in about twelve months. During this period she gradually lost flesh, and the tremors increased.

Her condition on examination at this time—decided symptoms of the disease having been present about one year and a half—was as follows: Blank expression; double internal strabismus, diplopia, slight dilatation of pupils; extreme emaciation. Sensibility intact, but almost complete loss of power in lower limbs, left more so than right. Can move toes and ankle freely, but cannot flex or extend the limb, nor can she raise it. Attempts to move the limb bring on rhythmical shaking. Hand-grasp good and equal. Tongue affected with undulatory tremor. On assuming a sitting posture in bed the body is violently jerked from side to side, and patient must be laid down at once. Horizontal nystagmus is present. Voice clear and distinct usually; at times speech slurred. Syllables are enunciated slowly. Replies intelligently to questions, and displays no abnormal emotional tendencies. Tremor of hands; steadies one hand with the other in eating or writing; handwriting characteristic. Temperature frequently subnormal.

Case II.—Thomas F., aged eight years and nine months (one of two brothers affected with the same disease, reported by Dreschfeld).[2] Father and mother healthy; two other children, aged two and four and a half years, healthy. Patient well until fourteen months old, when he had two convulsions, followed by trembling of the eyeballs and limbs and general weakness. After this sudden onset the case progressed steadily and without any remissions of noticeable duration up to the time of his examination at the age of eight years.

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[1] British Medical Journal, November 25, 1876.

[2] Medical Times and Gazette, February 9, 1878.

Condition on examination, the disease having been established seven years.—Body well nourished and limbs well developed, but cannot walk or stand. Attempts to move cause tremor of entire body. Face vacant, staring; lower lip large and dependent. Vision good, fundus normal, nystagmus constant, pupils normal. Intelligence markedly impaired, dull; speech slow and scanning, unable to pronounce certain letters,—*n*, *g*, *t*, *d*. Bulbar paralysis quite marked. Mobility of tongue much impaired, though not atrophied. Saliva constantly dribbling, and difficulty in swallowing. Uvula normal. When he drinks, the head shakes violently. There is no tremor of the head when at rest, though nystagmus is constant. Upper extremities paretic, and movements are accompanied with tremor. There is no atrophy, but the arms feel flabby. There are no contractures of the upper extremities, and there is no wrist-clonus. The lower extremities are much more paretic, the patient being unable to stand or walk. The feet are extended and begin to show contractures. Ankle-clonus is present, and knee-jerks are exaggerated. The functions of the bladder and rectum are normal. Sensibility is intact, and the electric reactions are unimpaired.

Case III.—Mrs. M., aged twenty-six.[1] Her grandfather died quite suddenly in bed. Father died in a fit, thirteen years ago, aged thirty-eight. He had fits (epileptic) for ten years previous to death, and was paralyzed during last five years of life. Paralysis occurred suddenly while patient was in bed, and was diplegic in distribution, involving both upper and both lower extremities. Muscles of face not affected, and speech normal. Does not remember as to presence or absence of rectal or vesical symptoms. The patient's mother died of Bright's disease. Both parents drank heavily. The patient is the oldest of eight children, five of whom are living. One child died in convulsions, aged two days, another from convulsions at the age of two months, and a third from spinal meningitis at the age of eight years. The other living children are free from nervous trouble. The patient had measles, scarlet fever, and pneumonia in early childhood, but was free from nervous trouble until eight years old. At that age she was kicked on the back of the head by a horse, being knocked down and rendered temporarily senseless. She does not remember her subsequent condition distinctly, but has been told that for some time after the injury she was unable to walk, and ever since the accident she has suffered from constant headache, dizziness, and trembling, and lately with weakness in walking. She never had a convulsion.

Condition on examination.—The woman is fairly well developed, and muscles well nourished and without wasting. There is no facial or cranial asymmetry, and no evidence of fracture, depressed or otherwise. Cicatrices of old suppurating glands of neck, and a very offensive nasal catarrh (ozæna?), suggest syphilis, though no other evidences are to be found and the patient denies personal infection. She has been married four years, but has no children. The expression is heavy and stupid, and patient represents a low type of intelligence, though she can read and write and is fairly intelligent in her replies to questions. Both pupils are dilated, responding very sluggishly to light, scarcely at all in accommodation to distance. There is no ptosis or strabismus, and nystagmus is absent, though the patient states that at times, under excitement, her eyeballs have been noticed to tremble. Vision is ordinarily good, though she is frequently annoyed by a blurring, amounting at times to a complete obliteration of a part of the field of vision. There is no hemianopsia, and ophthalmoscopic examination fails to show any atrophy or neuritis. There is present a coarse general tremor affecting all the extremities, the head, and the body. The tremor is somewhat more intense upon the right side. When the patient lies down the tremor disappears, but upon assuming the erect or sitting posture it begins at once, starting with lateral motions of the head and affecting the entire upper half of the body. There is no tremor of the legs except on extension, although in the hands and arms it is constantly present unless the arms are held by another. The tremor is greatly intensified by voluntary efforts, such as drinking water or eating, the teeth clattering at times against the glass or fork like castanets. She holds one hand with the other at times in performing finer co-ordinate movements, on account of the ataxic tremor which is present. The handwriting is characteristic. There are no bulbar symptoms, and speech is only slightly affected, the enunciation being slower than normal, but neither syllabic nor scanning. The most noticeable mental defect is of an amnesic character, the memory being decidedly poor and the attention concentrated with difficulty. In the lower extremities there is weakness, especially in the right leg, according to the patient's statement, and the right-hand grasp is perceptibly weaker than the left. There is no drag, nor is there any peculiarity in gait except a slight staggering or swaying. No Romberg. Both knee-jerks are markedly exaggerated, and about equally so. There is no ankle-clonus. There are no paræsthesiæ, and tactile, muscular, and pain sense are intact. Temperature sense not tested. Bladder and rectum normal.

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[1] Clinic for Mental and Nervous Diseases of Prof. L. C. Gray, Polyclinic, New York City, 1889.

The above histories have been selected from a large number as presenting symptom-groups typical of the disease to the degree of almost classical perfection, especially in Cases I. and II. The proportion, however, in which such a group of symptoms will be observed is relatively a small one: a much larger number will be encountered in which the diagnostic data are far less decisive. Many of the most striking symptoms will occasionally be absent, and it is necessary to note their relative constancy and importance. I shall, therefore, consider briefly each symptom in detail.

Tremor.—I am not able to recall an example of the disease in a child in which this symptom was noted as absent. Cases have been observed in man in which the sclerotic patches were dorsad of the pons, in which tremor did not occur, and Hammond[1] and others lay special stress upon this fact in contending for the recognition of a distinct spinal type. The tremor of disseminated sclerosis is peculiar, as follows: it is coarse in character, affecting one extremity, or, it may be, the entire body; it is intensified by voluntary muscular exertions; it ceases when the body or limb is at rest. The tremor may be unilateral,[2] bilateral, or general, the latter being the type in advanced cases, the tongue, head, and neck even being involved at times.

Ataxia.—This symptom is quite constant at all ages in certain forms. Station ataxia or Romberg's symptom is not common, but there is more or less marked incoördination in the movements of both upper and lower extremities. In the patient's gait it is at times almost identical with the ataxia of tabes dorsalis. In other cases the gait is simply staggering, like that of a man under the influence of alcohol, and in still a third form the patient walks with a mixture of ataxia and spastic rigidity. In the upper extremity the ataxia is first noted perhaps in a loss of fine movements; subsequently, an inability to carry food to the mouth (not due to actual paralysis or to tremor alone), or to control the hand in writing, may develop.

Eye-Symptoms.—Strabismus is frequently an early symptom. It may be only temporarily present, it may be present alone, or there may exist co-incident ptosis, diplopia, or paretic dilatation of the pupils. Strabismus is the most constantly observed of all the symptoms referable to the organs of vision in multiple sclerosis as seen in children. Ptosis[1] is much less frequently encountered, while dilatation of the pupils has been repeatedly observed. Diplopia occurs probably more frequently than is supposed, but because of its transient character and the early age of the patient it is more rarely mentioned. Optic atrophy was noted in less than ten per cent. of the cases tabulated by the writer. It is not stated to have been of the partial type referred to by Gnauck except in one or two instances. Nystagmus, which Charcot has observed in fifty per cent. of his cases of multiple sclerosis, is also quite common in children, the percentage being about the same. It is sometimes constant, in other instances it is only occasionally manifested under excitement, and in still others it is demonstrable only upon lateral or horizontal movements of the eyeballs.

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[1] Diseases of the Nervous System.

[2] Duckworth, Latham, London Lancet, May 16 and August 29, 1885.

Speech-Defects.—Some abnormality of speech is present in a very large proportion of cases (ninety per cent.). The defect may be due to tremor of the tongue, it may result from actual paralysis of the tongue or lips (bulbar origin), or it may be amnesic in character. As regards the form of the defect, it may consist in a simple indistinctness—a thickness or slurring—which is in contrast with previous healthy speech; there may be a difficulty in enunciation, the syllables of words being widely separated and tremulously spoken; finally, there may be a peculiar rhythmical inflection of sentences, constituting the typical scanning speech. It should be remembered, however, in weighing the diagnostic value of this symptom in early life, that the speech of childhood is often peculiar without being of pathological significance. It has no clinical importance alone, and unless it be typically scanning, or in marked contrast with previous relative perfection, it is of little value in early childhood.[2]

Mental Impairment.—A low standard of intelligence is the rule in disseminated sclerosis occurring in children. In some cases this is quite marked, amounting to absolute dementia; in others it is a simple silliness or an unduly emotional state. In a small proportion of cases the intellect remains unimpaired for many months or years, although very apt to be involved as the disease advances.

Headache: Vertigo.—Both of these conditions are comparatively infrequent, so far as can be determined by the records, though it is not improbable that they have been often overlooked. Vertigo was noted by Charcot in three-fourths of the cases observed by him. There is nothing characteristic of the headaches of multiple sclerosis as regards the locality or the intensity of the pain. Its most remarkable feature is the persistence with which it sometimes remains for years.[3]

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[1] Ptosis was present in Sparks's case (Medical Times and Gazette, December 29, 1877), and in Westphal's case, George Reinghold, nine years old. This case also showed optic neuritis and nystagmus among the eye-symptoms.

[2] Pollard's patient, a boy seven and a half years old, could speak no words distinctly, and had a very limited vocabulary. London Lancet, August 25, 1878, p. 183.

[3] Unger's case, three years; case quoted by writer, sixteen years.

Convulsions.—The frequency with which convulsive seizures inaugurate the disease in early life is rather remarkable. They were observed in more than half the cases recorded. In some instances the relationship (causal) of the convulsion is not as clear as could be desired, but in many cases other typical symptoms supervened immediately, leaving no room for doubt as to the intimate connection between the two events. The well-known susceptibility to eclampsia in infancy and childhood affords a partial general explanation of their presence in this disease, but the relationship is decidedly more than a mere coincidence in the majority of cases. The seizures may be epileptic, apoplectiform, or simply syncopal. The epileptic form is the most common. The convulsions may be followed by paralysis, localized or general, the paralytic symptoms passing away rapidly in some cases, in others remaining for some time. The residual paralysis is more frequently permanent in childhood than in adult life.

Paralysis.—This is most frequently of some cranial nerve, affecting oftenest the third, or motor oculi, in the early stages. Paralysis of the extremities is common in the advanced disease, though there is frequently a weakness which appears early, not genuinely paralytic. This may remain as the only form of motor weakness in a small number of cases.[1] Facial paralysis was observed in Schüle's case and others, and well-marked symptoms of bulbar paralysis involving the tongue, lips, and pharynx have been repeatedly noted (Dreschfeld, two cases, Schüle).[2] Muscular atrophy is rare, though it was present in one of Dickinson's cases and in Westphal's.[3]

Contractures.—These occur late, if at all, and are of no special diagnostic importance. The literature of the subject shows comparatively few cases of multiple sclerosis in children in which contractures were observed, but it is to be remembered that most of these histories are incomplete, the late stage of the disease escaping observation. The comparative frequency with which they develop in adult cases argues a similar condition in the same disease in early life. Both Charcot and Oppenheim[4] call attention to the fact that spastic paralysis should always excite a suspicion of multiple sclerosis.

Sensory Disturbances.—The paræsthesia of older cases is rarely observed. It is difficult to test the muscular sense in a child, and few observations are noted as regards its impairment. The most common disturbance of sensibility noted is a diminution of tactile perception. Sensation, as a rule, is not appreciably affected in children suffering from this disease, or, if it is, the difficulty and uncertainty attending the establishment of the defect are such as to make it of little value.

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[1] J. M., a boy four and two-thirds years old, reported by Dickinson (Medical Times and Gazette, February 2, 1878), could not walk on account of ataxia, but there was no paralysis.

[2] Schüle, Deutsches Archiv f. Klin. Med., 1871, Bd. viii. p. 223.

[3] Dickinson, Medical Times and Gazette, February 2, 1878; Westphal, Charité Annalen, 1887, Bd. cxiii. p. 454.

[4] Charcot, Jour. de Méd. et de Chir., 1887; Oppenheim, Berlin. Klin. Wochenschrift, 1887.

Reflexes.—An exaggerated patellar tendon reflex is the rule. It is often quite an early symptom. In a few cases the tendon reflex is much diminished or is even abolished, indicating a predominating sclerosis in the posterior columns. The superficial reflexes are exceptionally affected until the terminal stage is reached. It is sometimes a matter of difficulty to determine the condition of the reflexes in very young children.

Among other symptoms occasionally noted is deafness, which was observed by Drummond in a boy eight years old affected with the disease. This patient had also symptoms of diabetes, accounted for by the presence of a sclerotic patch in the medulla found post mortem.[1] Glycosuria has been repeatedly observed where this region was the site of sclerotic foci. Oppenheim claims to have found a new symptom "in a rapid exhaustion of muscular power in non-paralyzed parts,—viz., a first movement being made with full power, a second with less, and so on to complete inertia."[2]

Etiology.—Heredity plays an important *rôle* in the etiology of multiple sclerosis. Pelizaeus[3] observed the transmission of the disease directly through successive generations, the symptoms manifesting themselves in the male members of the family only, of whom five were affected. Several instances have been recorded of brothers and sisters who manifested unmistakable symptoms of disseminated sclerosis early in life. Dreschfeld published the histories of two brothers, in one of whom the initial symptoms developed at the age of fourteen months, in the other at four years; and both Erb and Frerichs have observed the presence of the disease in several members (brothers and sisters) of the same family, developing, however, in adult life. The presence of some ancestral taint other than the disease itself is much more frequently observed. Any neurosis in the parent is to be considered as rendering the offspring liable to the inheritance of a nervous system less resistant to disease than normally, and in this sense any neuropathy in the parent may be considered a predisposing cause of disseminated sclerosis. This statement is equally applicable to very many other affections of the nervous system, and why the ancestral taint should manifest itself in any particular case, in the development of multiple sclerosis, depends possibly upon some peculiarities of either vascular supply or anatomical conformation of the nervous axis. Among the most striking examples of conditions noted as predisposing through heredity to the disease are alcoholism in the father and syphilis in the mother, in the case of Dickinson; the father's death in a fit (chorea in a brother), in Cheadle's case; and epilepsy in an uncle (and a brother), alcoholism in the father, and migraine, with a highly nervous temperament, in the mother, observed in Unger's case. The proportion of cases, however, in which it is possible to determine the existence of an hereditary predisposition, either direct or indirect, does not amount probably to more than twenty or twenty-five per cent., and there is almost invariably an exciting cause which must be superadded.

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[1] London Lancet, 1887.

[2] Ibid., referred to by Seguin in Ann. of Univ. Med. Sci. for 1888.

[3] Ernest Engelman, eight years old, was the only child among the five affected. The next youngest patient was twenty-three years old. Archiv für Psychiatrie und Nervenkrankheiten, Bd. xvi. S. 698.

Among adults, cold, and especially prolonged exposure to damp cold, is recognized as a well-known etiological factor. I do not recall an instance in which the disease has been attributed to this cause in childhood, though it perhaps holds a certain responsibility.

Mental shock, such as sudden and intense fright, has inaugurated symptoms of disseminated sclerosis in several well-authenticated instances among adult examples of the disease.[1] Spitzka cites a case developing from such a cause, in a Bohemian cigar-maker;[2] and Wilson's case, in which the patient, a child one year old, was thrown into the initial convulsion from fright at having a dead goose hurled at her, is a probable example of the disease in an infant from this factor, though the typical symptoms did not appear until a few years later.

Traumatic injury or shock is a most important exciting cause of the affection. The injury may be congenital, and the symptoms of disseminated sclerosis may be present in association with evidences of arrest of development mentally and physically. The disease may originate from trauma or shock received later in life. Trauma is cited as the exciting cause in about fifteen per cent. of all the cases reported.

The development of multiple or disseminated sclerosis as a sequel to various acute diseases, especially of an infectious character, is quite noticeable in the type met with in childhood. Scarlet fever, measles, pertussis, diphtheria, and probably small-pox, typhoid and other fevers, stand in a peculiar and as yet unexplained etiological relationship to the disease. It seems highly probable, however, that the relationship is at most only an indirect one, since in those cases in which such diseases have been mentioned as present other factors existed which were undoubtedly of far greater importance in developing foci of nervous degeneration. The case of Willie R., for example, reported by Pollard, in which typical tremors were first noticed during convalescence from scarlatina, was much more probably due to damage set up by repeated convulsions which occurred during the attack of fever. The occurrence of other nervous diseases, however, characterized by degenerative changes during or immediately after the acute infectious fevers (poliomyelitis) gives credence to the idea that it is a possibility in multiple sclerosis, and cases have been cited in which no other cause could be assigned. As a unique example of the relationship of other diseases to the one in question may be cited the case of Sparks, in which tremors and weakness in walking, the first symptoms noted, developed immediately upon an attack of herpes zoster affecting the neck and head.

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[1] Focke, Inaugural Dissertation, Berlin.

[2] Pepper's System of Medicine, vol. v., art. "Disseminated Sclerosis."

As regards sex, individual experiences differ from general statistics. In the cases which I have tabulated, the sexes are about equally divided, with a small excess of males, while Charcot expressly states that the disease is more common in females, as do Ross[1] and Moncorvo, the latter finding twelve girls and nine boys respectively in a total of twenty-one cases. It must be remembered, in considering the value of Charcot's statement, that females largely predominate in the clinics at La Salpêtrière.

Pathological Anatomy.—The few autopsies made upon the bodies of children dying from multiple sclerosis revealed nothing radically at variance with the results of examinations in adult cases. In a few instances anomalous conditions and variations, dependent perhaps upon histological differences, are mentioned, but they do not alter the probable truth of the statement that the pathological conditions are practically the same at all ages. I shall confine myself, therefore, to a description of the morbid anatomy and pathology as it has been observed in the disease generally.

The distinctive characteristic of the affection, so far as its morbid anatomy is concerned, is one of location. To say that the lesion is a sclerotic degeneration does not distinguish it from any one of several diseases of the nervous centres. Sclerosis, identical in character with that which occurs in the disease we are considering, is always present in locomotor ataxia and in Friedreich's disease, and it has been repeatedly observed in general paralysis of the insane, as well as in other affections of the brain and cord. Indeed, the similarity is still more marked in general paralysis, the two diseases often being indistinguishable during life, autopsies showing patches of sclerosis disseminated here and there through the nervous tissues, besides other and more characteristic lesions of general paralysis, without which a diagnosis of the latter affection could not have been established.[2]

Sclerotic degeneration, therefore, while it is an essential post-mortem condition in multiple sclerosis, is not a peculiar or distinctive lesion as regards the process itself. It is only in the erratic and multiple distribution of these sclerotic foci that we have a condition which is pathognomonic. Any portion of the nervous axis may be attacked, and "even the roots and trunks of nerves have been found affected." Sometimes the patches or plaques are so numerous as to leave scarcely a single region uninvolved. Hirsch, for example, reported an autopsy in which "there were twenty spots on the left side, forty on the right, in the medullary substance of the hemispheres, with the pons, medulla oblongata, optic thalami, and much of the gray matter of the cord similarly affected; whilst in a case mentioned by Liouville both substances of the hemispheres, the corpora striata and optic thalami, cerebellum, crura cerebelli, corpora mammillaria, pons, all the right olivary body, the olfactory nerves, the optic, the motores oculi, the left fifth, the left facial, and the spinal nerves were all in a state of sclerosis."[1]

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[1] Diseases of the Nervous System, p. 11.

[2] Mickle on General Paralysis of the Insane, 2d ed., p. 231.

These spots or nodules, varying in size from microscopical smallness to the dimensions of a pea or a penny, are often so distinctly outlined as to appear clearly defined beneath the pia to the naked eye. Although exceptionally noted as almost normal in appearance, pigmentation is the rule, the color of the diseased tissue being a dirty gray or faint yellow, often presenting a marked contrast to the surrounding healthy structures. To the touch these spots of degeneration vary in the sensation produced, in some instances feeling hard and shot-like, in others simply tough and fibrous, though always harder than normal. On section a nodule is found to consist of "hypertrophied neuroglia, dense, fibrillated connective tissue of new formation, fatty and granular cells, and remnants of degenerated nerve-elements." This newly-formed connective tissue results from the proliferation of previously normal neuroglia nuclei, the cells becoming enlarged and giving off a multitude of fibrillar processes.[2] An excessive development of spider-cells is considered by most observers as a factor largely responsible for this change. The myelin is always affected, a rapid wasting, resulting in a complete loss of the axis-cylinder covering, having been frequently observed.[3] The axis-cylinder itself in the white substance, and the nerve-cells in the gray, are said to show a decided resistance to the morbid process, being affected only in the terminal stages, if at all. This, with the fact, noted by several observers, that the sclerotic foci are encountered in the white substance of brain and cord with much greater frequency, the gray being relatively exempt, is the only evidence of any selective affinity on the part of the morbid process for any particular region or system. In advanced cases, however, both substances are apt to be involved.

As regards the primary step in the formation of these patches of scattered sclerosis, there exists much difference of opinion among pathologists. The process is interstitial in origin according to the views of one school, by others it is considered parenchymatous; by some the disease is said to begin as a simple nutritional change, while others assert that the initial process is essentially inflammatory and attended with increased vascularity, the vessels becoming turgid and other evidences of inflammation being present. The vessels surrounding and traversing the diseased areas are certainly often, if not always, involved, the "adventitial sheath being thickened, the nuclei increased, and an actual sclerosis being often noticed." We have the authority of Rindfleisch for the statement that examination of the gray pinhead foci from which sclerosis starts will reveal the fact that the centre is always a red spot or line (according to the section), said spot or line being a distended blood-vessel; and that these vessels are in a state, under microscopical examination, which we should not hesitate to describe as that of chronic inflammation.[1] This is true also of the peri-vascular lymph-spaces, and it is here, probably, that the initial step in the development of the disease will be found in many cases. There are several factors which are responsible for the confusion and divergence of opinion existing among pathologists as regards this phase of the subject. The difference of opinion, dating from the controversy of Cohnheim and Virchow, as to the histological character of the neuroglia, and as to the possibility of the development of connective-tissue new-growth or hyperplasia in the nervous system except as a result of preceding inflammation, is still a factor in the disputed pathology of sclerosis.[2] It seems to be the opinion of the majority to-day that the neuroglia is truly a normal connective tissue; consequently we may say that sclerosis consists of neuroglia hyperplasia.

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[1] Fox, Pathological Anatomy of Nervous Centres, p. 119.

[2] Spitzka, Pepper's System of Medicine, vol. v., art. "Disseminated Sclerosis."

[3] Bourneville and Benedikt assert that the process is a "diffuse neuritis," and that the term "sclérose en plaques" is a misnomer.

With reference to the primary cause of this connective-tissue overgrowth, much of the difference in teaching is due, I believe, to a variance in the interpretation of the term inflammation. The traditional idea that inflammation necessarily requires a preceding injury, or direct and more or less violent exciting cause, is the Gibraltar preventing the acceptance of an inflammatory origin of the disease, with many observers. With such the process is said to start in a functional disturbance of nutrition, Van der Kolk teaching that sclerosis was the result of repeated congestions with excessive exosmosis through the vessel-walls. Bevan Lewis, whose exceedingly scientific and accurate researches into the microscopical anatomy of nervous diseases have done much to lift the veil of obscurity enveloping the subject, finds, in investigating the pathology of the various insanities in which sclerosis occurs, many facts which apparently confirm the theory of an inflammatory origin. In discussing the subject of miliary sclerosis in connection with the presence of that lesion in insanity, he states that in immediate proximity to these patches of miliary sclerosis the tissues are found to be in a state of parenchymatous degeneration, which results in the destruction of the essential elements, becoming a *genuine sclerosis.* He further suggests that the miliary degeneration is directly due to changes in the blood-vessels of a subacute inflammatory character, "the exudation from the vessels inducing such swelling of the myelin as to rupture the delicate investing albuminous sheath, or possibly acting directly upon the latter" (by pressure?).[3]

To sum up the subject, the evidence seems decidedly in favor of an essentially inflammatory (subacute?) origin for the sclerosis, with preceding or coexisting alterations of nutrition, dependent upon functional (vaso-motor) disturbances in the local vascular supply, such disturbances occurring as passive or active congestions, oft repeated. The sclerosis seems to be parenchymatous in some cases, interstitial in others.[1] The syphilitic cases develop primarily through the lymph-ducts, and are characteristically interstitial, and this form is considered by Moncorvo and others, among them Marie and Jendrassík, to be quite frequent. Syphilis itself, however, or the transmitted cachexia, is not a very common etiological factor in disseminated sclerosis, except in the experience of Moncorvo, Marie, and perhaps a few others, and it should not be accepted as a basis for pathological deductions unless absolutely demonstrated.

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[1] Rindfleisch, Pathology.

[2] For full discussion see Stricker's Histology.

[3] W. Bevan Lewis, Text-Book of Mental Diseases, p. 463.

Recurring to the subject of the locality affected in the disease, many facts have been observed which are of great interest and importance from a clinico-pathological stand-point. The symptoms manifested during life will often point with remarkable consistency to the exact or approximate location of the sclerotic patches. Thus, in Drummond's case the presence of persistent glycosuria with polyuria suggested the very probable involvement of the medulla, an hypothesis fully confirmed by the finding of a well-defined patch of sclerosis in the floor of the fourth ventricle. The nuclei of origin, or the root fasciculi, of the hypoglossal, the facial, and other nerves, have been invariably found to be foci of disease in cases in which paralysis of the parts supplied by such nerves had been observed. In the cord a spastic or pseudo-spastic gait indicates a focus of disease, usually in the lateral columns, which seem, by the way, peculiarly liable to invasion in multiple sclerosis. Decided trophic changes, such as wasting or atrophy (comparatively rare), point to an involvement of the anterior horns, while marked ataxia with paræsthesia or other disturbances of sensation suggests very emphatically an involvement of the postero-lateral columns. The deep reflexes are of value here, in that an exaggerated patellar tendon reflex is almost conclusive evidence of disease in the lateral or pyramidal tracts, an abolished reflex pointing with equal certainty to a predominating sclerosis in the posterior columns and root zones. The location of the diseased patch or patches in the cerebrum may be almost as readily determined, the symptoms being more or less distinctly diagnostic according to the region involved.

Differential Diagnosis.—The question of a differential diagnosis in multiple sclerosis as seen in patients of mature years is often one of extreme difficulty. The very insidious development, extending over years perhaps, and the absence in many instances of symptoms at one time considered essential, with the presence of others of an anomalous character, are factors adding much that is confusing. Tremor, for instance, the absence of which a few years ago was considered sufficient to refute a diagnosis of multiple sclerosis, has been demonstrated as wanting in several cases among adults in which typical post-mortem lesions were found. Nystagmus is by no means invariably found in the adult cases, and the same is true of characteristic speech-defects.

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[1] Spitzka, Text-Book of Mental Diseases, p. 463.

In children the symptom-group typical of the disease, as described clinically by Charcot, is present with apparently much greater uniformity, and, with the one exception of hereditary tabes, or Friedreich's ataxia, there is no definite affection of the nervous system in childhood which should present any difficulties in diagnosis. These two diseases are, however, in many respects remarkably similar symptomatically. In both there is ataxia, in both nystagmus, and in both speech-defects occur. It would be difficult to establish a diagnosis of either without the presence of one at least of these three symptoms. Tremor is also a common symptom, though differing in quality and degree, and paralytic affections of the eye-muscles, with optic-nerve atrophy, have been not infrequently observed in both. Among other symptoms mentioned as occurring in the two diseases, though with less frequency, are contractures and sensory disturbances.

So remarkable an identity in symptomatology argues an identical pathology, and the relationship is undoubtedly a close one in this respect. There are, however, certain well-defined clinical landmarks which clearly distinguish the one from the other. The tremor, for instance, is a constant symptom in multiple sclerosis in children, having been present without exception in every case reported. It is peculiar in type also, as I have explained in a preceding paragraph. In Friedreich's ataxia the tremor, which is present in only a small proportion of cases, is usually of the choreiform variety, and is never of the voluntary or intention type. Nystagmus in hereditary tabes is almost invariably ataxic, manifesting itself only when the eyes are steadily directed towards an object. In disseminated sclerosis the nystagmus is of the ordinary or static form, sometimes demonstrable, however, only by movements of the eyes in certain directions, as up or down (horizontal),[1] or laterally. Difficulties in articulate speech may be exactly similar in both diseases,—halting, with separation of syllables, simply indistinct, or typically scanning. In Friedreich's disease it is more apt to be ataxic, and occurs later, as a rule, than in multiple sclerosis. Ataxia of the extremities is decidedly less constant in the disease which forms the subject of this article, occurring in about sixty per cent. of the reported cases, while according to the statistics of Griffith it was present in either one form or another in nearly all of the one hundred and forty-three cases of Friedreich's disease which form the basis of his paper.[2] That form of ataxia which is manifested in an inability to stand steadily with the feet approximated and the eyes closed, and which is known as Romberg's symptom, is rarely observed in multiple sclerosis in children except when dependent upon actual or absolute loss of power in the lower extremities.

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[1] In Wilson's case, a girl, Anne S., aged eight, the nystagmus was always and only horizontal. British Medical Journal, November 25, 1876.

[2] American Journal of the Medical Sciences, October, 1888, p. 377.

It is said to have been present in more than fifty per cent. of the cases tabulated by Griffith. Finally, in multiple sclerosis the ataxia is a true incoördination, while the titubating or staggering gait is said to be the most characteristic form in the other affection.

Abnormal sensations, or paræsthesiæ, not infrequently occur in hereditary tabes, especially the girdle or belt sensation. They have not been observed in children affected with multiple sclerosis, though in older patients this symptom is not uncommon. Sensory impairment is likewise rare, while a diminution of either tactile or muscular sense has been repeatedly noted in children the subjects of Friedreich's ataxia. Paralytic affections of the eye-muscles are infrequent in the latter disease, while either a strabismus, a ptosis, or a diplopia is quite common in disseminated sclerosis. On the other hand, ophthalmoscopic examinations of the fundus show an optic neuritis or atrophy in probably an equal percentage of cases in both affections. Contractures are not common in either disease, though rather more frequent, perhaps, in hereditary ataxia.

In addition to the clinical distinctions already enumerated, there are at least three symptoms which afford a marked contrast in the two diseases, and these are the condition of the reflexes,—especially the patellar tendon,—the mental state, and the tendency to convulsive seizures respectively. As regards the first, in Friedreich's ataxia the knee-jerk is almost invariably abolished, though in rare instances (two per cent.) it may be normal or even exaggerated. In disseminated sclerosis the opposite is true, the knee-reflex being much more commonly exaggerated, rarely abolished. The mental faculties are, almost without exception, obtunded in various degrees at some stage of the disease in multiple sclerosis, usually in the form of a simple dementia. In Friedreich's ataxia the intellect is unimpaired, exceptional cases manifesting a slight mental weakness at most. Convulsions were noted in more than fifty per cent. of the cases of disseminated sclerosis in childhood in the literature of the subject to which I have had access. Though occasionally observed in hereditary tabes, the convulsions were very probably an accidental coincidence, and not essentially related to the morbid process.

Chorea may be readily eliminated in a diagnosis by the difference in the character of the tremor, and by the absence of nystagmus, of scanning speech, of ocular paralysis, and of true ataxia. In the so-called post-hemiplegic variety of chorea the symptoms may closely simulate those of multiple sclerosis, and a diagnosis then becomes a matter of more difficulty.[1] The history will aid, however, in determining the identity of the affection, and it is only rarely that such cases give rise to any confusion.

Paralysis agitans need only be referred to incidentally, since it is exceedingly rare to meet with it early in life, although Charcot has seen two cases aged respectively twelve and sixteen years.[1] The type and location of the tremor, the peculiar "bread-crumb" rolling position of the thumb and finger, the rheumatoid pains, the gait,—"propulsive and retrogressive" in character,—together with the absence of nystagmus, of ocular paralysis, and of ataxia, will readily decide the case to be one of paralysis agitans, and there should be little trouble in distinguishing them.

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[1] Osler, in his monograph, "The Cerebral Palsies of Children," pp. 66, 67, calls attention to the difficulty attending a differential diagnosis in some of these cases.

Brain-tumors or neoplastic growths anywhere in the nervous centres, especially if multiple, may give rise to symptoms absolutely identical with those of multiple sclerosis, and often in such cases a diagnosis will become impossible. Within the past few years Prof. L. C. Gray has observed the symptoms of a multiple sclerosis during life in cases which showed simply a lepto-meningitis at the autopsy.[2]

Prognosis.—It is an accepted fact that, spinal or cerebral nervous tissue having been destroyed by disease, it is not regenerated. It follows as corollary to this statement that a permanent recovery in a well-established case of disseminated sclerosis is not to be expected with our present means of combating the morbid process. In this respect the affection is the same at any age. The process of degeneration may, however, under favorable influences and treatment, occasionally be arrested, at least temporarily. This occurs at times spontaneously. Such arrests constitute the well-known periods of remission, periods in which the symptoms may remain quiescent for many months and even for years, though such remissions are not so apt to occur in cases developing in children, nor are they so complete or long in duration. An explanation of this statement is to be found in the fact that nervous structures in the child are peculiarly vulnerable because of their unstable and highly-vascular condition of development and growth, and the damage consequent upon a lesion is much more wide-spread because of the subsequent interference with development. Affections of the central nervous system in children therefore offer, broadly speaking, a less favorable prognosis than in the adult. Multiple sclerosis seems to be no exception to the rule, and it is in accordance with this view that we find the disease less amenable to treatment, both as regards the prolongation of life and the amelioration or arrest of the symptoms, even temporarily.

The disease provokes a condition of physical helplessness much more rapidly in children, and there is a consequent increase in the liability to complications, mechanical and trophic in origin, which characterize the bedridden stage of many nervous diseases. Statistics as to the duration of life after the disease has been recognized are not sufficiently numerous or reliable to allow of any positive deductions in this particular, but it is probable that the duration is much shorter. Death from disseminated sclerosis *per se* is not common at any age, but the general impairment of nutrition associated with or resulting from the bedridden state, or the supervention of bladder-troubles, or of bulbar paralysis, with consequent inability to take food, or of pulmonary congestions, may in any case give rise to a fatal termination. Convulsions are an added factor in the disease of the gravest import in prognosis, since a seizure may abruptly precipitate a fatal result.

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[1] Duchenne has reported a case in a boy of sixteen, and Meschede one in a patient twelve years old, also a boy.

[2] Medical Record, September 18, 1888.

The intelligence is almost invariably defective sooner or later in this disease at all ages, though with this difference, that the defect is in children more in the sphere of receptivity, and in adults of an amnesic character. The child's psychical centres never having been stored with impressions, or only partially so, his vacuity and dementia are more obtrusive; and this is true in degree the earlier the child is affected.

Treatment.—There is no known drug or combination of drugs which can be demonstrated as possessing a specific property in arresting the morbid process in multiple sclerosis. It may be still more positively stated that we are utterly powerless to restore the damage which has been done to the nervous axis or any portion of it with any therapeutic agent. In view of these facts, the treatment to be observed in any case of disseminated sclerosis must be largely experimental and symptomatic. I do not mean that it should be inferred that we are without any known resources which are of value in this affection. On the contrary, there are several drugs, besides other measures, which have in repeated instances produced an amelioration of the symptoms. Iron, ergot, potassium iodide, nitrate of silver, turpentine, arsenic, and mercury have all been persistently administered by many observers, and with apparent benefit in the case of some of the number.

Of these, iron seems to be of use solely in counteracting, by its tonic properties, the debility which occasionally is an accompaniment of the disease. Ergot, though widely used, I have never seen followed by evidences of improvement in adult cases. It seems to have crept into the therapeutics of multiple sclerosis through the claims of Brown-Séquard and other advocates that its use was followed by benefit in that kindred disease tabes dorsalis. It has become the fashion to suggest potassium iodide in any and all chronic affections of the nervous centres; nor is this suggestion in the line of empiricism, though it would be of undoubted advantage to outline more clearly its indications. In multiple sclerosis in children there are few data to show that its use has ever been followed by benefit which could be directly attributed to the drug. I do not care to assume the extreme position of condemning its use entirely, but it seems to me that only in those cases which afford some special indication in a syphilitic inheritance (a small percentage) should the drug be relied upon to the exclusion of other and more useful remedies. In such cases it is probable that the conjoint administration of the iodide with mercury, or the two alternately, would give better results than either alone. Nitrate of silver has long been in use, and at one time was held in high favor, in all forms of chronic degenerative or sclerotic disease of the spinal cord and brain. A consensus of opinion tends more and more to the belief, in the light of careful and scientific observation, that the beneficial effect following the use of silver in this disease was *post* and not *propter hoc.* The same may be said, in passing, of gold and barium chlorides, although neither of these drugs has had any extensive trial in the cases of multiple sclerosis which have occurred during childhood. Arsenic, from its almost specific action in chorea, a disease which in some important respects is known to resemble multiple sclerosis, and which it is not improbable will be found to be still more closely related to it, should, I believe, be tried faithfully in every case. The liquor potassii arsenitis (Fowler's solution) is the form most effectively administered.

Among the many other drugs which may be mentioned as having been tried, phosphorus, on physiological grounds, at one time held a position of some prominence. Its use has, however, been attended either by results negative in character, or, as in the cases of Bristowe, by an aggravation of the symptoms.[1] Cod-liver oil in the debility or emaciation sometimes present is indicated, and its use in conjunction with phosphate of iron was followed in one case (Wilson's) by decided benefit and a marked remission of symptoms.

With the German school of neurologists electricity in the form of galvanism is a routine measure of treatment. Following the teachings of Erb, small doses and short and infrequent applications are recommended. From three to five milliampères administered through large sponge electrodes, the negative over the cervical sympathetic ganglia, the positive passed slowly down the opposite side of the spine, on alternate days, is the method in use in Erb's clinic. The séances are of three minutes' duration. In the English clinics and in this country much stronger currents are used and the duration of the séance is longer. In the early stages of the disease the German method is advisable, except that the applications should be made daily or twice a day; but in cases which have passed through the initial stage, and in which the disease is established as a chronic organic affection, stronger currents (from five to ten or twelve milliampères) should be used and the séances should be longer. Very decided improvement follows in some cases. Humphrey's patient (Annie J. S.) was relieved of tremor for one month following the use of galvanism, though the symptoms all returned afterwards and remained uninfluenced by any treatment. The faradic current is serviceable in arresting the wasting of muscles which sometimes, though not often, occurs. In such cases mild currents from the secondary coil should be passed through the affected muscles daily.

Recognizing the absence of any knowledge of a specific for the disease, the treatment in many cases has been purely symptomatic, and even here the results have not been very encouraging.

For the relief of tremor, the symptom which is most annoying and harassing to the patient, rendering him helpless and dependent at times, many remedies have been suggested. A partial list includes conium, atropine, hyoscyamus (Charcot, Seguin) and its alkaloid, the bromides, arsenic, cimicifuga, strychnine (Hammond, Trousseau), zinc sulphate, physostigma, curare (Erlenmeyer), veratrum (Feris, Spitzka), and electricity.

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[1] Bristowe's case, female, aged thirteen, Medical Times and Gazette, June 21, 1879, p. 673.

Of these the most reliable is hyoscyamine, given in doses of from one two-hundredth to one one-hundredth of a grain twice or three times daily, the tremor disappearing within a few hours after its administration, and remaining absent for several days during the continuance of the drug. Conium seems objectionable, because of the depressing effect which follows its use, as well as from the fact that it only exceptionally modifies or controls the shaking. Atropine acts very similarly to its ally hyoscyamine, though the dryness of the mouth and the vertigo which result from its use are strong objections. Besides, it is not nearly so effective as hyoscyamine. Strychnine has been recommended by Trousseau, Hammond, and others; but Charcot states that it aggravates the tremors. No instance is recorded in which its use was followed by any benefit in the disease as observed in childhood. Physostigma, sulphate of zinc, and cimicifuga are all objectionable either negatively or positively, and are probably only worthy of consideration after all others have been tried ineffectually. The bromides will undoubtedly to a partial extent control the tremors of disseminated sclerosis in man, though it is necessary to push the drug to its full physiological effect, and the attendant depression and disturbance of digestive function make the slight relief an expensive one. In a patient of Erlenmeyer's the tremor disappeared for two days under curare. Spitzka, following the suggestion of Feris, found veratrum in small doses repeated every few hours quite effective, but considered it objectionable because of the necessity of increasing the dose to almost toxic quantities. Arsenic has been tried repeatedly, and next to hyoscyamine seems most reliable. It has been used by Eulenberg hypodermically for the relief of tremors met with in cases other than multiple sclerosis, and with success. Reasoning by analogy, it might seem justifiable to resort to the same measure in these cases, but it appears probable that the unstable temperament of a child would render hypodermic medication a questionable procedure in this disease. Galvanism applied as in the method described for general treatment is sometimes palliative as regards the tremor.

There are few other symptoms which require special attention or treatment. The headache usually observed only during the earlier period may persist and prove troublesome, as in Unger's case, in which it remained for three years obstinately. Galvanism applied to the brain, one electrode to the occiput and the other over the frontal bone, has been suggested for the relief of headache when present. The current in such cases should be applied very gradually, through a rheostat or the physician's hand, beginning at zero and increasing to two or at most three milliampères. The pains in the extremities which are not uncommon in adult patients affected with multiple sclerosis seldom occur in childhood.

TUBERCULAR MENINGITIS.

By A. JACOBI, M.D.

The first accurate description of tubercular meningitis was given by Robert Whytt (1768), under the name of hydrops of the cerebral ventricles. Capuron and Chauvel employed the term brain fever (*fièvre cérébrale*). Fabre et Constant introduced the term "tubercular meningitis" into our present nomenclature in 1835. Since that time the literature of the subject has been so immense that, as this work is designed to supply the clinical wants of the practitioner, I prefer to abstain from extensive references and limit my remarks to as comprehensive a review of the subject as can be accomplished within the briefest possible space.

A large percentage of the cases of tubercular meningitis admits of a subdivision of the symptoms into three parts, or stages, which correspond more or less distinctly with the development of anatomical changes. The first is that of hyperæmia of the pia mater and irritation, the second exhibits the symptoms of exudation, pressure, and consecutive anæmia, and the third those of the extinction of the functions of the centres. Still, it is impossible fully to discriminate the boundary-lines between them in every case.

The first signs of a diseased condition are of a very indefinite nature. Many of the children have a previous history of pallor and debility, diarrhœa and vomiting, "colds," bronchitis, conjunctivitis, eczema, and lymphadenitis,—"scrofula." They emaciate, are pale, lose their appetite, become quiet, unwilling to play, peevish, cry a good deal, or appear perfectly listless.

The physical, intellectual, and moral changes are well remembered by the attendants when the further development of the case suggests inquiry into the previous history. The moroseness, peevishness, and depression are often relieved by sleep, which, however, is frequently interrupted. Many, indeed, sleep badly, complain of headache, avoid the light, and hold their head in their hands. Often it is one and the same spot that is constantly supported or pressed. There is, as a rule, no elevation of temperature at this time, and rarely is there an increase in the frequency of the pulse; on the contrary, it begins rather early to be retarded and slightly irregular. The tongue is furred, an occasional brief vomiting-spell occurs, and the "gastric" symptoms are treated unsuccessfully. The vomiting is not preceded by nausea, is quick and propulsive, and mostly takes place while the child is being moved on the bed or raised from the pillow the same as in pregnancy or sea-sickness. There is constipation, with much headache, and the quantity of urine diminishes. Some hyperæsthesia of the abdomen, reminding of peritonitis, is liable to mislead the diagnosis. Gradually there is more somnolence, more photophobia, and now and then some increase of temperature, which usually, however, does not exceed 100½° or 101° F. In this way, in addition to a few weeks filled with the premonitory symptoms detailed above, a week or more may pass.

The pulse becomes more slow and irregular, even intermittent, the headache more severe; there is a constant frown. Though the patients do not complain, they are evidently suffering much. They grind their teeth, cry in their somnolence suddenly, unexpectedly, at longer or shorter intervals, are delirious or dull and apathetic. From their apathy they are easily roused, however, look surprised, give a brief answer, and drop again into their drowsiness. They yawn often; respiration is interrupted, and restored by deep and long sighs; the abdomen is retracted. The pulse is still more retarded, 60 or less, more irregular, for many days in succession. The secretion of urine becomes more scanty, the constipation more obstinate; the eyes are but half closed, under the falling eyelid the balls are seen to move slowly about from side to side, and with a peculiar expression of utter fatigue and forlorn absent-mindedness the upper eyelid is gently raised, reveals the blank soulless ball, and gently falls again. Now and then one of the eyelids is more drawn than the other, one eye more perceptible than its mate. The pupils begin to dilate, sometimes both, sometimes one more than the other, and the staring expression becomes more intense. The face is apt to flush suddenly, mostly in circumscribed spots; the skin in contact with the pillow is congested, and the mark of the touching finger is easily seen for some time. A gentle friction results in the appearance of a well-circumscribed blush corresponding with the size of the friction (Trousseau's "mark"): it appears slowly, after a few seconds or a minute, stands out in distinct relief a few minutes or more, and disappears as slowly. Consciousness is gradually waning more completely, a slight twitching is seen round the mouth or eyelids, or suddenly a general convulsion may set in, followed by ptosis, facial paralysis, strabismus, or paralysis of the extremities. This paralysis is more apt to be unilateral, however, than general. The convulsions may reappear, may be general or partial, and are accompanied with more dilatation of the pupils, and more loss of consciousness, which rarely returns for a few moments. Indeed, all the cerebral symptoms may exhibit peculiar alterations: sometimes quite unexpectedly the paralysis may pass by, or become more local; sometimes there is an arm paralyzed without the leg or the face participating,—never, however, a lower extremity without the upper. Even ptosis may disappear temporarily.

The convulsions are not always clonic,—sometimes there are tonic contractions; the rigidity of the neck becomes more intense, the face more flushed, perspiration sets in, the pulse increases in frequency, the temperature begins to rise to 102° or more. This is the beginning of the end, which generally arrives in about three days. More convulsions, deeper coma, rapid pulse, from 150 to 240, higher temperature, from 103° or 104° to 107°, injected and purulent conjunctivæ, and Cheyne-Stokes respiration, predict and usher in the final dissolution, after an observation of generally three or four weeks, and a duration of all the symptoms, the premonitory included, of from five to six weeks.

A latent form of the disease is occasionally met with in children who have been phthisical for some time. In them the cerebral symptoms do not become perceptible until two or three days before death. The sopor, even at this late stage, is mild, and the convulsions are not violent. In other cases marked with previous general tuberculosis the symptoms are more severe. Vomiting and headaches are very marked, delirium and convulsions very pronounced, the pupillary symptoms often unilateral. In many there is diarrhœa in place of constipation, and a tumid abdomen instead of the retraction. In the infant the fontanel may be found raised and pulsating until near the end. It is at that early age that the premonitory symptoms and the initiatory stage described above are often absent. The attack is very sudden; temperature is high in the beginning; strabismus, trismus, opisthotonus, or general convulsions set in at a very early period, and death occurs in a few days. Thus it is that the diagnosis is often difficult. There are quite a number of cases, particularly in older children and in adults, in which from the beginning a peculiar typhoid condition obscures the recognition of the exact condition of things. In these, somnolence, headaches, delirium, and convulsions appear early, and alternate with one another; the abdomen is not retracted, the fever is very high from the beginning, the thoracic symptoms predominate, and the patients die soon with the symptoms of both cerebral and general acute miliary tuberculosis.

Ophthalmoscopic examination reveals hyperæmia of the retina. Sometimes tubercles are discovered in the choroid, in the shape of spherical white spots near the optic papilla. But they are far from being of constant occurrence.

Pathological Anatomy.—The morbid changes are mainly found in the pia, on the surface of the brain, in the ventricles, and principally all over the base. The fossa Sylvii, pedunculi, chiasma, cerebellum, and pons are the main seats of exudation. The pia is less transparent than normally, thickened and succulent, and can easily be detached. Exudation is sometimes copious, either serous, or purulent, or gelatinous, sometimes quite consistent; the cavities are dilated with either a clear or a turbid fluid. In the exudation are found a number of small tubercles, yellow when older, semi-transparent when recent. They are mostly found in the walls of small blood-vessels, the lumen of which is often contracted by their presence and by the accompanying proliferation, particularly of the adventitious membrane (Cornil), and of the nuclei of the neuroglia and the varices of the capillaries (Hayem). The mechanical obstruction of circulation depending thereon, and the supervening arteritis, together with the pressure of the solid exudation on the blood-vessels, often give rise to softening of the cerebral substance, either locally or over a large area. In the brain-substance there are other changes depending on the presence and amount of the exudation. The ventricles are dilated, their walls sometimes hardened by the pressure, but more often softened; the convolutions are flattened, the substance is œdematous, and capillary apoplexies resulting from the changes described as having occurred in the blood-vessels are frequent. Such capillary hemorrhages are particularly common in the choroid plexus.

The tubercular conglomerates are sometimes found to be quite numerous, and still there is little or no meningitis. In others there is either a hydrocephalic effusion, or a jelly-like or solid exudation without a local tuberculosis, while in all of them the number of tubercles in the other organs is very large, thus rendering the nature of the intracranial affection certain. Indeed, there are but few cases in all the forms of tubercular meningitis in which the bacillus has not been found.

The other organs show tubercular degeneration. In most cases the whole course of the disease, and the results of the autopsies, are those of miliary general tuberculosis. In adolescents the lung-symptoms are the most predominant, in the child (besides the brain) the peritoneum and the bronchial glands are thoroughly affected. The liver, the spleen, the kidney, and particularly the pleuræ, exhibit tubercles in all their stages, mostly recent; the bronchial and mediastinal glands, not rarely those of the neck also, are caseous or suppurating in their centres, and the lungs are seldom intact. Bones are often carious, parts of the surface are covered with eczema, and now and then we find an arthritis ("tumor albus"), or lupus. Old tubercular infiltrations in the lungs and glands are very commonly found in the autopsies of such cases; occasionally also in the kidneys, spleen, and liver. In some of these cases the cerebral changes are but trifling.

Etiology.—Tubercular meningitis is not frequent in the first months of life, but is more so than is supposed by many. Indeed, as the symptoms differ from those exhibited in later years, mistakes in diagnosis are easily made. After the seventh year its prevalence is not great. The large majority of cases, therefore, are observed between the end of the second and the seventh year. Dentition, worms, frights, falls, blows, have troubled more seriously the brains of writers than of children dying with tubercular meningitis. This and meningeal tuberculosis generally are infectious diseases, or rather parts of a general tubercular infection. Thus the main cause is in a specific invasion, which, with our present knowledge, means the importation of the tubercle-bacillus. It spreads along the lymph-ducts and blood-vessels and thus excites inflammation. It is true, however, that the amount of inflammation and the number of eruptions need not be parallel to each other: sometimes there may be even meningitis without a localized tubercle, while there is a general tubercular infection of the whole body. Children of phthisical parents are more liable to be affected, among them boys more than girls. But, according to our present knowledge of the rare occurrence of direct hereditary transmission, it is certain that the principal danger lies in the congenital insufficiency of the tissues, and, more positively, in the many opportunities for contagion by food (breast-milk and cow's milk) and inhalation.

I knew a family in indigent circumstances and with limited dwelling-space, in which ten children died of tuberculosis, many of them of tubercular meningitis. But one of the eleven survived,—now a man of over thirty, with a narrow chest, but in fair health. A number of cases in the same family are observed frequently. The children are often delicate, of less than normal weight, lively, intellectual, prematurely overburdened with mental work, impressionable: some I have known to be active masturbators. But there are those who are fat, rotund, of apparently vigorous health, but with long, thin eyelashes and occasional catarrh of the conjunctivæ or the nose; again, those who have always been believed to be in health, in whom nothing but an accidental exposure to the virus, often not traced, can be accused of being the cause of the mischief. But the history of every patient must be thoroughly studied. In many cases the origin of tubercular meningitis can be traced to the bronchial glands, in which tubercular degeneration may remain dormant for years. Through them, whooping-cough and measles may give rise to a general tubercular invasion after years have elapsed; another proof of the perversity of the "expectant" treatment in "self-limited" diseases, and of the fact that therapeutic nihilism is as dangerous as over-medication.

Uffelmann, Oehme, and Baeumler report cases of tubercular meningitis connected with (or depending on) erythema nodosum, which is itself both the cause and the result of embolic processes not only on the extensor sides of the extremities and the tendons, but also in other regions of the body (mouth, throat, conjunctiva, endocardium, pleura, kidneys). Oehme's case died of tubercular meningitis a few weeks after the termination of a febrile erythema nodosum. Baeumler saw three cases of the same affection complicated with vesicular eruptions on the conjunctivæ. Two of them had a protracted high fever, one multiple lymphomata and tumefaction of the spleen, one bilateral pleurisy and neuralgias on arms, legs, and head, and one was suddenly taken with the symptoms of acute hydrocephalus. At the autopsy he found miliary tuberculosis of the bronchial glands, the pia, pleura, liver, spleen, and kidneys, and central softening in a gland adjoining the tracheal bifurcation.

Diagnosis.—The diagnosis of tubercular meningitis, in the majority of cases occurring between the second and the seventh year, is not very difficult. After the premonitory stage of irascibility, peevishness, or depression, the temperature being normal or nearly so, the respiration becomes irregular, the pulse retarded and irregular, vomiting and constipation set in, the urine is diminished in quantity, the abdomen is retracted, and the intense headache changes gradually into, or alternates with, stupor increasing into coma.

Sighing is noticed at a very early period. Cheyne-Stokes respiration is a symptom of paralysis and an ominous sign.

The pulse may be rapid at first, particularly in infants, but is mostly slow while being irregular, from sixty to eighty beats, or less, in a minute, of changeable quality, now and then almost imperceptible, towards the end (the last three days) very rapid and then more regular. It must be remembered, however, that the pulse may also be both quite slow and irregular in gastric disorders and in chronic anæmia. I have observed this pulse in anæmic children through years, and have often been in anxious suspense about its significance.

Vomiting sets in suddenly, without preceding nausea, usually while the head is raised, but also while on the pillow, and is propulsive and quick. It is mostly a symptom of irritation of the pneumogastric nerve, and is therefore characteristic of meningitis at the base. In meningitis of the convexity there is little or no vomiting, and the pupils remain equal, but there is ptosis; there is more unconsciousness, sopor and coma, and the disease is of shorter duration.

Headache accompanies the whole course of the disease. At first it is continuous and uniform; the patient frowns constantly, and is apt to carry the hand up to the head. Afterwards there are occasional abrupt shrieking yells, which are apt to persist until complete stupor sets in.

Constipation is quite obstinate, but there is diarrhœa sometimes in very young infants. As in them the retraction of the abdomen may be absent, the differential diagnosis between tubercular meningitis and typhoid fever may become difficult.

The temperature of the body (rectum) is not characteristic; it is not typical, and has no regular curves. It is at first usually nearly normal, sometimes subnormal; only in the very young it is high, because in them tuberculosis is apt to be universal. When in children of four or five years the temperature is high and less intermittent at an early period, the case is probably one of purulent meningitis. There is no regular proportion between pulse and temperature. Towards the end it becomes very high, in proportion to the increasing paralysis. The latter may result either from pressure brought about by the presence of the exudation, or from anæmia. In this case the paralysis may be but temporary.

Convulsions may set in at almost any time. They may be localized or general, mild or severe, clonic or tonic. Death is rarely the immediate result of an attack of convulsions, but a local paralysis (motor oculi, facial, an upper extremity, or total hemiplegia) may set in immediately after such an attack. This symptom is pathognomonic for the diagnosis from uræmia, in which a local affection is extremely rare.

D'Espine and Picot describe a symptom which they call static ataxia.

It appears at a pretty early period. When the child is placed on his feet, he totters, his walk is undulating, his features exhibit fear and anxiety.

Retraction of the head and stiffness of the muscles of the neck constitute a frequent symptom of tubercular meningitis. But the same symptom is met with in other cerebral and spinal diseases, as in cerebro-spinal and spinal meningitis, and in either idiopathic or secondary muscular affections. Torticollis is observed as both an acute and a chronic affection, as the result of rheumatism, trauma, angina, articular inflammation, and adenitis, the possible existence of one of which must be taken into account in every case of retraction. In most of those cases it begins quite suddenly, and with some fever and moist surface; passive or active motion is exceedingly painful, but both the rigidity and the pain are mostly confined to one side only. There is no lack of consciousness, as in meningitis, and there are no convulsions. The course of the acute symptoms is short, though convalescence be ever so protracted and relapses frequent. Still, A. Legroux[1] reports a case the symptoms of which were so doubtful as to render the differential diagnosis from tubercular meningitis quite difficult. It lasted eleven days. Among the symptoms of these acute retractions Legroux counts fever, perspiration, lassitude, pain, rigidity, vomiting, and constipation. A very close local examination is required to diagnosticate in such cases between meningitis, pharyngitis, otitis, and bronchitis. The author relies on the localization of muscular hardness as one of his best differential signs, and states that in his cases the abdomen was not retracted as in tubercular meningitis, and the vomiting lasted but a short time.

The differential diagnosis of tubercular meningitis from simple, and syphilitic, and cerebro-spinal meningitis is by no means easy in every case. Meningitic symptoms occurring in a syphilitic child will find their explanation in a conscientious consideration of the history and of the general signs of syphilis; they are mostly not accompanied by high fever. This form is liable to recover after a protracted anti-syphilitic treatment. Cerebro-spinal meningitis is frequently seen as an epidemic, but the number of cases occurring in a community is apt to be so small that the practitioner who is called upon to decide in an individual case will find his task quite difficult. It is not preceded by a long term of prodromi, as tubercular meningitis mostly is, and begins with a higher temperature than the latter. In a short time the characteristic eruption makes its appearance in cerebro-spinal fever, but it may be absent. After a while hemorrhages will appear in the latter; consciousness is preserved more intact than in the tubercular form; the dilatation of the pupils, or one of them, is perceptible at a very early period, it being the result of irritation of the sympathetic, while in tubercular meningitis it results from oculo-motor paralysis. The vaso-motor disturbances (Trousseau's "marks") are observed at the very beginning of a cerebro-spinal meningitis, while they do not appear until late in the tubercular cerebral form. In simple meningitis, the "acute hydrocephalus" proper, there are hardly ever any prodromi. Its onset is more sudden, the symptoms more marked, its temperature higher, and its course more rapid. Recoveries are possible, as they are also in rheumatic meningitis, which occurs more frequently than some authors (D'Espine and Picot) appear to believe. They quote but fifteen cases. Still, this kind of meningitis will be more frequently diagnosticated when observers become aware of the fact that acute rheumatism in infancy and childhood is easily overlooked, for the reason that its articular symptoms are liable to be but trifling, the local pain and swelling not being necessarily in proportion to the dangerousness of the affection. In rheumatic meningitis the onset is sudden, and the fever high from the very beginning. The suddenness of the attack makes it resemble the cerebral symptoms produced by the influence of an acute eruptive fever, such as scarlatina, which is apt to prove fatal within a day, with high temperature, unconsciousness, convulsions, and early heart-failure, without the characteristic eruption making its appearance.

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[1] L'Encéphale, 1885.

I have seen cases in which the diagnosis between tubercular meningitis, typhoid fever, and acute yellow atrophy of the liver was very difficult; in a few it became impossible. The latter, rare though it be, will exhibit, besides the high temperature and the grave brain symptoms, a large amount of albumin in the urine, and jaundice. Typhoid fever, when running its typical course, may exhibit its peculiar temperature-curve, which, however, is absent in most cases. Ehrlich's test is not always conclusive. Abnormal cases of typhoid fever, moreover, will commence with a high temperature, and can easily be mistaken for the abnormal cases of tubercular meningitis, which may also begin, particularly in the very young, with elevation of temperature. Besides, typhoid fever, while influencing the cerebral functions either directly or through participation of the kidneys in the morbid process, is liable to be actually complicated with meningitis to such an extent that the diagnosis may become very difficult indeed.

Acute nephritis, or the uræmia of chronic nephritis, may often be mistaken for tubercular meningitis. The examination of the urine ought never to be omitted, even in cases which do not appear doubtful. Inequality of the pupils speaks more for the presence of some form of meningitis, but there are cases of uræmia, occasionally, though rarely, in which the dilatation of the pupils is not equal. This dilatation, when produced by direct cerebral paralysis, is more stationary and less easily influenced by light, while very often the pupils of uræmia, when dilated, will contract somewhat under the influence of light, dilate again, and appear to float gently between its two extremes.

The cerebral symptoms which accompany inflammatory diseases of the lungs are frequently mistaken for genuine simple or tubercular meningitis. The brain suffers in pneumonia mainly when this occurs in the upper lobes, but there are exceptions to this rule. In a number of cases I made the diagnosis from the absence both of prodromal symptoms and of slowness and irregularity of respiration. The latter is apt to be accelerated in pneumonia, even under the influence of a secondary meningeal congestion or exudation. On examination, the physical symptoms of pneumonia were then easily detected.

The ophthalmoscopic examination may be very useful in doubtful cases, but is so actually but seldom; for there are proportionately but few cases of tubercular meningitis in which it is not negative.

In Marshall Hall's "hydrocephaloid" condition there are a number of symptoms which also occur in tubercular meningitis, such as a moderate amount of fever, gritting the teeth, and sighing, also pallor and collapse, half-closed eyes, injected conjunctiva, coma, and convulsions. But there is also the history of an acute disease (gastro-enteritis) preceding the cerebral symptoms, which facilitates the diagnosis.

Prognosis.—The prognosis of tubercular meningitis is a very bad one. It is true that recoveries have been reported. Rilliet has seen some cases getting well: one of them had a relapse, and the post-mortem examination showed the correctness of the diagnosis. Lebert also proved by an autopsy that one of his cases had terminated favorably, previously. A few such cases are reported in *L'Union Médicale*, April 12, 1881. Many recoveries are claimed by those authors who recommend specific treatment, as, for instance, Hahn, whose treatment consisted in the application of tartar emetic ointments. A famous New York teacher and consultant claimed six recoveries out of a hundred cases, but admitted that he saw each of the cases but once, that he had to rely on the reports of the attendants casually given, and that mistakes in diagnosis were possible.

I have seen what I thought to be a recovery; it was followed by death from necrosis of the cranial bones and secondary meningitis superinduced by the tartar emetic torture. Another case of mine was that of a boy of two years, who "recovered." When growing up he was stupid, eccentric, wilful, lazy. He is now nearly thirty years old, and lives in an insane asylum. A third case, in which the diagnosis of tubercular meningitis was possibly correct, terminated in incurable blindness. These are the best results I can boast of.

D'Espine and Picot and Baginsky never saw a case getting well. Cadet de Gassicourt claims that his recoveries were those of meningitis occurring around tumors, syphilitic gummata, and cerebral sclerosis.

Thus, when the diagnosis is beyond doubt, the prognosis may be considered absolutely bad. Doubt, however, is justified in a number of cases of acute simple meningitis of the base and ventricles.

Treatment.—Every baby in a tubercular family or surroundings is liable to be infected with meningeal tuberculosis. Therefore whatever preventive treatment of scrofula and tuberculosis has been detailed in the second volume of this work and found available in practice ought to be utilized. Besides, the hair ought to be worn short, the head kept cool, no feather pillows used, the skin accustomed to cold water. Eczema must be cured, the bowels must be kept regular. Though it be true that tubercular meningitis is an infectious disease, it is still a meningitis, and irritation and hyperæmia of the brain are predisposing causes. Therefore before the eighth year there ought to be for a predisposed child no schooling, and after that time no overwork must be permitted. Cod-liver oil and arsenic may be administered for months and years in succession, the latter with occasional interruptions. Iron is to be given in digestible and small doses, as long as there is no vascular excitement.

When tubercular meningitis is diagnosticated during the prodromal stage,—a rare occurrence,—noise and light must be excluded, and absolute rest enforced. Even at this early period the prognosis is bad. Altogether, no treatment can be entered upon with any degree of probability of saving the patient. There are indications for treatment, and in the present condition of therapeutics we can do no better than to fulfil them, with conscientiousness and—hopelessness. Leeches and vesicatories I have seen to do no good. The latter irritate and bother. Other external measures have been the use of blue ointment, oleate of mercury, croton oil, ointments containing tartar emetic (1 to 8–12), and lately iodoform. Forty years ago it was the former that "saved" the patient; four years ago[1] it was the latter. A dose of calomel may be given to relieve constipation, and repeated from time to time; or some other convenient purgative and enemata. On the supposition that the inflammatory element was predominant, mercurials have been administered persistently.

Iodide of potassium appears indicated partly as an absorbent and partly for its effect on the circulation. I know that in some cases that took large doses for weeks, though there were hundreds of tubercles, but little effusion was found in the post-mortem examination. Large doses appear to be required for that end; they are easily tolerated, a child of two years readily taking, and retaining, from one to two or three drachms daily.

Persistent vomiting requires pills of ice and opiates; if the stomach be intolerant, the remedy may be introduced into the rectum, or Magendie's solution into the mouth, on the tongue, where it is readily absorbed. So long as the intolerance of the stomach continues, rectal alimentation must be resorted to. During all this time, the heart being weak, cardiac stimulants may be given,—digitalis, strophanthus, spartein, camphor; but no caffein, and no alcohol.

Convulsions indicate the inhalation of chloroform, the use of chloral (occasionally morphine) by mouth or rectum or subcutaneously, and the bromides. A warm bath may be given, but care must be taken that there is no undue tossing about. Cold to the head ought not to be thought of except when the fontanels are raised and the head hot and the temperature elevated,—hence mainly towards the end of the disease. Infants do not stand ice to the head for any length of time; unless watched, it produces collapse and heart-failure. As the bacillus proliferates fast in high temperatures, antipyrin or phenacetin will act agreeably, provided they be not given without one of the permissible cardiac stimulants.

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[1] Nilson, in Arch. f. Kinderk., 1886.

Tubercular peritonitis appears to have been benefited, occasionally, by laparotomy. Will tubercular meningitis ever be improved by similar interference? It is not probable. For no operation of any kind could be made, without the danger of shock, on the skull of a child afflicted with tubercular meningitis, except where fontanels and sutures were still open; and the intracranial space, though opened, is not a cavity accessible to air and capable of being emptied, in the sense of the abdominal cavity. Thus, if there be any cases of spontaneous recovery in tubercular meningitis, it does not appear that our direct therapeutics in that malady are other than hopeless.

CEREBRAL HEMORRHAGE, THROMBOSIS, AND EMBOLISM.

By B. SACHS, M.D.

THE brain of the child, as of the adult, is subject to marked disturbances of its blood-supply. We may have a large effusion of blood from rupture of one or more blood-vessels (hemorrhage); or an occlusion of one or more blood-vessels due to coagulation of blood *in situ* (thrombosis); or the occlusion of a blood-vessel by a plug whirled into it from some distant part of the circulatory apparatus (embolism). Much as these pathological processes differ from one another, the clinical symptoms resulting therefrom have many points in common, so that we are justified in considering cerebral hemorrhage, thrombosis, and embolism under one head. We shall be compelled, however, to note carefully certain important distinctions, not only as regards the etiology and morbid anatomy of each process, but also with reference to the variations in the mode of onset and in the development of other symptoms due to these several lesions.

Hemorrhage, embolism, and thrombosis have long since been recognized as the most frequent causes of adult apoplexy; not so with regard to the cerebral apoplexies of early life. There was supposed to be a marked distinction between the child and the adult individual in this respect. Only a few years ago Bernhardt considered cerebral hemorrhage a rather rare occurrence in the child, and referred to an exceptional case of Bastian.[1] Henoch, Rilliet and Barthez, and other authors were of the same opinion. Certain it is that these vascular disturbances are very much rarer in childhood than in later years; but in a recent study of cerebral palsies of early life by the author and Dr. F. Peterson[2] it was clearly shown that in one hundred and five cases of cerebral infantile palsies thirty-five cases were due either to hemorrhage, thrombosis, or embolism, and that this percentage would have been materially increased if the reported cases of atrophy, sclerosis, and cysts, forty out of one hundred and five, most of which were originally due to hemorrhage, had been added to the list. Osler,[3] in sixteen cases out of ninety autopsies of hemiplegia, found embolism seven times and hemorrhage nine times.

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[1] Lancet, 1883.

[2] Sachs and Peterson, Journal of Nervous and Mental Diseases, May, 1890.

[3] The Cerebral Palsies of Children, Monograph, Philadelphia, 1889.

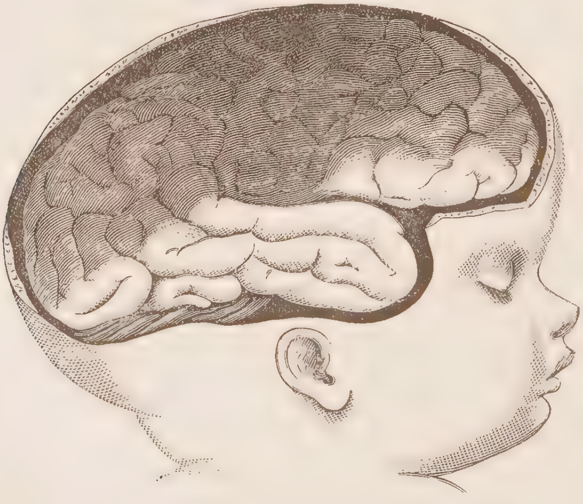
The scarcity of early autopsies accounts for the prevailing, but mistaken, views on this subject. For detailed proofs of the writer's views the reader is referred to the paper mentioned above, which will be quoted again and again in the course of this article. The entire subject-matter was gone over so thoroughly in that paper that reference to it will avoid frequent repetitions.

ETIOLOGY AND PATHOLOGY.

Under this heading it will be necessary to consider each process separately.

HEMORRHAGE.—Rupture of a blood-vessel in the brain of a child may be brought about by a variety of causes,—either by a diseased condition of the blood-vessel itself, or by the application of some extraneous force. Taking the latter cases first, we have to include among them those in which the difficulties of labor (contracted pelvis, protracted labor, instrumental delivery) are equivalent to a traumatic injury to the skull and brain. Dr. Sarah McNutt[1] has shown that the extravasation of blood (chiefly meningeal) is more apt to be at the base in cases of head-presentation, and over the convexity in cases of foot-presentation. The application of the forceps was formerly supposed to be the element of greatest danger, but by a comparison of statistics it was shown by us (S. & P.) that protracted labor was

FIG. 1.



Meningeal hemorrhage in a new-born child; death on twenty-second day. (After Dr. Sarah McNutt.)

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[1] American Journal of Obstetrics, 1885.

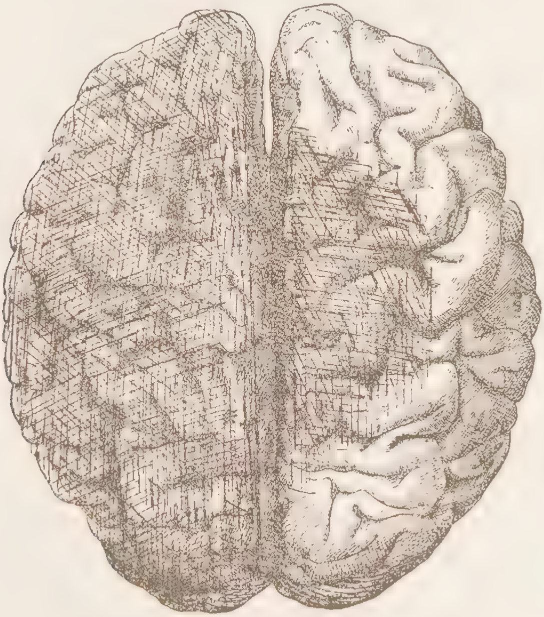
a more potent cause of harm to the child's brain than the application of the forceps.[1]

First-born children are most apt to suffer in this way. Gowers[2] found in twenty-four cases of meningeal hemorrhage that seventeen were first-born. The intense venous congestion which accompanies prolonged compression of the brain is made worse by the asphyxia at birth. It is directly responsible for the hemorrhage, which issues in all probability from the smaller vessels of the pia and not from any large artery.

Apart from these congenital cases, traumatism is a factor of some, though not of great, importance in the causation of hemorrhage. In ninety-one cases of acute cerebral palsies six only were due to traumatism, and these six were no doubt cases of hemorrhage. The following case, with autopsy by Dr. Peterson, is to the point:

Case I.—A. W., male, aged fifteen, bright at school, expert swimmer, at the age of eight or nine years was in the habit of diving a distance of twenty to thirty feet from a railroad bridge. He soon began to have intense headaches, growing worse until the age of twelve years, when mental changes began to be apparent. Admitted to Poughkeepsie Asylum. One month later, severe epileptic fit. Two days thereafter, right hemiplegia, with constant right-sided hemi-epilepsy; coma; four days later, death. The wide-spread pachymeningitis hæmorrhagica interna will be seen on the accompanying figures (2 and 3).

FIG. 2.



Showing extent of pachymeningitis hæmorrhagica over superior surfaces of hemispheres. (Sachs and Peterson.)

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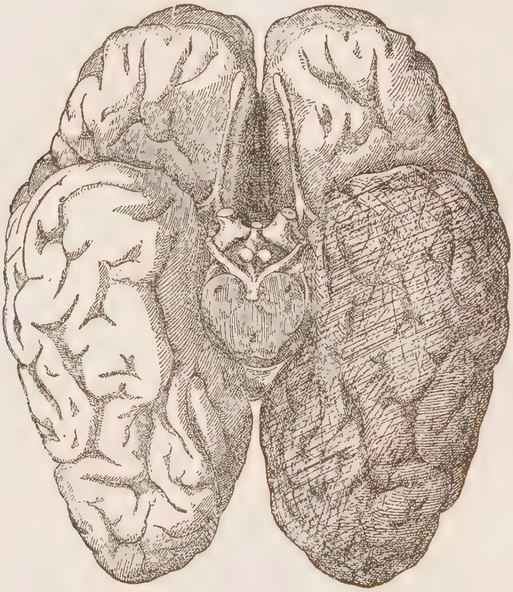
[1] Loc. cit., p. 308.

[2] Diseases of the Nervous System, p. 801.

Over the convexity of the left hemisphere the hæmatoma was very thick, consisting of strata of various ages, some of them undoubtedly dating from the beginning of the symptoms. The brain weighed thirty-five and one-fourth ounces. No other lesion in any part of the brain.

FIG. 3.



Showing extent of process on inferior surfaces of the left hemisphere.
(Sachs and Peterson.)

However severe the traumatic injury may be, a subdural or subarachnoidal hemorrhage is much more likely than an intracerebral clot; but such intracerebral hemorrhages do occur, and in these cases the symptoms very much resemble those of adult apoplexy.

We now pass to a consideration of the diseased condition of the blood-vessels which renders them liable to rupture and hemorrhage. That a normal blood-vessel (artery or vein) will resist any sudden increased pressure, and that if diseased it will probably give way under normal and surely under increased pressure, are facts so well known that we need not do more than merely mention them. In the adult we have accepted it as a fact that the bursting of a miliary aneurism is the direct cause of an effusion of blood; the researches of Virchow, of Kölliker, of Meynert, and, above all, of Charcot and Bouchard, have settled this beyond dispute. The miliary aneurism, more apt to occur in the smaller intracerebral vessels, is formed by a proliferation of the cell-nuclei in the adventitia or outer coat,—periarteritis. Eichler and Zenker, however, maintain that the process begins in the inner coat,—an endarteritis. Whatever the true condition may be, the pressure of the blood against the diseased wall causes a bulging which gradually forms into a small aneurismal sac. In the adult these changes in the smaller blood-vessels occur in conjunction with atheromatous changes in the larger blood-vessels, yet they do not hold any direct causal relation to one another. Inasmuch as there are no atheromatous changes in the blood-vessels of the young, the existence of aneurisms in early life was formerly seriously questioned; but they have been found in a youth of twenty (Gowers), in a boy of fifteen (Baker),[1] and Prof. Osler has found a large aneurism of the anterior cerebral artery[2] in a boy six years of age. With further inquiry on this point there can be no doubt that miliary aneurisms will be often found.[3]

As a rule, the autopsies have been made years after the onset of the trouble. It is often difficult to find the initial lesion. For this reason it has not been possible to formulate for children such a table as Durand-Fardel gave for adult apoplexies. According to this table, miliary aneurisms occurred most frequently in the blood-vessels of the optic thalamus and corpus striatum; then followed in regular order the blood-vessels of the cerebral convolutions, of the pons, of the centrum semi-ovale, of the cerebellar peduncles, of the crura cerebri, and of the medulla. A study of a large number of cases of cerebral hemorrhage in the adult proves that the order named above will also stand for the relative frequency of lesions in the different parts of the brain.

On several occasions[4] I have called attention to a peculiar degeneration of the walls of the cerebral blood-vessels of the young. Von Recklinghausen[5] lays some stress upon the fatty degeneration occurring in the blood-vessels of children, but, as far as I can see, this condition has never been considered in connection with this subject. In view of these facts, it is surprising that hemorrhage is not more frequent. The following interesting table, taken from Gowers's work,[6] shows a steady increase in the liability to hemorrhage from the first year of life up to the age of eighty.

AGE.	LIABILITY (number of cases in 1000 x persons living in each decade).
1–10	1.8
11–20	3
21–30	6
31–40	12
41–50	19

Direct Causes.—All conditions which bring about increased blood-pressure are apt to cause hemorrhage. Thus, we have hemorrhage in renal disease, with hypertrophy of left ventricle and increased blood-pressure.

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[1] Annals of Anatomical and Surgical Society, Brooklyn, 1879.

[2] Canada Medical and Surgical Journal, 1886.

[3] See this Cyclopædia, vol. ii. p. 874, article by Dr. Warren.

[4] Journal of Nervous and Mental Diseases, 1887 and 1890.

[5] Pathologie des Kreislaufes und der Ernährung, p. 84.

[6] Diseases of the Nervous System, p. 777.

Traube has shown that in the compensatory hypertrophy of valvular disease of the heart the blood-pressure is not increased, and these conditions are not so apt, either, to produce hemorrhage. It will be well to keep these facts in mind, though I cannot recall the record of a single case of early cerebral hemorrhage in which renal disease played an important part. The sudden increase of pressure in the cerebral vessels while lifting heavy weights, while violently throwing the head backward, while straining at stool, and during the paroxysms of whooping-cough, favors the occurrence of hemorrhage; but there are other and still more potent causes, and here we must mention first and foremost the acute infectious diseases,—measles, scarlet fever, pneumonia, typhoid, small-pox, diphtheria, and cholera. In one case which I reported some years ago, the hemiplegia, which was in all probability due to an intracerebral clot, came on after an ordinary non-diphtheritic tonsillitis.[1]

In some instances the apoplexy follows immediately upon the initial convulsions; in other cases it develops in the later course of the disease. If the palsy follow immediately upon the convulsions, the mechanical congestion during the convulsions may be chiefly to blame. Then, again, we (S. & P.) have shown that the cerebral palsies of early life, of which a large percentage is due to hemorrhage, occur after ordinary convulsions, and sometimes after a single convulsive seizure. I append a table showing the causes in eighty-three cases of hemiplegia. It should be remembered, however, that not all of these cases were due to hemorrhage, thrombosis, or embolism.

HEMIPLEGIA.

CAUSES GIVEN.	NO. OF CASES.
Convulsions	20
Pneumonia	6
Trauma to head	6
Pertussis	4
Measles	2
Scarlatina	3
Onset with fever	2
Hereditary syphilis	2
Cerebro-spinal meningitis	2
Onset with fever and convulsions only	2
Fright	2
Hydrocephalus	..
Vaccinia	1
Typho-malarial fever	1
Small-pox	1
Tonsillitis	1
Epileptic seizure	1
Gastro-enteritis	1
Unascertained	26
Total	83

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[1] Intracerebral Hemorrhage in the Young, Journal of Nervous and Mental Diseases, September and October, 1887.

As a matter of fact, Eichler[1] and Rilliet and Barthez[2] have found that changes in blood-vessels are most apt to occur after typhoid, rheumatism, scarlatina, cholera, icterus, acute miliary tuberculosis, and scrofula. Most of these diseases figure in our clinical statistics (see table), and there would be considerable justice in regarding this as evidence that many of these early cerebral palsies are due to vascular troubles.

Hereditary syphilis is a factor not to be overlooked. No doubt thrombosis is the more common sequel of syphilis. But since Lechner[3] has shown that twenty-three per cent. of three hundred cases of syphilis in the adult exhibited some form of cerebral hemorrhage, there is every reason to fear that congenital syphilis is apt to do the same for the brain of the child.[4]

O. Heubner[5] refers to pachymeningitis hæmorrhagica in a syphilitic infant under one year of age, and Hans Chiari[6] describes the syphilitic degeneration of the cerebral blood-vessels in a child fourteen months old.

Hemorrhage may occur in the course of a general tuberculosis, and also during the growth of a cerebral tumor.

If an effusion of blood has occurred into the substance of the brain, the blood rapidly coagulates and forms a dark-red clot; by degrees this clot turns a lighter color,—brown or yellowish brown. The red blood-corpuscles become disintegrated, the coloring-matter of the blood is changed into a large number of hæmatoidin crystals, the white blood-corpuscles assimilate fat, and, as granular corpuscles, abound in the clot. By degrees the clot grows smaller, its surface is well worn off, and in course of time a so-called apoplectic cyst marks its former seat. In the course of years even this cyst may disappear, and nothing but a scar then remains. In many of the cases which have been examined post mortem no trace of a hemorrhage could be discovered, while the secondary conditions, such as atrophy, sclerosis, and cystic formations, were very prominent.[7]

THROMBOSIS.—In the adult, thrombosis occurs with well-marked atheromatous degeneration of the arteries. The next most frequent cause is syphilitic endarteritis; and, lastly, thrombosis is the result of disordered states of the blood. In the child the two latter are the more important factors. Our table shows that thrombosis was present in five out of seventy-nine cases of hemiplegia. It would, therefore, appear to be less frequent than either hemorrhage or embolism. This fact should be borne in mind, though on the post-mortem table it is at times impossible to say whether the plugging of an artery is due to embolism or to thrombosis. Abercrombie relates two cases which are very much to the point:[1]

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[1] Archiv für Klinische Medizin, 1878, vol. xxii.

[2] Maladies des Enfants.

[3] Jahrbuch für Psychiatrie, 1881.

[4] On this subject, see Rumpf, Die Syphilitischen Erkrankungen des Nervensystems, chapter v.

[5] Virchow's Archiv, vol. lxxxiv. p. 269.

[6] Ibid., vol. lv. p. 368.

[7] The subject of secondary degenerations after cerebral lesions is treated in another portion of this volume.

Case II.—Boy, aged six years; diphtheria; on the fifteenth day convulsions, left hemiplegia; death eleven days later. Thrombus in right middle cerebral artery; no heart-disease; no source of embolism.

Case III.—Girl, aged six years; history of syphilis; fell down paralyzed on right side. Autopsy five years later. Old thrombus in middle of longitudinal sinus. Atrophy of left hemisphere and sclerosis of convolutions; thickened membranes; middle cerebral arteries diseased.

This last case of Abercrombie and the clinical history of an interesting case by Seibert[2] point to thrombosis as the direct cause of hemiplegia, possibly of other forms of cerebral palsies in children with hereditary syphilis. Thrombosis in the course of a tubercular meningitis deserves notice, though little attention is, as a rule, paid to the resulting hemiplegia, which often occurs only a few days before death.

Thrombosis in consequence of altered states of the blood is conceivable and probable. This may account for the cerebral paralyses of exhausting diseases, and particularly for those which are subsequent to summer diarrhœa and chronic gastro-intestinal catarrh. And, lastly, we have to consider a theory of Gowers, who insists that a thrombosis of superficial cerebral veins and sinus-thrombosis constitute a frequent cause of hemiplegia in children. Hemorrhage may be associated with such thrombosis. This morbid condition is a well-known one, without, however, being necessarily associated with hemiplegia.[3] Attractive as this theory of Gowers is, the proof is still lacking. I repeat what was said in the article on cerebral palsies: "The autopsies analyzed give no evidence whatever of this condition."[4]

EMBOLISM.—It cannot be surprising to find that paralysis from embolism of a cerebral artery is a not uncommon occurrence in children, for the conditions favoring embolism are often present. Wherever there is a diseased valve with vegetations or thrombosis of the pulmonary veins, as in a case reported by Henoch,[5] there is liability to embolism. Whence it follows that the acute infectious diseases, particularly scarlatina, diphtheria, measles, small-pox, acute and chronic bronchitis, and pneumonia, are the diseases most frequently complicated with cerebral paralysis (generally hemiplegia) due to embolism.[6] I must remind the reader, however, that in all these diseases cerebral hemorrhage is possible.

The reported cases of cerebral embolism in children are too few in number to yield any proof as to the greater liability of the plug reaching one artery rather than another; but, since the conditions are the same as in the adult, the order of preference is probably the same. In the adult the middle cerebral arteries and their branches are the chief seat of embolism. Those of the left side are more often affected than those of the right. Next in order come the posterior cerebral and the vertebral arteries. Embolism of the anterior, the cerebellar, and the basilar arteries is extremely rare. A case of embolism of the basilar artery was reported by Gowers in 1882.[1]

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[1] British Medical Journal, June 18, 1887.

[2] Jahrbuch für Kinderheilkunde, No. 22, 1885.

[3] Case of Handford, British Medical Journal, 1887, p. 1098.

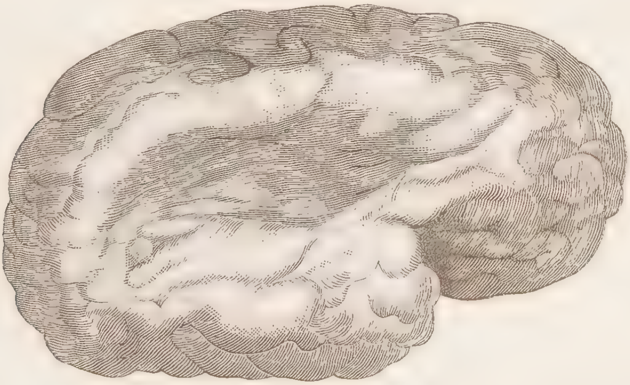
[4] Gowers refers to one such case by Money (Treatment of Disease in Children, p. 445).

[5] Vorlesungen über Kinderkrankheiten, 1881, p. 223.

[6] Osler, op. cit., p. 93, suggests that in diphtheria there may be plugging of the smaller cerebral arteries with micrococci.

Of the large number of cases of acute cerebral palsies which the present writer has seen, not one has exhibited symptoms pointing to lesions in any other part of the brain except the cortex or the vicinity of the internal capsule. The large majority of these were evidently due to lesions within the distribution of the middle cerebral artery,—another proof of the fact that the same laws obtain here as in the cerebral lesions of adult life. A few cases in which embolism or the secondary effects of embolism were found post mortem will be of interest in this connection:

FIG. 4.



Cyst formed by softening of brain-substance secondary to obstruction of middle cerebral artery beyond the first branch. Child, nineteen months old. Death seven months after onset of paralysis. (After Ashby and Wright.)

Case IV.[2]—Boy, one year old; marked cyanosis, due to obstructive pulmonary disease and constant dyspepsia; was seized with vomiting and convulsions, followed by paralysis of left arm and leg. Child apparently quite blind; no retinal hemorrhages; optic disks normal. Face drawn to right side; apparent loss of sensation in arm and leg (child was semi-comatose.—S.). Rapid recovery from most of the symptoms, but rigidities in arm and leg. Seven months after seizure, death from bronchitis. Autopsy showed a large cyst occupying the central part of the convexity of the right hemisphere (Fig. 4), within the distribution of the right middle cerebral artery, excepting the branch to the inferior frontal convolution. Thrombosis or embolism of middle cerebral artery.

Case V.—Boy, aged two and a half years, suffering from chronic pneumonia and caseous degeneration of bronchial glands. Sudden development of right hemiplegia, with contracture. Post-mortem examination revealed embolism of left arteria Sylvii. The heart was normal, but the embolus had evidently come from a thrombus in a large branch of the right pulmonary vein (a similar occurrence in the case of Henoch referred to above).

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[1] Brain, vol. i.

[2] Ashby and Wright, Diseases of Children, p. 389.

Case VI.[1]—A boy, seven years of age, suddenly manifested right hemiplegia with aphasia. Autopsy showed plugging of the left anterior and middle cerebral arteries. The particles of fibrin composing the plug were evidently derived from a coagulum attached to the margin of the mitral orifice.

Thrombosis and embolism may give rise to the occlusion of cerebral arteries. In the former case it is brought about gradually; in the latter, suddenly. The post-mortem appearances are often very similar, and in many instances it is not possible to state whether the clot has been formed *in situ* or whether it has come from a distance. In cases of thrombosis, if the clot is not too old, the successive layers of deposit can be recognized by a difference in color; if old, it is very firmly attached to the artery-wall, while on the side away from the heart there is apt to be the more recent and darker deposit. A thrombus may take up any part or the whole of a cerebral artery, while an embolus is commonly lodged at a point between two arteries, or at least at a point where the lumen of the artery becomes suddenly narrower. If the embolus is of old standing, it has gathered so much additional blood about it that it cannot be distinguished from a thrombus.

The immediate effect of occlusion of an artery is to cut off the blood-supply of a definite region. This is a permanent effect, except in those rare cases in which an embolus is broken up and the smaller particles are carried away by the blood-current. In so-called terminal arteries such a procedure is impossible. Heubner[2] has reported a very unusual case in which two clots, one in each middle cerebral artery, were found. Each clot was perforated and permeable. But such perforation does not take place early enough to be of any service to the diseased territory.

The area supplied by an occluded artery soon undergoes marked changes. At first the tissue grows paler, the nerve-elements disintegrate, and the whole area becomes softer (anæmic necrosis). A little hemorrhage often takes place, in consequence of the rupture of the capillary vessels, and the area of softening assumes a reddish appearance. By degrees this red softening is changed into yellow softening. White softening is found in very late stages of red and yellow softening, and more particularly in those areas of the brain which have a very limited blood-supply.

If the person survive for a long time, the area of softening will have undergone still further changes. There is a distinct line or area of demarcation between the diseased area and the surrounding healthy parts. At this boundary slight hemorrhages are very apt to occur; the softened area sinks in, there is an accumulation of fluid over it, and in the course of time a distinct cyst is developed. Such embolic cysts cannot be distinguished in every instance from cysts due to hemorrhage, except that in the latter, if the lesion be not of too old a date, we are apt to find an abundance of hæmatoidin crystals. In other cases connective tissue forms in the softened area; the nerve-elements disappear, and the fluid is absorbed. Finally a cicatrix with considerable atrophy marks the former area of softening. No doubt some of the cases of infantile cerebral palsies associated with atrophy, sclerosis, and cysts were originally of embolic origin.

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[1] Mentioned by Dr. Sansom, in his article on chronic endocarditis, this Cyclopædia, vol. ii. p. 832.

[2] Wiener Med. Blätter, 1883.

Softening may occur in any part of the brain, but the cortex, corpus striatum, and optic thalamus are the areas most frequently affected. The corpus striatum and optic thalamus are supplied by terminal arteries. Collateral blood-supply is impossible unless there be an anomalous distribution of blood-vessels. The cortex, on the other hand, has a capillary net over the larger part of its surface, which may permit the restoration of circulation to a very limited extent; but the frequent occurrence of cortical softening proves that this capillary net-work cannot be depended upon. The area about the fissure of Sylvius suffers most from obstruction of the middle cerebral artery, while the regions higher up often escape.

SYMPTOMATOLOGY.

Excepting perhaps the mode of onset, cases of cerebral hemorrhage, thrombosis, and embolism may present the self-same symptoms. This part of the subject has been gone over so minutely in the paper published together with Dr. Peterson that the statements here made are based upon the deductions contained in that paper. The study of the symptoms of onset will be easier for us if we divide the cerebral spastic palsies into those which come on at birth (birth-palsies), or are congenital, and those which come on in an acute fashion in the earlier years of life.

The truly congenital cases—*i.e.*, those not due to traumatism at birth—are in all probability not cases of embolism, thrombosis, or hemorrhage, and do not, therefore, concern us here. Among these are undoubtedly to be classed many cases of porencephalus, defective development, and cases of cortical agenesis, such as have been described by Kast[1] and the writer.[2]

The birth-palsies and some of the truly congenital cases are, as a rule, cases of difficult labor,—either merely protracted labor with head-presentation, or cases of foot- or breech-presentation in which the head has been compressed tightly in the pelvic canal and instrumental delivery was necessary. At birth the child is asphyxiated ("blue child"), the funis may or may not be wound about the neck, and considerable effort is necessary to resuscitate the child. When once regular respiration is established, no further symptoms may be noted for days, weeks, or months; in other cases, a closer examination reveals paralysis of one or more members of the body, and the paralysis may take the form of a hemiplegia, a diplegia (both arms and both legs), or a paraplegia (both legs). Monoplegias occur, but are rare, while the simultaneous involvement of both sides of the face and of both arms and legs has, so far as I can see, never been noted. In all such cases rigidity of an arm or of a leg is soon established, and convulsive seizures of the parts paralyzed are apt to occur. These convulsive movements very often do not appear until weeks or months have elapsed, even in cases in which the paralysis dates from birth. This is to be accounted for by the well-known fact that the convulsive movements are due to irritation of the cortical motor areas, and that these areas do not become "irritable"—*i.e.*, are not sufficiently developed—until some time after birth.[1]

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[1] Archiv f. Psych., 1888, vol. xix. p. 297.

[2] Journal of Nervous and Mental Diseases, August, 1887.

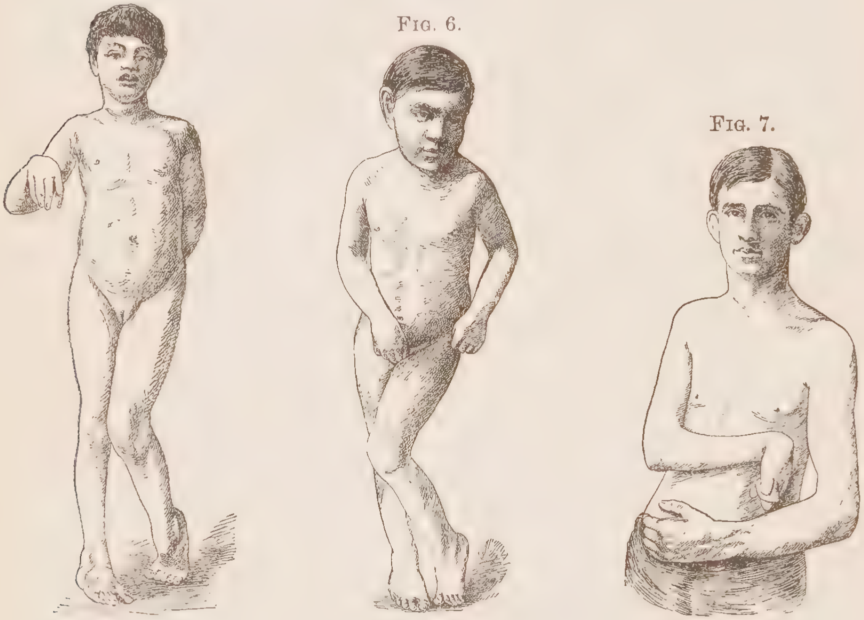

Fig. 5.

Fig. 6.

Fig. 7.

Figs. 5, 6, and 7 are intended to show deformities in cases of infantile cerebral palsy.

In a few cases both the paralysis and the convulsions do not appear until some weeks after birth, although everything may point to the birth act as the cause of the injury sustained by the brain. From now on, such cases as we have described behave exactly like those with acute onset.[2]

In cases in which there is an acute onset of the paralysis, the latter may appear in the midst of perfect health, or, as is more often the case, during or after an acute infectious disease. The onset is marked in the majority of cases by loss of consciousness, more or less prolonged coma, and convulsions. Exceptions to this rule will be given later on. A rise of temperature generally accompanies the onset of these acute cerebral spastic palsies, varying from 101° to 104° and in some cases to 107° F. Unusually high temperatures appear as a foreboding of a fatal termination. Bourneville insists that there is subnormal temperature at the very beginning of an attack, but I have never been able to convince myself of the truth of this in children. Perhaps the cases are not seen early enough. During the condition of coma one side of the body may already give evidence of paralysis, or the fact that convulsive seizures begin in or are limited to definite parts of the body points to a probable paralysis or paresis of such parts. The eyes may look to the side of the lesion. The distribution of the paralysis may result in a hemiplegia (the most frequent form), or in a diplegia or a paraplegia. Aphasia is often present. A spastic rigidity or slight contracture may be added to the paralysis. The tendon reflexes in the paralyzed members are exaggerated; this is particularly true of the wrist, biceps, and triceps reflexes, and of the knee-jerks. Ankle-clonus is not so frequently elicited, because the opposing contracture forbids the free excursion of the foot. In all these cases peculiar disturbances of motion are developed, resulting in choreiform, athetoid, or associated movements. In addition we find a retardation of growth of the part or parts paralyzed, and in many cases considerable mental impairment. No marked changes in sensibility (tactile or otherwise) were observed in the many cases the writer has seen, nor has he been able in any case to discover hemianopsia, although Freud[1] of Vienna has described two undoubted instances of this in very young children. The electrical reactions are not seriously altered; there is at least in no single instance an approach to a reaction of degeneration. In consequence of the wasting of some muscles and the contracture of opposing muscles, the faradic and galvanic responses may be considerably diminished.

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[1] Soltmann's observations (Meynert, Psychiatry, translated by Sachs, p. 166) are to the point.

[2] In the paper so often referred to, we have given distinct proof that it is a mistake to attribute all diplegias and paraplegias to meningeal hemorrhage at birth, and to suppose that the acute cases are almost invariably hemiplegic in form. We have hemiplegia due to traumatism at birth, and diplegias as well as paraplegias of acute onset.

Several of the symptoms referred to above deserve further consideration.[2]

THE FORM OF PARALYSIS.—Hemiplegia is so frequent a form of paralysis that many authors have made the "acute cerebral [spastic] palsy of children" and "infantile hemiplegia" synonymous terms. We need not insist again upon the injustice of this procedure. Remembering that meningeal or cortical hemorrhages are so much more frequent in children than in adults, and that this hemorrhage may be, and often is, bilateral, we have at least one reason why diplegias or paraplegias, and not simple hemiplegias, are developed. The face is at times involved, but not so frequently as in adult apoplexy; this also is accounted for by the position of the face-centre in the cortex, which is not so frequently covered by clots as are the arm- and leg-centres. If recovery sets in, the paralysis diminishes very much, after the fashion of adult apoplexy,—the leg first and most, the arm last and least; the face recovers more rapidly than in adults; a case now under my observation at the Montefiore Home, of a boy six years of age, with left hemiplegia, is one of the few in which there is permanent paralysis of one-half of the face. Strabismus I have observed in one case of right hemiplegia, in four cases of left hemiplegia, and in three cases of diplegia.

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[1] Wiener Med. Wochenschrift, Nos. 32 and 33, 1888.

[2] Coma and convulsions will be discussed under the head of Diagnosis.

As for aphasia, my own studies have led me to believe that the same laws obtain as in the cases of adult apoplexy. I have recorded seventeen cases of hemiplegia with aphasia. Ten were cases of right hemiplegia and seven were cases of left hemiplegia. Eight of these seventeen cases were observed in private practice, about which the information was thoroughly reliable.

Of these eight, five had been distinctly aphasic, and three of the five were cases of left hemiplegia. This relatively large proportion of aphasia in cases of left hemiplegia is in striking contrast to the conditions in the adult. My own experience is in accord with that of Bernhardt,[1] who finds that aphasia in children accompanies left as well as right hemiplegia. Prof. Osler, on the other hand, in thirteen cases of aphasia finds but one case with left hemiplegia. By way of parenthesis, I add that defective speech is present in many cases, and is to be regarded as one of the symptoms of mental impairment.

The reflexes are exaggerated, but in eight cases of hemiplegia we found them normal in four, diminished in one, and absent in three cases. These exceptional cases, and also those in which the element of spasticity is entirely wanting, must be accounted for on the supposition that the initial lesion was slight, or that, for some other reason, secondary changes did not ensue.

The majority of cases of peculiar disturbances of motion[2] are cases of early cerebral palsies. We distinguish choreiform, athetoid, associated, rhythmical, and ataxic movements. Thus, in one hundred and five cases of hemiplegia choreiform movements occurred in six, athetoid movements in twenty-one, associated movements in fifteen, rhythmical movements in one, ataxia in one, and tetanoid contractions in one; and in twenty-four cases of diplegia choreiform movements occurred in one, ataxia in one, athetoid movements in one, and nystagmus in two.

A word about associated movements. In some cases of infantile hemiplegia the movements of the sound side, particularly of the sound arm and hand, bring about movements in the corresponding parts of the paralyzed side, and *vice versa*. Thus, if a patient attempt to squeeze the hand of the physician with his sound hand, the paretic hand also closes, or at least imitates the movement. We succeeded in obtaining tracings of such associated movements in the case of a patient in whom this symptom was so well developed that in buttoning or unbuttoning his clothes the paretic hand would repeat every movement.[1]

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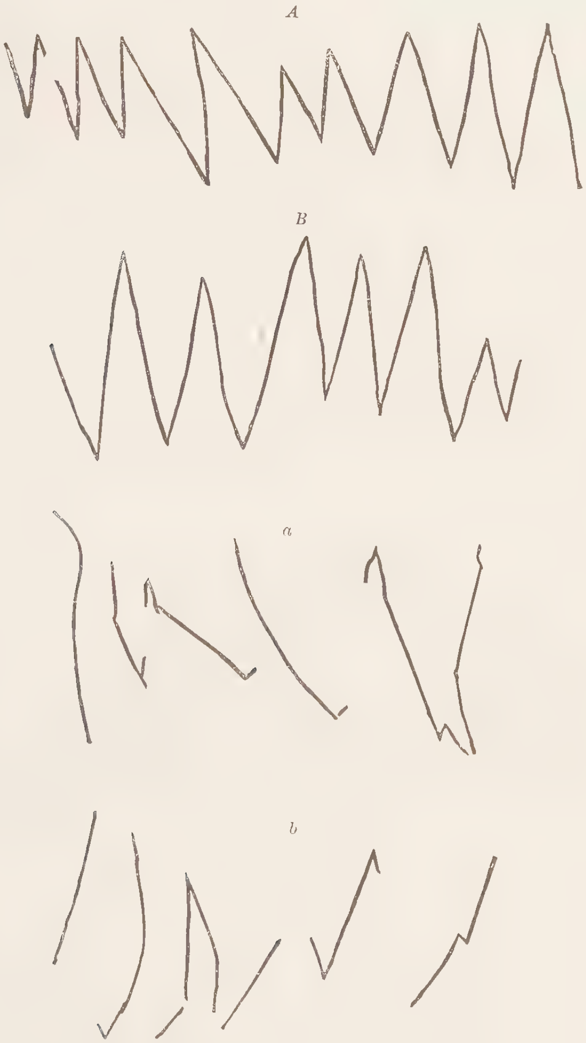
[1] Virchow's Archiv, 1885, vol. cii.

[2] This condition has been ably described and analyzed by P. C. Knapp, Journal of Nervous and Mental Diseases, 1887, p. 480.

Fig. 8.



A and *B*, movements of the normal hand; *a* and *b*, movements of the paretic hand. The patient was asked to draw a zigzag line on a blackboard, using his sound hand.

Contractures are very apt to be developed early and permanently. In some cases the contracture is so extreme as to make a leg or an arm entirely useless. The common forms of contracture are given in the following table:

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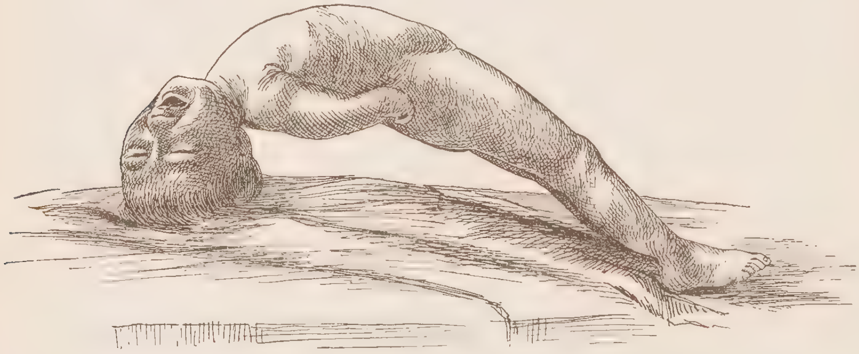
[1] Sachs and Peterson, p. 313.

Form of Contracture.	Hemiplegia.
Flexors of elbow	29
Flexors of carpus and fingers	23
Flexors of knee	5
Extensors of knee	1
Talipes equino-varus	17
Talipes equino-valgus	3
Talipes equinus	2

Flexors are more apt to be involved than extensor muscles; adductors more than abductors. In this the construction of joints may come into play. Wernicke[1] has recently shown that these groups showed the same difference as regards the development of paralysis in adult hemiplegia. This fact was well known to me, and has been regularly demonstrated to my classes of students at the New York Polyclinic. But this is not peculiar to hemiplegia: I have found it to be true, also, of paralyses due to spinal-cord lesions.

Since epilepsy is not only a degenerative neurosis, but also a symptom of cortical disease, it need not be surprising that it is so frequent an accompaniment of infantile cerebral palsies. And such patients may suffer either from general epilepsy or from the Jacksonian form. Among one hundred and five cases of infantile hemiplegia there were forty-one cases of general epilepsy, nine of the Jacksonian type, and one case of petit mal. About fifty per cent. of all hemiplegics were epileptics. This percentage tallies fairly well with the statistics given by Gandard, Wallenberg, and Osler. In at least one case which I have seen, of a girl aged seventeen years, the epilepsy was the only active symptom of a lesion which had caused left hemiplegia four years previously.

Fig. 9.



Case of paraplegia. Photographed during an epileptic seizure.

The following tables are given for brevity's sake, and will impart all needed information with regard to mental defects and cranial deformities:[2]

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[1] Berlin. Klin. Wochenschrift, 1890.

[2] See also a paper by Drs. Fisher and Peterson, Cranial Measurements in Twenty Cases of Infantile Cerebral Hemiplegia, New York Medical Journal, April 6, 1889.

TABLE—*Showing the Relation of Mental Defect to the Age of Onset of Palsy.*

AGE OF ONSET OF PARALYSIS.	FEEBLE-MINDEDNESS.	IMBECILITY.	IDIOCY.	TOTAL.
Congenital	6	15	14	35
Under three years	10	18	6	34
From three to five years	2	3	..	5
From five to ten years	2	3	..	5
Over ten years	1	..	..	1
Total	21	39	20	80

TABLE—*Showing the Relation of Mental Defect to the Form of Palsy.*

FORM OF MENTAL DEFECT.	HEMIPLEGIA.	DIPLEGIA.	PARAPLEGIA.	TOTAL.
Feeble-mindedness	16	2	3	21
Imbecility	31	7	1	39
Idiocy	7	8	5	20
Insanity (epileptic)	1	..	..	1
Total	55	17	9	81

TABLE—*Analysis of Stigmata Degenerationis (exclusive of Contractures) present in Fifty-Seven Cases of Cerebral Palsy in Children.*

	HEMIPLEGIA.	DIPLEGIA.	PARAPLEGIA.	TOTAL.
Microcephalus	21	5	2	28
Leptocephalus	19	1	..	20
Macrocephalus	4	..	1	5
Marked cranial asymmetry	25	..	2	27
Marked facial asymmetry	19	..	..	19
Cranium proganæum	5	2	..	7
"Gothic" palate	9	1	..	10
Imperfectly-developed teeth	10	1	1	12
Supernumerary teeth		1	..	1
Hirsuteness		1	..	1
Neuropathic ear	1	1	..	2
Strabismus	5	3	..	8

In the above the writer has assumed that, if paralysis follow upon cerebral hemorrhage, thrombosis, and embolism, it will take the form of hemiplegia, diplegia, or paraplegia. That this is practically so, may be inferred from the fact that in over one hundred and sixty cases of infantile cerebral palsies I have not seen a single case which would not come under one of these clinical subdivisions. But hemorrhage, thrombosis, and embolism may occur in other parts of the brain as well as in the cortex and in the vicinity of the subcortical ganglia; and, if so, other symptoms will arise. Such cases have been reported.

Lannelongue[1] refers to a boy eight years of age who had left hemiplegia with aphasia, followed by convulsions, coma, and death. Autopsy revealed a clot in the right crus cerebri, also red softening along the left fossa Sylvii, and thrombosis of the longitudinal sinus and of the veins of the pons. This was a complicated case, and of the one important symptom of crus lesions, paralysis of the oculo-motor nerve of the side of the lesion (opposite to the paralyzed side), nothing is said. Höven[1] and Wallenberg[2] have reported cysts in this same region. Lesions in the pons would be characterized by paralysis of the fifth, sixth, or seventh nerve, with hemiplegia of the opposite side; if the lesion be high up in the pons, it will be difficult to distinguish such a case from ordinary hemiplegia. In addition we may expect marked sensory disturbance, loss of the conjugate movements of both eyes towards the side of the lesion. According to the position of the lesion, we may also have paresis of both lower and both upper extremities, as I have seen in one adult case. There is no case that should be more carefully examined than one of suspected pons lesion; but, inasmuch as I have not found a single well-authenticated case of this kind in a child, I have not the right to enter at length upon the symptoms due to disease of the pons. Lesions of the medulla, which are particularly prone to prove fatal, have little practical interest; cases of syphilitic disease of the basilar arteries, with thrombosis and softening, are the only ones which might call for a differential diagnosis. The well-known bulbar symptoms, difficulties of speech and of deglutition, irregular respiration, hemiatrophy of the tongue, associated with unilateral or bilateral paralyses, would suggest a lesion in the medulla oblongata. As a review of the more important symptoms of cases of possible thrombosis, hemorrhage, and embolism in children, I give the condensed histories of a few cases of cerebral spastic palsies:

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[1] Cited by Steffen, Gerhardt's Handbuch, vol. v. p. 339.

Case VII.—A. F., aged four and a half years, male, first child, difficult labor and instrumental delivery. From very first day, right hemiplegia. Slight athetosis and associated movements. Contracture at elbow, formerly pes equino-varus, improved by operation. All reflexes of right side lively. Mental condition fair.

Case VIII.—J. K.,[3] male, aged seventeen years. Right hemiplegia at eight years of age following typho-malarial fever; was delirious and unconscious during nineteen days; no convulsions. After recovering from coma, right arm, face, and leg were found paralyzed. Complete aphasia and entire loss of memory of everything occurring before typhoid. Had to be re-educated. Athetoid and associated movements. Reflexes exaggerated on right side. Enormous contracture of flexors of right hand and fingers, and great retardation of growth of right upper extremity. Right leg somewhat smaller than left; right talipes valgus; asymmetry of face. Electrical reactions and sensation entirely normal. Has recovered speech fully, and is bright, but several years behind others in education.

Case IX.—J. O., female, aged sixteen. Congenital diplegia; mother kicked in abdomen by horse two months before birth of child and made unconscious thereby. Three other children, all healthy. Tedious labor; no instruments used; no fits or convulsions. Did not attempt to creep or walk; teeth at usual age. Patient has menstruated since tenth year, and was weak in back, arms, and leg from earliest childhood. Extreme spastic contracture of adductors and flexors of thighs; double talipes varus, equinus on right side. Left arm worse than right. Athetoid movements of left hand. Has frog walk. Intelligence good.

Case X.—M. L., male, aged three years. Congenital diplegia. Asphyxiated during labor. Mother had pneumonia, and died five days post partum. Rigidity of arms, legs, and back. Hands did not unclinch for two years. Frequent convulsive seizures alternately of right and left side, including face. Cannot talk, walk, or stand. Feeble-minded; cross-legged position and all reflexes exaggerated.

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[1] Arch. f. Psych., 1888, vol. xix. p. 563.

[2] Ibid., vol. xix. p. 297.

[3] See Fig. 7.

Case XI.—M. M., boy, two and one-half years old; one and one-half years previously had pneumonia and tonsillitis, with fever up to 102° F. Four days after this, typical right hemiplegia and aphasia, positively without coma or convulsions. The onset was as typical as in the ordinary mild hemorrhage into the internal capsule in the adult, and the progress of the disease and the mode of recovery (which became complete) were quite like what we see in adult cases.

DIAGNOSIS.

The general diagnosis of infantile cerebral palsy, or spastic cerebral paralysis, of infantile cerebral hemiplegia, diplegia, or paraplegia, is easy to make. But how can we distinguish between meningeal and intracerebral lesions, and between thrombosis, hemorrhage, and embolism?

In children the presumption is in favor of cortical or meningeal lesions. We must bear in mind the unusual delicacy of the blood-vessels of these parts. It is only or chiefly in those cases which resemble adult apoplexy (clinically) that we need be suspicious of intracerebral (capsular) lesions. Other things being equal, prolonged coma and *repeated* convulsions are in favor of meningeal lesions; coma of short duration or entire absence of coma and convulsions is indicative either of intracerebral lesion or of very slight surface lesions. The rapid development of idiocy and epilepsy is also more apt to follow meningeal (cortical) lesions. Paralysis of arm and leg without involvement of face, and monoplegia, point to the cortex. Convulsive movements of paralyzed parts are generally due to cortical irritation.

Birth-palsies are apt to be due to *meningeal* hemorrhage; palsies developing during acute infectious diseases are often of intracerebral origin.

Cranial deformities are more apt to accompany surface lesions.

The points of differential diagnosis between hemorrhage, thrombosis, and embolism are difficult to give. Almost every author has endeavored to formulate the differential points, but one of the latest, and a very able author,[1] realizes that the task is as difficult as it ever was. In many cases it is well-nigh impossible to come to any definite conclusion on this point, and the scarcity of autopsies on children makes it impossible properly to test our theories. The symptoms will vary not only according to the nature of the morbid process, but also according to the location and size of the area affected. The symptoms of the onset are of the greatest importance, for they are the symptoms which indicate the general effect upon the brain as a whole, and not the local effect of the lesion itself.

What can we infer from the occurrence of coma and convulsions? The most diverse views have been held with regard to these symptoms, as any one can discover who will read Dr. Browning's paper on the occurrence of coma in sudden spontaneous brain lesions.[2]

I submit the following points, not that I think them absolutely correct, but because they are the result of much reflection on the subject and have been deduced from an unusually large clinical experience. This has especial reference to children.

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[1] Edes, Pepper's System of Medicine.

[2] Medical News, February 18, 1888.

MENINGEAL CASES.—*Hemorrhage.*—Birth case; coma; convulsions; paralysis present at birth or developed very soon thereafter; convulsions often repeated; cranial asymmetries.

Thrombosis.—Acquired; syphilis or marasmus; gradual onset; no loss of consciousness; convulsive twitchings precede paralysis.

Embolism.—Very sudden development of paralytic symptoms; heart-disease; increasing paralysis; monoplegias. Coma and convulsions may or may not be present.

INTRACEREBRAL LESIONS (less frequent in children).—*Hemorrhage.*—Acquired case; if slight, little or no coma; no convulsions; paralysis involving face, arm, and leg of one side; rapid improvement; very little, if any, mental change; possible complete recovery. If hemorrhage is large, prolonged coma, initial convulsions, but convulsions are not apt to be repeated; sudden complete hemiplegia; little mental change, but contractures may remain; onset during acute infectious disease.

Thrombosis.—Specific or scrofulous history; gradual onset, with prodromata, such as headache, dizziness, weakness, etc.; no coma, or paresis leads up to coma; no convulsions unless ganglionic masses are involved; paralysis develops gradually, but remains as it is for a long time.

Embolism.—Sudden onset of motor symptoms; coma possible, but, as a rule, rapid recovery therefrom; cardiac disease; initial convulsions, not apt to be repeated.

Previous to the publication of Strümpell's[1] lecture in 1884 on polioencephalitis acuta, it would not have been necessary to question the occurrence of hemorrhage, thrombosis, and embolism in many cases which are now supposed (by many) to be due to polioencephalitis. Strümpell's views—that infantile cerebral palsy is the analogue of infantile spinal palsy, and that the former is due to a polioencephalitis, as the latter is due to a poliomyelitis—have taken such a hold upon the medical public that this condition is often diagnosticated without good cause. I cannot do better than to reproduce here what was said in the article written a few months ago, and those who have thought that polioencephalitis helped us out of the difficulty will be surprised to find what little evidence there is of such a morbid process:

"First of all, in order to distinguish this from two other lesions which have been termed polioencephalitis inferior (progressive bulbar paralysis) and polioencephalitis superior (nuclear ophthalmoplegia), let us speak of this as polioencephalitis corticalis. What proof have we that there is such a condition? Anatomical proof, none: we are willing to concede, however, that some of the many cases of atrophy and sclerosis may have been due to this polioencephalitis, but it is unfortunate for Strümpell's theory that all of the autopsies made soon after the onset of the disease have shown other conditions, and not a polioencephalitis. But let us be charitable or just, and say that even these autopsies were not made in cases sufficiently recent. We must add, however, that cases which correspond very closely to the cases which Strümpell considered typical of polioencephalitis corticalis showed hemorrhage, embolism, etc., of recent origin. Strümpell says, however, that not all cases of infantile hemiplegia need be due to this cause, and that many authors have misinterpreted his views. Is there no probability, then, that a few or any of the cases of infantile hemiplegia are cases of polioencephalitis corticalis? There is some circumstantial evidence showing that there is a brain-lesion which would seem to be analogous to spinal palsy of children (poliomyelitis anterior). Möbius gives the history of two children of one family, aged one and one-half and three years respectively, who were stricken down with fever, loss of appetite, and somnolence. One developed a typical poliomyelitis of the upper extremity; the other, spasmodic hemiplegia without aphasia. This is striking clinical evidence, though some might claim it to have been merely a coincidence. Another proof: Strümpell has but very recently reported two cases of adult apoplexy in which every one would have made the diagnosis—and indeed he made it—of embolic softening, but the post-mortem examination revealed a condition of encephalitis hæmorrhagica of the gray as well as of the white matter. Marie, who is inclined to support Strümpell, expressed the opinion in 1885 that the encephalitis would attack the white as well as the gray matter, and thinks that this would not destroy the analogy with poliomyelitis, for in that condition the white fibres are sometimes involved. Jendrassík and Marie favor the perivascular (inflammatory) origin of the condition of lobar sclerosis, which they have carefully described. In view of all this, we venture the opinion that polioencephalitis corticalis may be the cause of some of the cases of infantile palsies; but, we add, not of the hemiplegia alone, for we have seen several cases, including one seen by the courtesy of Dr. Holt, in which all the symptoms were those of Strümpell's disease, but there was a diplegic and not a hemiplegic form of palsy. In these cases the cerebral character of the symptoms was so distinct that a confusion with poliomyelitis was out of the question.

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[1] Jahrb. f. Kinderheilk., N. F., 1884, vol. xxii.

"We insist that, until further pathological proof shall be forthcoming, polioencephalitis corticalis shall be diagnosticated last, not first."

All that we can safely say is that some of the cases of acute cerebral palsy in children coming on during acute infectious diseases, or without any known cause, with high fever, coma, and convulsions, *may* be due to a polioencephalitis.

I am inclined to suspect this condition in those cerebral cases in which the *invasion* symptoms are very marked and entirely out of keeping with the lasting paralyses.

It is known that hemorrhages and other vascular troubles are apt to come on in the course of general tuberculosis; hemorrhages also occur in the case of tumors of the brain; moreover, tumors sometimes cause the sudden development of cerebral palsy. This fact should be borne in mind; but the general symptoms of intracranial tumor, headaches, dizziness, optic neuritis, will furnish the points of differential diagnosis.

Lastly, in the case of congenital hemiplegias, diplegias, or paraplegias, it will be necessary to differentiate between those cases due to hemorrhage or early softening, and those due to other processes, to an early encephalitis, or to a condition of cortical agenesis, to a defective development (porencephalus), and so on. It is no easy task to give such points, and only a few can be given.

Birth palsies are generally due to meningeal or cortical hemorrhage; the child is blue. In the truly congenital cases of early encephalitis or porencephalus we generally get a history of traumatism to the mother, while the child is apparently healthy at birth, though often prematurely born. Defective mentality and paralysis appear soon after birth.

Where there is a very high degree of idiocy without convulsions, but with wide-spread paralysis, the condition of cortical agenesis, a simple arrest of development, as described in cases by Kast and myself, may be present. Any active inflammatory condition of the cortex which sets in during the intra- or extra-uterine period is apt to cause convulsions in early life.

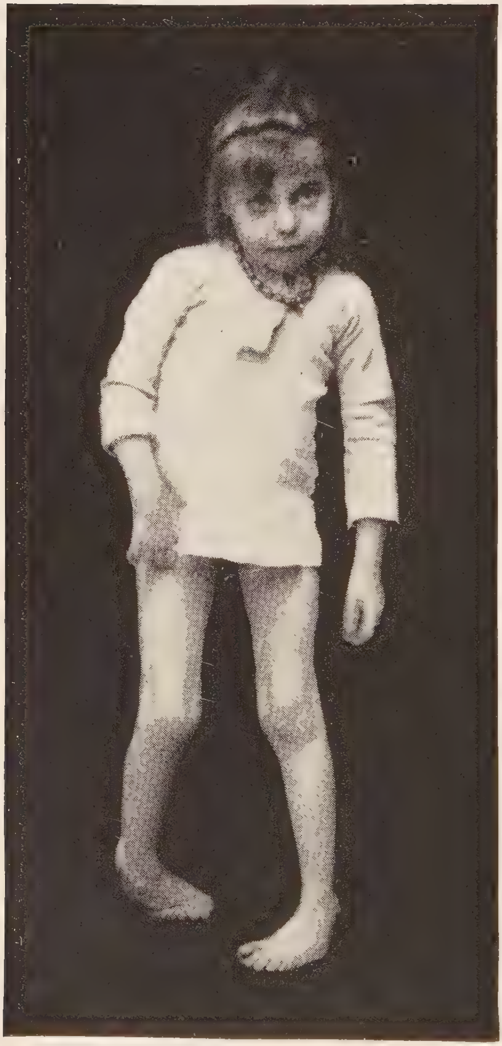
Between the conditions of cortical agenesis and idiopathic porencephalus I can see no points of distinction other than these. The cortical agenesis is apt to be a diffuse condition; hence idiocy and wide-spread paralyses.

Porencephalus may be limited to a definite area; and we may have extreme paralytic symptoms without any mental defect. But all these points have a tentative value only. It remains to be seen how far further autopsies will bear out the writer's reflections.

PROGNOSIS.

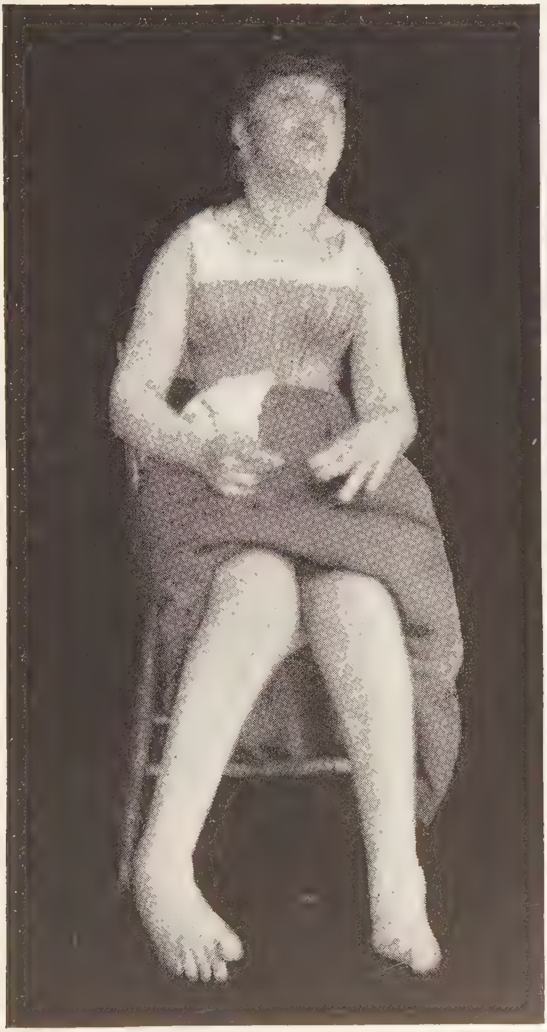
Under this heading it will be wiser to make a distinction between the congenital cases (including birth palsies) and the acute cerebral paralyses of children. In giving the prognosis of congenital affections, little can be said until the first few months have elapsed, except in instances in which the child is born deeply asphyxiated, has repeated convulsions, and breathes stertorously. Under such circumstances the gravest prognosis must be given, as death usually results within a few hours or at the utmost within a few days. As soon as regular respiration has been maintained for days, the prognosis *quoad vitam* is not bad. If there are no convulsions, there is good reason to think that the hemorrhage (for these are generally cases of hemorrhage) is not of unusual extent, and the possibility of the child's gaining fair use of its limbs is to be borne in mind. As regards the future development of the child's mind, absolutely nothing can be said within the first few months. If at the age of four or five months, or even up to the

PHOTO. I.



Case of Spastic Diplegia. Attempt to Walk. Cross-legged Progression.

PHOTO. II.



CASE OF CONGENITAL DIPLEGIA

age of nine months, the child begins to notice its surroundings, to play with things that are given it, complete idiocy will probably not ensue. In every case let the physician be extremely cautious in expressing his opinions, for he is apt to be surprised by unlooked-for changes in the child's progress. Anxious parents should be told that the brain will have the most favorable conditions for development if the child is carefully nourished, if the hygienic conditions are properly regulated, and if the parents themselves will refrain from putting such a brain to constant but useless tests. No amount of medication and no training in the earlier years will avail much in developing a defective or injured brain. The development of idiocy is the feature most to be feared. The fewer the convulsions, the earlier some outward signs of mental activity appear, the better the outlook in this respect.

In a large majority of cases, idiocy is developed in those cases in which paralysis is most extreme and convulsions most frequent. In some cases, however, of which I have seen some striking examples, marked paralytic troubles are associated with a normal development of mind. The case of J. O., and that of another little girl aged six years, whose photograph is reproduced in this article (Photos. I. and II.), are very much to the point. But of course I cannot speak with any certainty as to the exact morbid process in these two cases. From the table on p. 541 we may infer that, on the whole, the prospects of a normal development of mind are better in cases of hemiplegia than in cases of either diplegia or paraplegia. Striking exceptions to this rule are often found.

The prognosis of acute cerebral palsies must be given on entirely different lines. Prolonged coma, severe and oft-repeated convulsions, stertorous breathing, constitute the serious features of a given case, and death is apt to supervene unless a decided improvement in one or all of these symptoms takes place within a few days. As long as the child is in deep coma, it is safer to make the prognosis unusually grave. As soon as the coma diminishes, the danger of death is very much less, but the possibility of the child coming out of the comatose condition and relapsing into the same condition again should be borne in mind. In the initial stages of coma an inference as to the amount of paralysis or as to the side paralyzed can sometimes be made by examination of the extremities. In some instances, however, there may be little paralysis and yet the lesion may be a serious one, for it should be remembered that very large portions of the brain, particularly the frontal and occipital regions, may be involved without any resulting paralysis. The motor areas may have escaped, but if the general cerebral symptoms point to a severe lesion, there is all the more reason to fear that the mind will be impaired.

As soon as the child comes out of the comatose condition the prognosis as regards the amount of paralysis will be demanded of the physician. Here, again, it is well to be on one's guard, and to say that the sooner the movements return in any part of the body the greater the likelihood that the child will recover partial use of that part. As regards the future course of the disease, the physician must not be misled by his experience with cases of apoplexy in the adult, for the younger the child the more serious the results of an apoplectic stroke are apt to be. In my large experience I have seen very few cases in which permanent rigidities and contractures have not set in. I have given elsewhere the histories of one or two exceptional cases. The rigidities of the upper extremities are most frequent, and yet it is to be considered a very fortunate circumstance if a child that has had an apoplectic stroke ever regains an entirely normal walk. A very large percentage of the worst cases seen in orthopædic dispensaries and hospitals are due to early cerebral disease.

TREATMENT.

In the birth and truly congenital cases, treatment should be directed entirely to the general condition of the child. Constitutional defects should be remedied, and the ancestral history of the child carefully considered. Wherever there is the least suspicion of hereditary syphilis, antisyphilitic treatment should be instituted at as early a day as possible. Even in delicate children such treatment, by inunctions either of the gray ointment or of the oleate of mercury, drop by drop, is in order. In cases of premature birth, the well-known general rule of keeping the child wrapped up in cotton, or even in an incubator, should be applied. If the child is sufficiently developed to nurse at the breast, a good wet-nurse is superior to any form of medication. Cod-liver oil baths and small doses of the oil of phosphorus (a few drops daily) may be given, if parents demand that "something be done for the child." I have little faith, however, in the efficacy of either of these remedies, except possibly in those cases in which hydrocephalus and other symptoms of rickets are present.

Before leaving this subject, I repeat the caution given in a previous article, that the obstetrician do all in his power to prevent injury to the child's brain. Protracted labor being the greatest danger, a physician who is skilful in the use of the forceps should lose no time in applying them. A little hemorrhage from the temporary compression by the forceps is less dangerous than the venous congestion and extensive hemorrhage which are apt to occur if the child's brain has been compressed for hours within the narrow limits of a pelvic canal. As the child grows older, the treatment of secondary changes should be the same as in the acute cases, which we shall now consider.

In these acute cases the initial coma and convulsions call for immediate treatment. While it is not necessary nor desirable to arouse a comatose or semi-comatose child by the use of counter-irritants, vinegar injections into the rectum, and the like, it is absolutely imperative upon the physician to check convulsions, for the convulsions themselves are a source of great danger. The congestion during convulsions, the stertorous breathing, are the most favorable conditions for further and incalculable harm to the brain.

Much time is generally wasted through the old-fashioned mustard bath, blistering, and the like. It is far better, in case the physician reaches the child in time, to check the convulsions by the prompt use of inhalations of chloroform. A very few drops poured upon a handkerchief and placed at the child's nostrils are often sufficient. In every such case the physician should not leave the bedside until several hours after the initial convulsion, in order to use prompt measures as soon as the child shows the slightest twitchings in any part of the body. I have had an opportunity of testing this method in a number of cases, and have no reason to regret having used it.

As soon as the danger of convulsions is past, everything should be done to prevent a recurrence. I believe in the application of cold to the head, and in keeping the room in which the child lies absolutely dark and quiet. The child is not to be lifted from the bed or from the lap upon which it is lying, lest the mere change of position give rise to further trouble. These are the conditions under which bromides, which are generally so much abused, should be given. According to the age of the child, from five to ten grains of the bromide of sodium should be given in milk, or, if the child do not swallow, from five to ten or possibly more grains of the hydrate of chloral should be administered per rectum. Whatever one's practice may be with regard to adult cases, I consider the administration of ergot, and particularly of nitrite of amyl, very objectionable. If the pulse is extremely weak, the use of a drop of the fluid extract of digitalis or of a good infusion of digitalis or a drop or two of the tincture of strophanthus is very much to be preferred. There is no reason to depart from this medication for several weeks.

As soon as the acute symptoms have passed, and the paralysis has become established, the relief of this condition will come up for consideration. I have been in the habit of continuing the bromides, and with them the iodides, in small doses for the first weeks or months after an attack. The exact amount to be administered will depend upon the age and condition of the child. We must be guided by the effect of the bromides, and must use our judgment as to the amount of stupor that we are willing to develop in any given case. It is good practice, after the child has had bromides for some time, to lessen the amount gradually, and to diminish still further if there are no active symptoms; but I prefer to deepen or maintain a condition of semi-stupor in case the slightest twitching appears when the drug is reduced. The iodide is added as a sorbefacient; but both bromides and iodides should be discontinued as soon as the stomach becomes intolerant of them.

It is an important duty that devolves upon the physician to counteract the development of contractures, inasmuch as these depend upon the existence of secondary degenerations. It is doubtful whether anything is to be gained by active treatment. On the basis of practical experience, however, I favor the use of massage and faradic electricity. In this way the tendency to contractures may be partially overcome, and I have known old contractures to relax a little under the application of just these means. Certain it is that the condition of numerous muscles is very much improved by stimulating them into activity with the use of the electrical current. If the contractures do not yield to such treatment, the services of the orthopædic surgeon will be required. A number of cases with contractured arms and with various degrees of talipes equinus and equino-varus have done well under Dr. Gibney's hands, to whom I am indebted for a very large amount of my clinical material. I earnestly protest, however, against the application of any electrical current to the head for the purpose of diminishing the size of a clot or of improving cerebral circulation. Until we have more positive knowledge as regards the effect of a galvanic current upon the blood-vessels of the brain, it will be better not to experiment upon this delicate and diseased structure.

The treatment of the defective mind of a child is the most puzzling problem of all. As in the case of J. K., referred to on p. 542, a child that has had a normal mind preceding the attack may have to be re-educated, and often with good results. If there be idiocy or imbecility, careful training by a competent teacher, or placing the child in a fit school for feeble-minded children, will do some good, and, if the brain is doomed to remain below par, the youth may still be able to learn some trade and thus gain the means of subsistence. But, in spite of all that we may try, many of these patients will necessarily become the inmates of public institutions. In case the idiocy is associated with epilepsy, the treatment must be directed to the latter, and for this the reader is referred to the article on Epilepsy.

INTRACRANIAL TUMORS.

By M. ALLEN STARR, M.D.

In this article tumors of the brain, tumors of the meninges, and intracranial aneurisms are included.

Tumors of the brain and its coverings are not infrequent in childhood and youth, individuals below the age of nineteen being about as liable as those above that age.[1] The following table shows the varieties of tumor found within the cranium, the relative frequency of each variety in persons under nineteen, and the relative frequency of tumors in various parts of the brain. The collection of cases was made from the collections of Bernhardt[2] and Steffan,[3] duplicates being excluded, and from the journals, German, French, English, and American, of the past ten years.

TABLE I.—BRAIN-TUMORS.

Situation.	Tubercle.	Glioma.	Sarcoma.	Glio-Sarcoma.	Cyst.	Carcinoma.	Gumma.	Not Stated.	Total.
I. Cortex cerebri	13	6	1	..	..	1	..	..	21
II. Centrum ovale	6	1	5	1	15	1	1	5	35
III. Cerebral axis:									
1. Basal ganglia and lateral ventricle	14	3	5	..	1	1	..	3	27
2. Corpora quadrigemina and crura cerebri . .	16	1	3	..	..	..	..	1	21
3. Pons Varolii	19	10	5	2	1	..	..	1	38
4. Medulla oblongata . .	2	..	..	..	1	2	.	1	6
5. Fourth ventricle . . .	1	1	1	..	..	1	..	1	5
6. Base of brain	..	..	1	1	1	1	..	4	8
IV. Cerebellum	47	15	10	1	9	3	..	11	96
V. Multiple tumors	34	..	3	..	2	..	..	3	42
Total	152	37	34	5	30	10	1	30	299

The table shows that tubercular tumors are the kind most frequently found in young persons, that these are often multiple and affect all parts of the brain, but are most frequently located in the cerebellum and upon the base. They occupy the place in children held by gummata in adults. The next tumor in frequency is glioma, which is closely followed by sarcoma and by glio-sarcoma. Carcinoma, lipoma, myxoma, psammoma, and gumma are very rare in youth, and parasitic cysts, which appear to be frequently found in Germany, are quite exceptionally met with in this country. Since the diagnosis of the variety of tumor present in any case is a matter of inference and probability, such statistics have a certain diagnostic value.

PATHOLOGY.

I. Tubercular tumors, though occasionally appearing in the brain or meninges as the first evidence of tubercular disease, are commonly secondary to tubercular affection of the bronchial glands, lungs, mesenteric glands, and other organs; or to tubercular disease of the orbit, ear, or cranial bones. In over one-fifth of all cases tubercular tumors in the brain are multiple. Such tumors vary in size from a small collection of miliary tubercles to a large mass with hard cheesy centre and surrounding zones of tubercular infiltration and of congestion. They also vary in shape from a single round encapsulated neoplasm to a diffuse layer of tubercular deposit covering an extensive area of the surface. Very commonly several irregularly-shaped masses of tubercle lie on the base of the brain in the meshes of the pia mater, compressing the adjacent tissue and involving the cranial nerves. The infrequency with which tubercular tumors are found within the cerebral or cerebellar hemispheres indicates that it is especially upon the membranes of the brain that these tumors originate.[4] Starting from the vessels and lymphatics of the pia mater and invading the brain along the perivascular spaces, the tubercular cells and stroma so infiltrate both gray and white nervous tissue that their structure is altered and their function impaired. Finally there is formed a mass of tubercular tissue in which a few nerve cells and fibres are scattered. In other cases a distinct limiting layer surrounds the tubercular mass which is undergoing cheesy degeneration at its centre and is growing by accretion at its periphery, the brain being compressed and thus destroyed without being infiltrated with tubercle cells.

If the tumor is on the surface, the pia mater is thickened by a tubercular deposit and is adherent to the tumor; the dura mater may also be adherent; and in a few cases the cranial bones have been eroded. It must not be forgotten that a tubercular meningitis may develop secondarily to a tubercular tumor of the brain. The histological character of these various forms of tubercular tumor is to be found in text-books on pathology.[5, 6, 7]

The existence in the patient of hereditary tendency to tuberculosis, the history of symptoms pointing to tubercular disease, glandular, bronchial, or visceral, and the presence of local signs or general evidences of tuberculosis, should always be looked for in a child presenting any symptoms of brain-tumor; and in children who are tubercular the possible development of brain-tumor should be kept in mind.

II. Glioma is not uncommon in childhood. This tumor is a product of the neuroglia, and presents the appearance of a connective-tissue fibrillary net-work containing a greater or less number of small embryonal and of large branching cells, the latter being so-called spider-cells.[8] Gliomata vary in density, some being hard and separable from the brain-tissue like sarcomata, others being softer, of the consistence of the brain, and without definite limit, the tissue of the tumor shading off into the neuroglia of the brain. The greater the number of embryonal cells the softer the tumor.[9] They grow most frequently in the white substance of the brain, but sometimes develop in the gray matter, in almost any region; and as they increase in size they destroy the brain-tissue by extending around and between the nerve-cells and fibres. They grow less rapidly than sarcomata, and never involve the membranes. If a glioma is well defined it may undergo fatty degeneration and break down. In any case the vascular supply of a glioma is unusually rich, and hence symptoms referable to changes of circulation in the tumor, and hemorrhages within the tumor or in the adjacent brain-substance, are frequently observed. Glioma of the brain may develop secondarily to glioma of the retina, but is usually primary.

III. Sarcoma is next in frequency to glioma. Round-cell and spindle-cell sarcomata are met with more frequently than glio-sarcomata or myxo-sarcomata, and these forms are single. Multiple melano-sarcomata may occur in youth, but are very rare.[10] This form of tumor is well defined and easily separable from the brain-tissue, which is destroyed usually by compression, and sometimes also by an infiltration with sarcomatous cells. When a sarcoma infiltrates the brain, gliomatous cells are frequently found as well as sarcomatous cells, and then the condition is one of glio-sarcoma. Sarcomata of all sizes are found, but in shape they are usually round and nodular. They develop within the nervous tissue as well as in connection with the membranes, and are found as frequently in the white matter of the cerebral and cerebellar hemispheres, and in the gray matter of the basal ganglia, as on the surface of the brain. They grow rapidly, and therefore produce more marked symptoms than slowly-growing tumors.

IV. Cystic tumors of the brain, usually the parasitic cysts of echinococcus or cysticercus cellulosæ, are not infrequently found in children, but appear to be much more rarely met with in this country than on the continent of Europe.[11] Such cysts are found in all parts of the brain. They grow slowly, are usually latent, but occasionally produce symptoms by pressure. They never involve the brain-tissue directly. They are sometimes surrounded by a zone of softened or sclerotic brain-tissue. They rarely rupture. Küchenmeister[12] has collected eighty-eight cases of cysticercus, in all but thirteen of which multiple cysts were found. Of these, forty-nine were in the membranes, forty-one in the cortex, nineteen in the white substance, eighteen in the cerebellum, eighteen in the ventricles, seventeen in the basal ganglia, and a few in the corpora quadrigemina, pons, and medulla. These are not included in the table.

V. Carcinoma is very rarely found in persons under fifty years, is usually secondary to cancer elsewhere, and, being a tumor of extraordinary rarity in children, requires mention only. In about one-half of the cases on record below the age of twenty it was due to direct extension of the growth from the orbit.

VI. Gummata of the brain are so rare in childhood that Rumpf in his exhaustive work upon Syphilis of the Nervous System cites but two cases.[13] It is true that all forms of syphilitic disease have been observed in children as the result of inheritance or of syphilis acquired accidentally; and in any case of brain-tumor in which evidences of such disease are present the possibility of gumma must be considered. But the fact that in all literature gumma below the age of twelve and following inherited syphilis is almost unknown should lead to caution in making such a diagnosis. Cerebral syphilis in children has as its usual basis an endarteritis with thrombosis. Gumma as an evidence of tertiary syphilis is hardly to be expected in youth between the ages of twelve and eighteen, even though the individual may have acquired syphilis; and in fact the only case recorded in the table was in a young man aged eighteen. Gumma, therefore, must be considered as rare as carcinoma among children,—a fact of importance in view of its frequency in adults, and in view of the tendency to submit all cases of brain-tumor to specific treatment.

VII. The other varieties of brain-tumor—lipoma, papilloma, myxoma, psammoma, osteoma, cholesteatoma, and teratoma—are known to occur in children as well as in adults. They are, however, like tumors of the pituitary body and pineal gland, to be regarded as pathological curiosities, and their diagnosis from other forms of tumor cannot be made during life. For their pathological characteristics the reader is referred to text-books on pathology.

VIII. Intracranial aneurisms are rare in childhood, and are never very large. They appear upon the larger arteries of the base of the brain and on the Sylvian arteries. They are usually fusiform, occasionally round. They increase in size rather more rapidly than aneurisms elsewhere, and show a greater tendency to rupture. They produce symptoms by their pressure, and are occasionally to be diagnosticated by the pulsating headache or sensation accompanying them. Hemorrhages and thrombosis or embolism of smaller arteries are frequent complications of intracranial aneurisms.

The collateral effect of the presence of a tumor of any kind requires consideration. The pressure exerted by a new growth impairs the circulation in the surrounding brain-tissue, either directly by impeding the flow in the small vessels, or indirectly by compressing a large arterial trunk and thus causing wide-spread anæmia in its area of distribution. The resultant anæmia may be sufficient to impair the nutrition of the tissue and thus to produce suspension of its function. Pressure may after a time cause localized atrophy of the brain, in numerous cases the flattened and shrunken convolutions or the compressed white substance near the tumor having been found much smaller than the corresponding healthy parts on the opposite side. It may also result in white or yellow softening, a parenchymatous degeneration of the brain about the tumor. In some cases the tumor is surrounded by a zone of congested tissue, the tumor itself being vascular; and in some cases subacute encephalitis, with small or large hemorrhages, the so-called red softening, is found. This latter condition is especially frequent in the vicinity of gliomata and carcinomata. The pressure of a tumor may be exerted upon the veins and sinuses as well as upon the arteries, and this is indeed the more frequent condition. As a result, passive venous congestion and œdema of the brain are produced. This almost always occurs in tumors situated on the base of the brain and in the posterior cranial fossa. Tumors of the middle lobe of the cerebellum, of the corpora quadrigemina, and of the pons usually compress the venæ Galeni. The result is a serous effusion into the lateral ventricles of greater or less extent, causing their distention and consequent displacement outward of their walls and pressure upon the basal ganglia and adjacent parts. Hydrocephalus is a very frequent complication of brain-tumors in childhood, and is more easily detected in children below the age of six than in young persons or adults. When the cranial bones have not fully united, distention of the ventricles may cause a bulging at the fontanels and an enlargement of the head, and in infants this symptom is usually present with a brain-tumor. In any case of hydrocephalus the possibility of an intracranial tumor as a cause should be considered.

The collateral effects of tumors are not wholly dependent upon pressure. As already mentioned, an implication of the meninges in the growth, with the formation of adhesions, is frequent in tubercular and sarcomatous tumors; and such thickening of the membranes, if it occur on the convexity, may produce tenderness and localized pain in the skull; or, if it occur on the base, may result in compression of the cranial nerves.

Displacement of the brain-axis by a tumor anywhere often produces a stretching of intra-cerebral tracts or of cranial nerves, with corresponding impairment of their functions. Extension of a brain-tumor into the orbit, with displacement of the eyeball, has been recorded occasionally. And, lastly, brain-tumors have been known to erode the cranial bones and appear upon the surface.

Not infrequently in brain-tumor the immediate cause of death is hemorrhage from a diseased vessel in or near the tumor; and the possibility of such sudden apoplectic termination of the case must not be forgotten in giving a prognosis.

ETIOLOGY.

Few subjects are more obscure than the etiology of brain-tumors. As regards children, it is remarkable with what frequency a fall or a blow on the head is considered the cause by the friends. In not a small number the situation of the injury and that of the tumor coincide, a fact which seems to establish a causal relation. And yet the fact that few if any children escape such accidents, while the supposed effect is quite rare, throws doubt upon this relation. A causal relation is to be admitted as probable only when the blow or fall was severe, and its severity will be more certain if a scar remains. The result of the contusion may be to produce a local hyperæmia or capillary hemorrhages in the membranes or brain or in both, and this may be the starting-point for further pathological changes. Such changes must begin at once after the blow, even though the resulting tumor produce no symptoms for years. Thus, in a case reported by Keen the tumor removed (a fibroma) was thought to have been due to a blow received at the age of three and to have been growing for twenty-four years, though symptoms appeared but four years before its removal. It seems certain that, when a tendency to tubercular deposit is present, a blow on the head may influence the location of this deposit in the brain. Sarcoma of the membranes or brain, and even glioma, has been frequently found just beneath a scar of the scalp or a fissure of the cranial bones, or at the point just opposite to it on the other side, in which case it has been ascribed to *contrecoup;* and severe falls on the back of the head are mentioned in histories of cerebellar tumor too frequently to be merely accidental coincidences. There seems, therefore, to be no doubt that blows and falls on the head may cause intracranial tumors.

The majority of tubercular tumors occurring in childhood and youth are secondary to tuberculosis of other organs, and are therefore metastatic. Primary tubercle of the brain is very rare; some authors, indeed, deny its occurrence. Sarcoma and carcinoma have also been thought to be secondary in origin. And parasitic cysts are necessarily the result of the ingestion of infected food.

Extension of tumors of the scalp, orbit, ear, nose, and pharynx to the brain has been observed, but hardly requires discussion. Whether mental anxiety, intellectual overstrain in school, and fright have any causal relation to tumors is uncertain.

Boys are twice as liable as girls to brain-tumor. No age is exempt, a brain-tumor having been found in an infant four weeks old.

TABLE II.—AGE AT WHICH TUMORS ARE FOUND.

Under 1 year	7 cases.	10 years old	10 cases.
1 year old	8 “	11 “ “	12 “
2 years “	19 “	12 “ “	11 “
3 “ “	17 “	13 “ “	8 “
4 “ “	22 “	14 “ “	13 “
5 “ “	17 “	15 “ “	10 “
6 “ “	17 “	16 “ “	3 “
7 “ “	16 “	17 “ “	14 “
8 “ “	7 “	18 “ “	12 “
9 “ “	13 “	Total between 10 and 19	93 “
Total under 10 years	143 “		

As the table here given shows, children below the age of eight are somewhat more liable than those above that age.

SYMPTOMS.

The symptoms of intracranial tumors are very numerous, and may be divided into two classes,—general symptoms and local symptoms.

I. General Symptoms occur irrespective of the location of the new growth, and depend upon its rapidity of development, its size, its vascularity, and to some extent upon its pathological character.[14, 15, 16, 17] They vary in severity from time to time, probably in accordance with the activity of the pathological process going on in and about the tumor, and with the condition of blood-supply in the brain. Thus, when a tumor is increasing in size they may become severe, but disappear almost entirely when it becomes stationary. And they are decidedly affected by causes which alter the blood-supply: thus, in one case of my own, hot douches, either spinal or vaginal, increased the cerebral symptoms invariably, by producing cerebral anæmia. This is to be remembered in regulating the temperature of a child's bath in any case of suspected brain-tumor; and it offers an explanation of the therapeutic effect of hot mustard baths in arresting general convulsions.

The general symptoms of intracranial tumor are headache, general convulsions, changes in disposition and mental activity, optic neuritis without or with defects of vision, vomiting and vertigo, and insomnia.

Headache is the most prominent and constant symptom of brain-tumor. It varies in severity, but is in all cases at times very severe. It may be intermittent, and is usually excited by any exertion or mental effort, and it recurs with quite marked periodicity, and with increasing frequency as the tumor grows. It may be a dull, heavy, continuous pain, with sharp paroxysmal exacerbations, during which the patient is incapable of controlling his agony. It varies in its location, is usually frontal or occipital, and only occasionally over the seat of the disease. The general increase of intracranial pressure and the consequent stretching of the membranes, the variations in cerebral circulation, the existence of effusion into the ventricles, or the direct involvement of the sensitive dura mater and branches of the fifth nerve, are mentioned as causes of the headache. Such effects of the presence of a tumor are more likely to follow when the new growth is in the posterior cranial fossa beneath the tentorium cerebelli, and in such cases headache is most constant and most severe. But even in these cases it is almost as frequently referred to the forehead or to the temples as to the back of the head: thus, in a patient of Taylor's, a boy of nine, the pain was referred to the frontal region and vertex, while the tumor, a glioma, was found in the pons and cerebellum. If, however, the pain is constantly located in the occipital region, the tumor is probably in the posterior fossa.

The pain is often associated with indefinite cerebral sensations, described as fulness, pressure, confusion, tightness, as if a band were drawn about the forehead; and these give great distress and often interfere with sleep. In infants the existence of headache may be inferred from constant motion of the head, from movements of the hands grasping the head or pulling the hair, and from sudden outbursts of screaming without other ascertainable cause. Headache is probably less severely felt in cases of tumor in infants, as the expansion of the skull and the opening of the sutures prevent the extreme degree of intracranial pressure to which severe headache is chiefly ascribed.

Tenderness of the scalp and head is sometimes found, and if this is limited uniformly to one area, is always elicited by percussion, and is not due to sensitiveness of a single nerve-trunk, it is a more valuable sign of the location of a tumor than is a localized headache. It also awakens a suspicion that the tumor lies just beneath the bone, upon the surface of the brain, for it is more commonly found in cortical than in subcortical tumors. And under these circumstances it is usually associated with headache localized in the same area.

General convulsions are the next most frequent symptom of brain-tumor. The nervous instability of childhood, the lack of inhibition, and undue sensitiveness to peripheral irritation of any sort, predispose children to the occurrence of that general discharge of nerve-energy which causes convulsions. They begin early in the history of the intracranial growth, occurring at long intervals, then become more frequent, as many as twenty or thirty occurring in a day, and they may persist at short intervals through several days. They may be slight in degree, a little twitching of the face and eyes, with stiffening of the back and extremities, and with loss of consciousness lasting only a few seconds, constituting the attack ; or they may have all the stages of a regular epileptic seizure followed by coma. Sometimes a peculiar general tremor follows the attack and lasts an hour or more. Convulsions are usually indicative of rapid progress in the new growth, of effusion into the ventricles, or of a secondary affection of the meninges. They may occur from tumor situated anywhere, and do not point to a special seat of the disease. They may follow a local spasm which has gradually extended from a single limb to other parts. Their significance under these circumstances will be discussed under Local Symptoms. Death not infrequently occurs in convulsions, but this is more frequently observed in meningitis than in intracranial tumor, which usually produces a fatal termination by paralysis of respiration.

Changes of disposition and in mental activity occur independently of headache, and are noticed as an early symptom of intracranial tumor in children.[18, 19] The child becomes unusually fretful and irritable, refuses to notice its toys or to play, or, if it does so, soon becomes wearied, and requires constant attention. It may become indifferent to things in which it was formerly interested, may prefer to lie down and to keep quiet, in a manner unnatural to a healthy child, and may even become somnolent and lethargic, or it may from time to time be very irritable, may cry and scream without apparent cause, and act as if much alarmed, refusing to be quieted by ordinary means, and hence be subjected to undeserved punishment.

These changes in disposition should be considered as symptoms of mental distress, and, taken in connection with headache and convulsions, should excite concern rather than reproof. In an extreme degree these symptoms may appear as imbecility or as maniacal excitement, but this is very rare.

In older children a decided change in mental activity is noticed, as well as change of disposition. Forgetfulness, dulness, lack of interest, stupidity, are noticed, and the child no longer keeps up in school with its fellows or notices what is going on about it at home. This condition may develop into melancholia with or without hallucinations, or it may suddenly change into mania with periods of excitement, though these manifestations are rare. More frequently a state of apathy ensues, which is like a dementia, in which the child disregards the wants of nature and is so indifferent as to forget the restraints of decency.

Double optic neuritis and *optic nerve-atrophy* are very important diagnostic symptoms of intracranial tumor. Neuritis is usually associated with other signs of increased intracranial pressure, but may occur without such pressure. It is present in eighty per cent. of the cases, and should be looked for in every case which presents cerebral symptoms. A marked degree of optic neuritis may exist without any impairment of vision: hence the ophthalmoscope should be used whether the defect of sight is present or not. But when the patient shows impairment of visual power, or limitation of the visual field for colors or for light, or is becoming blind, it will be found that optic neuritis or optic atrophy is fully developed. Sudden loss of vision appears to be more commonly noted in histories of children's cases than in those of adults, possibly because a gradual loss is not detected. It is true that hydrocephalus may cause choked disks, and hence from this symptom alone a tumor cannot be diagnosticated. But in cases where the diagnosis is difficult no more important objective evidence of brain-tumor can be found.

Tumors of the cerebellum and corpora quadrigemina and tumors upon the base of the brain and in the basal ganglia produce optic neuritis more constantly and earlier in their course than tumors situated in the cortex or centrum ovale. Optic neuritis is usually double, though it always appears first in one eye and is rarely equally intense in both eyes; but in a few cases it has been found in one eye only, and then is thought to indicate disease of the nerve in the orbit or in front of the optic chiasm rather than a distant tumor. For the exact changes in the retina and for the pathological causes of optic neuritis the reader is referred to special text-books.[20]

Vomiting is a symptom of brain-tumor more frequently observed in children than in adults. It may or may not be accompanied by nausea. It may occur unexpectedly, without special relation to the time of meals, or it may be so continuous as to threaten inanition. It occurs not infrequently on any movement of the head after the patient has been confined to bed for some time, and then it is usually associated with vertigo. It also frequently accompanies severe headache.

Vertigo is sometimes a coincident symptom, but usually occurs independently of vomiting. The patient feels dizzy, feels himself turning or falling, and things about him appear to be in motion. He grasps at near objects for support, covers his eyes with his hands, or lies down on the floor and cries out with bewilderment and distress. Like vomiting, vertigo may be excited by changes of position. It occurs at intervals, in attacks of short or long duration. It occurs more frequently with tumors in the posterior fossa, in the cerebellum, or on the base involving the auditory nerve, than with tumors elsewhere. Such attacks of vertigo are not to be confounded with the slight constant vertigo due to double vision and secondary to paresis of the third and sixth nerves.

Insomnia may be due to disturbances of the cerebral circulation, or to the intensity of the other general symptoms, and is much more rarely complained of in cases of tumor in children and youth than in adults suffering from syphilitic tumors.

Fever and changes in the rapidity and rhythm of the pulse have been observed in the course of brain-tumors. The former is ascribed to inflammatory changes in the brain or meninges as a complication. The latter is regarded as evidence of increased intracranial pressure. Slow and irregular pulse is the form usually noted, but towards the close of life very rapid pulse has been observed. Irregular or Cheyne-Stokes respiration has also been noticed as a terminal symptom.

Occasionally attacks of syncope occur in patients with tumor of the posterior fossa, and a general feeling of weakness is not infrequently complained of.

The combination of several of these general symptoms in any case should lead to a strong suspicion of intracranial tumor. When, in addition, local symptoms are added, the diagnosis should not be difficult. In all cases the onset of the general symptoms is gradual, their intensity increasing as time goes on; and a careful study of their mode of development and progress is a great aid in the diagnosis of the kind of tumor present, of its size, and of the rapidity of its growth.

The distress produced by the general symptoms is usually much greater than that caused by the local symptoms, and consequently treatment is often required for their alleviation.

II. LOCAL SYMPTOMS depend upon the situation of the intracranial growth, irrespective of its character, and differ from one another in different cases, but are always the same when the tumor is in one place. They may be entirely wanting, and, as a rule, but a few occur in any one case. They usually commence gradually, and are limited, but increase in extent as the tumor grows. If they begin suddenly, they indicate the occurrence of hemorrhage in the tumor. If the tumor affects one hemisphere of the cerebrum or cerebellum only, the local symptoms are unilateral. If it lies upon the base and invades the cerebral axis, through which the tracts between the brain and the body pass, the local symptoms may be bilateral or irregular in their distribution. The significance of a local symptom as indicative of the seat of disease is the same whatever the form of lesion, whether tumor, abscess, hemorrhage, or local softening from embolism or thrombosis. The reader is therefore referred to the articles upon the localization of brain-lesions for their full consideration. They will be studied here in connection with tumors affecting various regions of the brain.

The local symptoms are spasm, tremor, and paralysis; alteration in reflex action; weariness, tingling, numbness, formication, feelings of heat and cold; pain; impairment of touch, temperature, pain, and muscular senses; ataxia; disturbances of the senses, either in the form of hallucinations or defective perception, which in the case of sight is in the form of hemianopsia; disturbance in the movements of the eyes and in the preservation of equilibrium; imperfect gait; interference with the acts of swallowing, respiration, and articulation; vaso-motor disturbances, polyuria, and glycosuria; disturbance in the mental action and in the memory and use of language; and symptoms referable to one or more of the cranial nerves.

A distinction is made between direct and indirect local symptoms, the first being due to irritation or destruction of a limited area by the tumor, the second being due to interference with the function of an area by disease at a distance from it which impairs its circulation or causes pressure upon it. Thus, a tumor of the cerebellum may cause incoördination and vertigo as direct symptoms, and also cause paralysis of the sixth and seventh cranial nerves as an indirect symptom of displacement of the pons Varolii to one side, which stretches these nerves unduly. Tumors usually cause both forms of local symptom, and therefore much care must be given to the question of their significance in any case.

I. Tumors involving the Cortex of the Cerebral Hemispheres.—These tumors are quite common, as is shown in Table I. They may be of any variety, but tubercle, sarcoma, and glioma are the more frequent. The two former usually involve the membranes as well as the cortex. They are more frequently traceable to blows and falls than tumors in other regions. Tumors of the cortex usually involve the white matter beneath the cortex in the centrum semiovale, through which pass the tracts connecting the cortex with the various organs of the body and the tracts between various cortical areas. Tumors may begin in this white matter and involve the cortex secondarily. It is at present impossible to diagnosticate a cortical from a subcortical tumor with certainty. Therefore in this section no distinction between them is attempted.

Tumors of the cortex produce headache, tenderness of the skull, general convulsions, and mental symptoms quite uniformly. Optic neuritis, vomiting, and vertigo are less frequently observed than with tumors of other regions. It is the function of the cerebral cortex to receive impressions coming in from the various organs and surfaces of the body as conscious perceptions, to preserve them so that they can be revived in memory or recognized upon their recurrence, to associate the perceptions and memories in groups which thus form the concept, and by a combination of concepts to build up ideas and carry on thought. It is also its function to initiate the voluntary impulses to which the thought leads, to co-ordinate them into orderly successive movements of definite intensity, and to give utterance to the thought in speech or writing. Its highest function is to exercise a control over the instinctive or impulsive tendencies to action, and to regulate conduct in view of remembered ideas rather than in response to present impressions. There is every reason to believe that the highest mental processes—judgment, reason, and the understanding of ethical and philosophical truth—are dependent upon the integrity of the cerebral cortex, since deficiency in its development impairs these mental powers; but any physical explanation of these psychical processes is as yet impossible.

The local symptoms of cortical tumors vary greatly, according to the area which is involved.

1. *Tumors of the frontal lobes* present few characteristic symptoms, and may be latent. If the tumor is situated near the orbital convolutions, it may destroy the olfactory tract and then cause loss of smell on the side affected. Lesions elsewhere in the frontal convolutions often produce a disturbance of mental action, shown by inability to concentrate the attention, to think connectedly, to learn easily, and to exercise self-control. This may go on to a state of imbecility, in which the patient may become dirty and disregard all restraints of decency. Integrity of the frontal lobes is necessary to complete mental action, yet no special loss of mental faculty can be said to result from their destruction.[21] Nor is there any apparent way to detect from the mental symptoms in which hemisphere the tumor lies. The diagnosis of tumor of the frontal lobes is therefore rarely made from a study of direct local symptoms. It is to be remembered, however, that a tumor when situated in other regions, excepting only the temporo-sphenoidal lobe, produces direct local symptoms, the absence of which may lead to the suspicion that the tumor is situated in the frontal or temporo-sphenoidal lobe.

Indirect local symptoms of irritation of the cortex often occur in tumors of the frontal convolutions.[22] Such irritation beginning in the frontal convolutions extends backward, and when it reaches the anterior central convolution (ascending frontal or pre-Rolandic convolution, Fig. 1, A), in which lie the motor centres, it may cause spasms or convulsions. Such spasm will begin in the eyes or face, in the arm, or in the leg of the side of the body opposite to the site of irritation, and is never followed by permanent paralysis. It may even go on to a general convulsion, extending from part to part and limb to limb till the entire body is involved. The occurrence of such spasms, not followed by permanent paralysis, is an indirect local symptom of frontal tumor. Should the tumor in its growth extend to the anterior central convolution, direct symptoms of its lesion will appear.

Tumors situated in the third frontal convolution of the left hemisphere in right-handed persons and of the right hemisphere in left-handed persons produce motor aphasia with, or possibly without, agraphia,—that is, a condition of inability to use language, to speak and to write, while the comprehension of language is unaffected. (See Fig. 2.) This aphasia is usually incomplete in cases of tumor, and is of slow onset, thus contrasting with the same symptom when due to embolism or hemorrhage. In children speech and writing are usually lost together, although these processes are doubtless distinct and become more independent as age advances.

The following cases are cited as examples :

Case I. (C. K. Mills: Journal of Mental and Nervous Diseases, December, 1887, xiv. 707.)—After a blow on the head, the boy, aged sixteen, had occasional convulsions for a year. He then began to suffer from headache, noises in his head, vomiting, and obstinate constipation, and his pulse was found to be slow. Then, quite suddenly, staggering gait, vertigo, diplopia, and severe pain in the head, with vomiting, developed. During the following week paræsthesiæ of the limbs, with stiffness, began, and he lost power to some extent in the left arm. His convulsions became more frequent, and during his fits he was drawn to the left side. He was ravenously hungry. Before his death, which occurred from exhaustion about one year after the onset of the convulsions, ptosis of the right eye and partial facial paralysis of the left side were present. Autopsy showed the presence of a reddish-gray tumor confined to the posterior portion of the right second frontal convolution, measuring about two and a half inches in all directions, involving the cortex and subjacent white matter.

In such a case the general symptoms clearly indicated a cerebral tumor, but no diagnosis of its location could have been made with certainty. The symptoms of paralysis in the left arm, coming on late, indicated a secondary involvement of the motor area. The symptoms of cranial nerve-disturbance showed a displacement of the entire brain-axis.

Case II. (Archer: Dublin Medical Journal, 1878, ii.)—A female, eleven years old, who had chronic hip-joint-disease, suffered for some months before her death from headache and general epileptic convulsions without local spasms or paralysis. She gradually became more and more stupid, and finally died of exhaustion. At the autopsy a tubercular tumor was found in the first and second frontal convolutions on the left side.

Occasionally all symptoms of brain-tumor are absent until a few hours before death, as in cases of Schweinitz * and of Janeway,† and in some patients the discovery of a tumor at the autopsy is a surprise, as in a case of Steiner. ‡

2. *Tumors involving the anterior and posterior central convolutions or the paracentral lobule,* which border the fissure of Rolando, produce, as direct local symptoms, spasm and paralysis in the limbs of the opposite side.[23, 24] The location of these motor symptoms varies with the site of the tumor in the motor area.[25, 26] Tumors usually produce irritation of the cortex before

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* Medical News, vol. li. No. 9.

† Transactions of the New York Academy of Medicine, iii. 187.

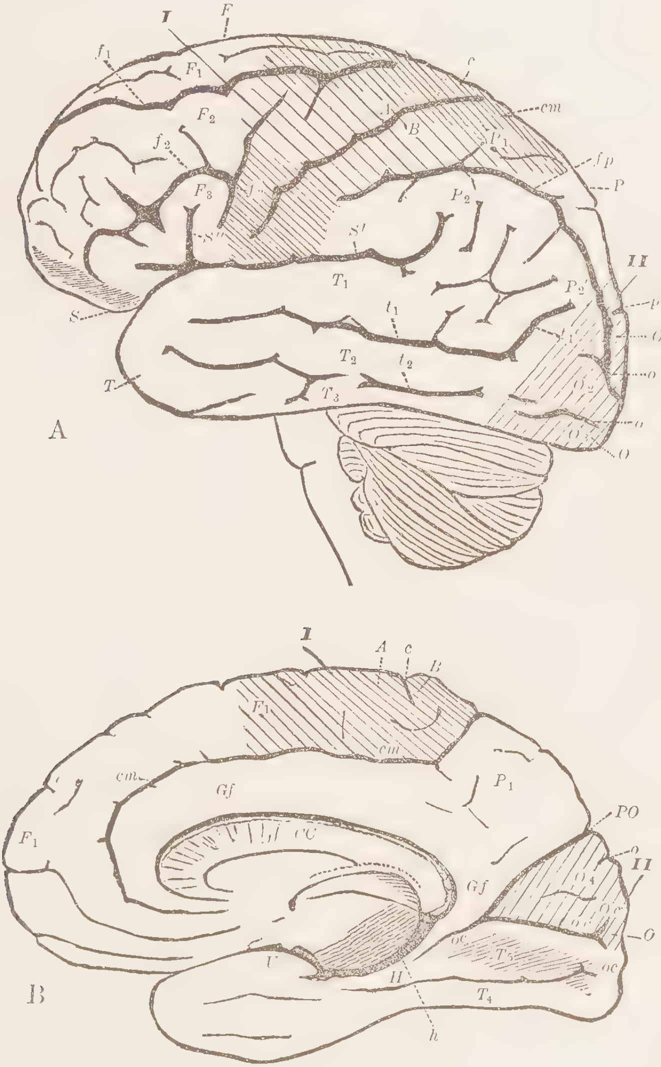
‡ Prager Vierteljahrschrift, Bd. lxxviii.

they cause its destruction: hence a patient may have attacks of local spasms (Jacksonian epilepsy) for some time before permanent paralysis develops.[27] If the paralysis develops first, it is probable that the tumor lies in the centrum semiovale under the motor area and has affected the motor tract, and has invaded the cortex subsequently, such invasion being indicated by the onset of the convulsions.[23] Local spasms are usually preceded or accompanied by numbness or slight anæsthesia of the part affected,[28, 29] and are followed by a condition of weakness. If this weakness is only temporary, the cortical motor area is not destroyed; but if it is permanent, it is an important indication that a serious lesion as well as a source of irritation is present. Slight anæsthesia and ataxia may accompany permanent paresis from cortical disease, but are not invariably found. They are much more common with tumors of the posterior central convolution which invade the parietal convolutions than with tumors affecting the anterior central convolution alone,[28, 29] yet the motor area and the sensory area for tactile sense probably coincide. Since local spasms and local paralysis are important indications of the exact situation of a tumor and may be used as guides to an operation for its removal, a careful consideration of their distribution and manner of extension is warranted.

The motor area of the brain is indicated by the shading in the diagram on the opposite page, and may be roughly divided into thirds as there shown. The lower third contains the centres which control the face; the middle third contains the centres which control the arm; the upper third contains the centres which control the leg. Horsley and Schaefer have located certain special movements in various sections of each area.[30] In the face area, irritation of the anterior third causes movements in the vocal cord, together with associated movements of other parts of the throat; irritation in the middle third produces movements of the upper part of the face and angle of the mouth; irritation of the lower and posterior third causes movements of the lower jaw and lip and of the tongue. Each of these areas blends with the others, there being no sharp line of demarcation between them. A very slight irritation in one area causes a spasm limited to the movements which it controls; a stronger irritation will cause movements in the muscles controlled by the other adjacent areas, due possibly to extension of the irritation to them, and possibly to a simultaneous representation of all the movements in less degree in one area. Similar distinctions in the effect of irritation are observed in the middle third of the motor area, different sections of this area governing different segments of the upper extremity. The shoulder is controlled in the upper and anterior part of this area, the elbow next behind and below, the wrist next below and posteriorly, the fingers next below and posteriorly, the thumb lowest and farthest back. The representation for the fingers and thumb is wider than that for the elbow and wrist, as the latter are rarely moved alone.

The upper third of the central area, including not only the paracentral lobule with its cortical portion on the median surface of the brain, but also parts of the cortex in front and behind it (as indicated in the diagram), governs the movements of the leg. The anterior division of this area, including the posterior part of the superior frontal convolution, presides over combined movements of the arm and leg, such as are made in climbing or

FIG. 1.



CORTICAL SENSORY-MOTOR AREAS AND VISUAL AREAS. A, convex surface of left hemisphere of the brain; B, median surface of right hemisphere of the brain. (Ecker.)—F_1, F_2, F_3, frontal convolutions; A, B, anterior and posterior central convolutions; P_1, P_2, superior and inferior parietal lobules; P_2', angular gyrus; O_1, O_2, O_3, occipital convolutions; O_4, cuneus; T_1, T_2, T_3, T_4, temporal convolutions; *Gf*, gyrus fornicatus; *U*, uncinate gyrus; *CC*, corpus callosum; *S*, fissure of Sylvius; *c*, fissure of Rolando; *cm*, calloso-marginal fissure; *po*, parieto-occipital sulcus. Lines drawn parallel to *I* indicate the motor area of the brain, and the difference in shading shows the division of this area into lower, middle, and upper thirds on the convexity, and the extent of motor area on the median surface. Lines drawn parallel to *II* indicate the visual area of the brain on the convexity and median surface.

swimming. The posterior part governs movements of the toes, and between these extremes the centres for the hip-, knee-, and ankle-movements are arranged, in the order named, from before backward. On the median surface of the hemisphere in the paracentral lobule and adjacent parts the muscles of the trunk are represented.

The motor centres for the motion of the head and eyes are placed by English physiologists[31] and by Munk[32] in the two upper frontal convolutions, just at their junction with the motor area, and by Landouzy and Wernicke[33] in the inferior parietal lobule, just at its juncture with the motor area. Pathological cases in man support the latter position more fully than the former.

When any irritation is sufficient to radiate from one area to another, it involves the areas in the order in which they lie. This is readily understood if we consider an irritation of the cortex as a nervous discharge which spreads, like the ripple on a pool, from the point disturbed outward in widening circles. If the point of irritation is in the lower third of the motor area, causing facial spasm, it will extend to the middle area, causing spasm of the arm, before it reaches the upper third and produces spasm of the leg. Spasms of the leg radiate to the trunk and arm before reaching the face. Spasm of the arm extends to the face and leg together. The same is true of the extension of paralysis from cortical destruction. And the order of extension of the paralysis is of especial importance as indicating the direction of growth of a tumor.

Irritation of the cortex by tumors is quite comparable in its effect with irritation produced by electricity in physiological experiments. In both, the manner of extension of the irritation from a certain area to other areas is the same.[21] It is therefore of very great importance to determine in a case of local spasm or paralysis the manner of onset and the order of extension of the symptoms. From these facts the exact point of irritation of the cortex can be deduced, and that point may then be taken as a guide to operation. Thus, for example, a spasm beginning in the thumb, extending to the fingers, wrist, elbow, and shoulder, and possibly to the face or to the leg, indicates an irritation whose point of origin was in the posterior part of the middle third of the motor area, in the posterior central convolution.[24] From this point of origin the irritation may be thought to have spread, thus invading the adjacent areas in a definite order, which order is indicated by the successive implication of various sets of muscles in the spasm.

The subjective sensations of the patient are also of value in determining the point of origin of the spasm, as tingling and numbness are felt first in the part in which the spasm begins,—*i.e.*, in the case supposed, in the thumb and fingers. This sensation has been termed by Seguin the "*signal symptom*" of a Jacksonian spasm. And subsequently to such a spasm anæsthesia and weakness will remain in the thumb and fingers longer than in the rest of the arm. In such a case an operation would be directed to the removal of a tumor from the middle third of the posterior central convolution, and that point would be made the centre of the trephined opening of the skull, as in a case successfully operated upon by Keen.[24] Hence in determining the location of a tumor in the motor area three things are to be observed: *first*, the starting-point of the sensory and motor symptoms in case of spasm or paralysis; *secondly*, the order of extension of the spasm or paralysis from the starting-point to other parts; *thirdly*, the point of greatest intensity of the post-spasmodic paresis and anæsthesia. These three sets of symptoms together will indicate the part of the motor area primarily affected, and the tumor will be found there.

Since the motor areas of the different limbs are separated from one another, it is not surprising that it is the rule to find in cases of tumor that paralysis is for a time at the outset limited to one limb. Monoplegia therefore, rather than hemiplegia, is to be expected in tumors of the cortex. When, however, the tumor begins deep in the centrum ovale, or lies in the basal ganglia or crus or pons or medulla, through all of which parts the motor tract containing fibres from the separate motor areas passes, it will involve this tract, and thus cause hemiplegia instead of monoplegia.

Whatever the form of the paralysis at the outset, the gradual extension of the tumor in the motor area results finally in hemiplegia; for in its lateral growth it either invades all the motor areas of the cortex, or in its downward growth it eventually involves the motor tract within the centrum ovale or in the internal capsule.

In all cases of cerebral paralysis there is an increase of the deep reflexes in the paralyzed part; there is no atrophy, or only slight atrophy from disuse, which is uniform in all the muscles of the limb; and there is no change in the electric reactions. There should therefore be no difficulty in differentiating a cerebral paralysis, even of the monoplegic type, from a spinal or nerve-trunk paralysis, even in infancy. Nor can the slight ataxia and impaired tactile sense which may accompany cortical paralysis mislead; for the anæsthesia is never limited in its distribution to the area of skin supplied by a single nerve or by a single spinal segment, but is usually uniform over the entire limb, or else segmental,—*i.e.*, it involves the digits, or the entire hand, or the entire forearm and hand, or the entire arm, forearm, and hand, and has not the distribution characteristic of spinal lesion.[34] Horsley has suggested that tactile sense, muscular sense, and motion must necessarily be represented together in the same cortical area, since all fine movements depend for their proper adaptation and co-ordination on these sensations; and he holds that the various layers of cells found in the cortex subserve various functions.[35] Recent pathological evidence lends much support to this view. Hence ataxia and anæsthesia should be looked for in cases of cortical paralysis.

The following cases are cited in illustration of tumors of the central area:

Case III. (Janeway: Transactions of the New York Academy of Medicine, iii. 184.)—Male, aged thirteen, began to suffer, two weeks after a fall, from headache, weakness in the left arm and leg, and internal strabismus of the left eye. Soon after vomiting began, and twitching of the muscles of the left shoulder, arm, and hand commenced. Dimness of vision, due to optic neuritis, developed in the fourth month, and six weeks later he was totally blind. Being examined at that time by Dr. Janeway, it was found that smell was impaired in the right nostril, hearing reduced one-half in the left ear, motions of the left arm and leg were slow, stiff, uncertain, and weak; sensibility was intact, but he suffered from pain in the left leg; the face was not affected. During the next eight months all these symptoms increased in severity, but no new ones were added. He then died.

Autopsy.—Membranes normal; convolutions flattened. A portion of the tumor appeared in the right posterior central convolution near the longitudinal fissure, while the middle half of the convolution was cyst-like from softening. On section, the tumor was found to affect the anterior and posterior central convolutions in their upper two-thirds, and to comprise and partly destroy the first frontal convolution, paracentral lobule, gyrus fornicatus, and superior parietal lobule. It was a firm gray tumor, lobular, of the size of an orange. It was a glioma.

Case IV. (Osler: American Journal of the Medical Sciences, 1885, i. 31.)—A female, aged fifteen, had an injury to the head when an infant, and seemed to suffer much after it for some weeks. Five months later spasms of a tonic character began in the left hand, and in the course of three months had extended to the leg, and then became unilateral and finally general, the mouth being generally fixed open during the spasms, which occurred as often as eight or ten in an hour. There was never loss of consciousness. These lasted two months, and then all symptoms ceased for a year, during which she was well. The spasms recurred one year later, lasted seven months, and again disappeared. Six years after this a gradually-increasing weakness of the left leg, with turning inward of the foot, developed, and the spasms began again, each spasm lasting half a minute or a minute, during which she would laugh and talk. They varied in frequency from that time until her death. She was a well-nourished, intelligent child. The only permanent symptoms were a slight wasting of the limbs of the left side and a turning of the left foot inward at a right angle to the leg. The left arm, though feeble, was not stiff and useless, as was the leg. Autopsy showed a glioma in the upper third of the anterior central convolution on the right side, in the white matter just under the cortex, measuring about one-half inch in all directions.

Case V. (Saundby: Transactions of the London Pathological Society, 1886, p. 39).—A boy, six years old, suffered for some weeks from general convulsions, mental dulness, and symptoms of nephritis. Then spasms of the right side without loss of consciousness began, and continued at intervals for five days, and were followed by partial paralysis of the right arm, with some jerky movements. The paralysis increased and extended to the right face and to the right leg. The boy died four weeks later. The autopsy showed a tubercular tumor on the surface of the left cerebral hemisphere in the motor area at the juncture of the middle and upper thirds.

Remarks.—In all of these cases the diagnosis of the existence of a tumor was possible, and also a diagnosis of its location in the motor area. With our present knowledge of the possibility of extirpating such tumors, an operation in a similar case would be indicated.

Cases of a similar character have been reported by Seeligmüller,* Sodermark,† Gliky,‡ Duchek,§ and Conner.‖

3. *Tumors in the parietal lobe of the brain* may produce no local symptoms, but usually cause disturbances of muscular sense, of tactile sense, and of the senses of temperature and pain.[28] The localization of these sensations

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* Jahrbuch f. Kinderheilkunde, xiii. 322. † Ibid., xxiii. 462.

‡ Deutsches Archiv für Klinische Medicin, xvi. 463.

§ Wiener Medicinische Jahrbuch, xxi. 1, 110.

‖ American Journal of the Medical Sciences, 1884, ii. 119, Case LXV.

in the cortex is still under dispute, but recent observations[29] seem to show that they are perceived in the central convolutions and parietal lobe, since injury there causes paræsthesia and partial anæsthesia in the limbs and face of the opposite side. This is not invariable, however, and a number of cases of lesion of this area have not been diagnosticated. The indirect local symptoms of irritation radiating to the central area and producing spasms, or of destruction invading the central area and causing paralysis, are all the more important. Destruction of the white tracts in the centrum semiovale under the parietal cortex may produce hemianopsia by invading the visual tract, and hence this too is an important indirect local symptom of tumor, especially if it is of slow onset. When a tumor exists in the inferior parietal lobule of the left hemisphere, it gives rise to disturbances of speech of a peculiar kind, called word-blindness. This is especially found in lesions of the angular gyrus and supra-marginal convolution and of the cortex between them. It consists in an inability to recognize written and printed words formerly understood. The patient thus loses his power to read and to write spontaneously, though he may be able to write at dictation or to copy. This symptom is often associated with word-deafness, but only when the first temporal convolution is invaded by disease. It is as yet impossible to say whether word-blindness is due to the destruction of the parietal cortex, or of association tracts between the occipital and temporal cortex which pass through the inferior parietal lobule. Word-blindness, however, must be considered a valuable sign of tumor or other lesion in this area. Like motor aphasia, it will be of gradual onset and incomplete in cases of tumor. Motor or ataxic aphasia, in which the patient, though able to understand what is said and to read what is seen, is unable to talk or to write, is due to a lesion in the posterior part of the third frontal convolution,—*i.e.*, in Broca's centre.

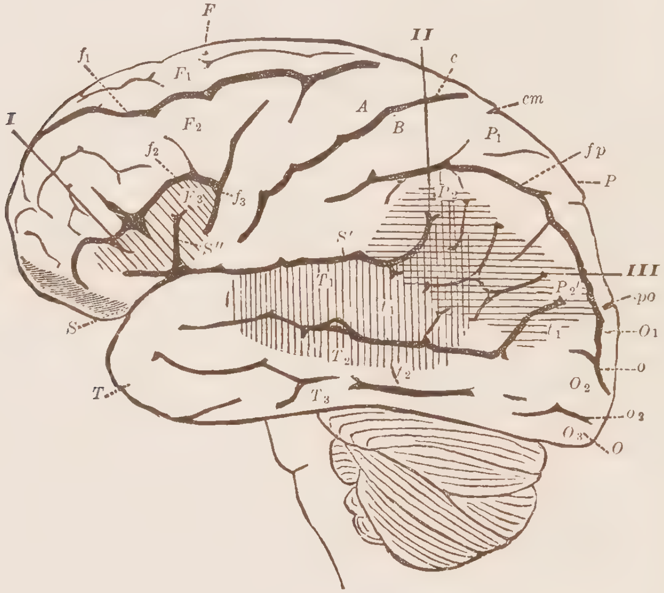
The figure on the following page shows the areas of the cortex concerned in the use of language.

The conjugate voluntary motions of the eyes are thought by Wernicke[33] to be controlled by centres in the inferior parietal lobule, and hence, should they be found to be impaired, a suspicion of disease in this region might be aroused. The impulses from the right inferior parietal lobule turn the eyes to the left. Thomson has reported[36] a case of depressed fracture of the left parietal bone just beneath the boss in a boy of fourteen, in which the only local symptom was conjugate deviation of the eyes to the right, which was entirely relieved by elevation of the bone by trephining.

4. *Tumors of the occipital lobe*, including the convolutions of the convexity and the cortex of the cuneus, produce bilateral homonymous hemianopsia on the side opposite the lesion ; that is, a lesion in the right occipital lobe produces blindness in the left half of both eyes, the patient seeing nothing which lies to the left of a vertical line directly in front of him.[37, 38] Central vision is usually preserved. If the tumor lies in the left hemisphere, there may develop, in addition to the hemianopsia, a condition known as

psychical blindness, in which a patient no longer recognizes faces or objects formerly familiar to him, and this is usually associated with word-blindness. In left-handed persons this may result from lesions in the right hemisphere. While destruction of one occipital lobe always causes hemianopsia, its irritation is only occasionally followed by hallucinations of light or of sight. If these are frequent and are the invariable precursors of unilateral spasms or of general convulsions and are followed by temporary blindness, there is sufficient evidence of irritation of the occipital cortex, with nervous "discharges" from it similar in kind to those occurring in the motor area. Hemianopsia coming on gradually, after such attacks have occurred at intervals, and associated with general symptoms of cerebral tumor, would warrant a diagnosis of tumor of the occipital lobe, as in a case of Seguin.[38] A large tumor of the occipital lobe may produce pressure upon the tracts from the parietal and central areas to the internal capsule, and thus cause indirectly hemianaesthesia and hemiplegia. These would, however, be slight in degree, and appear late in the course of the disease. Such a large tumor may also depress the tentorium cerebelli, and give rise in an indirect manner to hydrocephalus and to cerebellar ataxia with vertigo.

FIG. 2.



CORTICAL SPEECH AREAS. The convex surface of the left hemisphere. (Ecker.)—The speech areas of the brain. Lines parallel to *I* show the motor area of articulate language, lesion of which produces motor (ataxic) aphasia. Lines parallel to *II* show the sensory area of articulate language, lesion of which produces sensory aphasia (word-deafness). Lines parallel to *III* show the sensory area of written language, lesion of which produces word-blindness (alexia). The last two areas overlap: consequently word-deafness and word-blindness often occur together.

The following case illustrates this condition:

Case VI. (Huguenin, cited by Seguin: Journal of Mental and Nervous Diseases, 1886, p. 23.)—A female, aged eight years, suffered from headache, vomiting, vertigo, insomnia, and general convulsions for some time, and then, on examination, was found to have double optic neuritis and left homonymous hemianopsia. She went into a state of dementia and died.

Autopsy showed a tubercular tumor in the cuneus near the apex of the right occipital lobe.

5. *Tumors in the temporo-sphenoidal lobe* may be latent. Although physiologists assign the sense of hearing to the first and second temporal convolutions, and the sense of smell and taste to the uncinate convolution and hippocampus,[31] there are on record but few cases of tumor of these parts in man which confirm these localizations of function. Tumors of the left first and second temporal convolutions in right-handed persons cause the form of sensory aphasia known as word-deafness.[25] The patient is unable to recall the names of objects or persons, and cannot understand what is said to him, though his power of speech may be unimpaired. Such patients usually talk jargon or misplace words, and frequently are unable to read. The situation of the lesion is indicated in Fig. 2 by the lines parallel to *II.* Whether irritation of this lobe may produce sensory hallucinations in the course of tumors remains to be discovered, though in two cases a tumor at the apex of the temporal lobe has produced epileptic seizures preceded by an aura of smell.[39, 40]

The following case illustrates the difficulties of diagnosis of tumors in this area:

Case VII. (Mills: Journal of Nervous and Mental Diseases, 1887, p. 716.)—A girl, twelve years old, had a fall in September, 1886, after which she suffered continuously from headache, which became very intense in the course of six months. In April, 1887, she had tenderness over the right temporo-frontal region and located her headache there. She was found to have choked disks, dilated pupils, the right being larger than the left, and it was difficult to fix her attention. At this time she was suddenly seized with paralysis of the left arm and paresis of the left leg; her speech became indistinct, articulation being imperfect; and there was also slight paresis of the right side of the face. There was no spasm or anæsthesia. The question of operation was considered, but the local symptoms were not such as to point to the location of the disease. The patient died in May.

Autopsy showed a glioma of the right temporal lobe, three inches antero-posteriorly and an inch and a half in other directions, within which a hemorrhage had occurred.

6. *Tumors lying within the Sylvian fissure* and affecting the island of Reil produce numerous indirect local symptoms,—first, by affecting the circulation of the central area through pressure on the vessels which pass over the island, and, secondly, by pressure upon the tracts which pass beneath the island. Extensive paralysis may be due to the first, and paraphasia to the second. Paraphasia consists of inability to use language properly owing to a misplacement of words, and results in the patient's talking jargon. More extensive pressure upon the island of Reil may cause hemiplegia, by being transmitted to the lenticular nucleus and internal capsule.[21]

It is evident that tumors of the cortex produce a large number of local symptoms, and that these symptoms vary very much, in accordance with the area in which the tumor lies. It is quite easy to locate tumors in the central area and occipital lobe. It is very difficult to locate them elsewhere, unless they lie in the left hemisphere, when the form of aphasia present may determine the site of the new growth. Careful examination must be made in any case as to the condition of all the functions of the various areas, and too great caution cannot be exerted in determining the situation of the tumor or in recommending operative interference. And it must not be forgotten that tumors of the cortex are not yet to be differentiated with certainty from tumors of the white matter just beneath the cortex.

ADDITIONAL CASES OF CORTICAL AND SUBCORTICAL TUMOR.

REPORTER.	WHERE REPORTED.	SEX.	AGE.	LOCATION.	TUMOR.
Schweinitz .	Medical News, li. 233.	M.	8	Frontal.	Tubercle.
Birdsall . . .	N. Y. Med. Journ., xlv. 469.	M.	42	Occipital.	Sarcoma.
Soltmann . .	Jahrb. f. Kinderh., xx. 141.	F.	$1\frac{2}{12}$	Occipital.	Cysticercus.
H. Schmidt .	Jahrb. f. Kinderh., xxii. 353.	F.	14	Parietal.	Angio-sarcoma.
Charon . . .	Jour. de Méd. de Brux., January, 1886.	F.	7	Frontal.	Sarcoma.
Quincke . .	Deut. Arch. f. Klin. Med., xlii., No. 5.	M.	14	Central.	Glioma.

II. **Tumors of the basal ganglia** are not infrequent, and may be of any variety or size. They usually cause very marked symptoms, which are never direct local symptoms, since little is known of the function of the basal ganglia, but are indirect symptoms due to invasion of the internal capsule. It will be remembered that many of the fibres of the corona radiata from the cortex pass out of the base of the brain in the crus cerebri, which they reach by traversing the internal capsule. This capsule contains all the important tracts connecting the cortex with the body, and these tracts lie in the following order from before backward.

(1) A tract from the frontal lobe to the inner third of the crus cerebri, thence to the basal gray matter of the pons, thence to the cerebellum by way of its middle peduncle.

(2) The speech tract, from the third frontal convolution to the medulla.

(3) The motor tract, from the central convolutions to the middle third of the crus, thence to the pons (facial-nerve nucleus) and to the spinal cord (lateral columns) by way of the anterior pyramid and lower decussation of the medulla.

(4) The muscular-sense tract, from the parietal convolutions to the lemniscus in the tegmentum of the crus, thence to the spinal cord (posterior columns) by way of the upper decussation of the medulla.

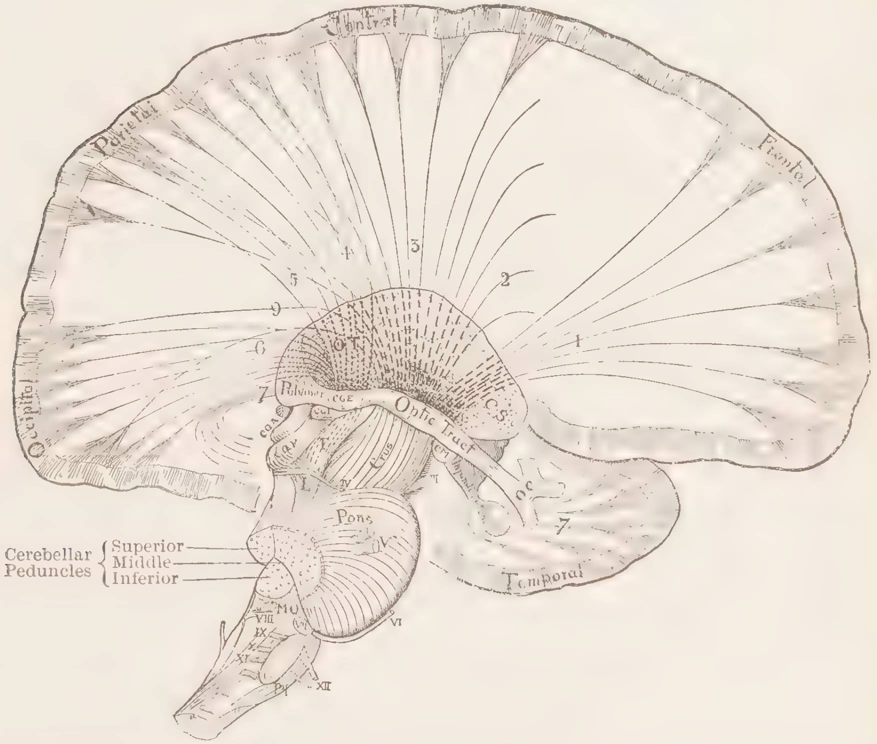
(5) The tactile-sense tract, from the parietal (?) lobe to the formatio reticularis of the tegmentum, thence to the spinal cord (posterior columns).

(6) The visual tract, from the occipital lobe to the pulvinar or posterior

part of the optic thalamus, thence by the optic tract to the optic chiasm and optic nerves.

(7) The auditory tract, from the temporal lobe to the corpus geniculatum internum, thence by way of the lemniscus to the auditory nuclei of the pons.

FIG. 3.



LATERAL VIEW OF THE BRAIN (diagrammatic), to show the tracts passing from the cortex through the internal capsule into the crus and pons Varolii. The numbers correspond to the tracts as numbered in the text. These tracts pass through the basal ganglia, and issue from the internal capsule beneath the optic tract upon the base of the brain, some entering the crus, others the tegmentum (*T*). *CS*, corpus striatum; *OT*, optic thalamus; *CQA* and *CQP*, corpus quadrigeminum anterior and posterior; *CGE* and *CGI*, corpus geniculatum externum and internum; *L*, lateral lemniscus; *III* to *XII*, cranial nerves; *CM*, corpus mammillare; *MO*, medulla oblongata; *Py*, pyramidal tract. Dotted lines in *T* (tegmentum) indicate position of sensory tract. The motor tract, after occupying the middle third of the crus, issues from the pons in the pyramid of the medulla.

(8) The fibres connecting the various areas of the cortex with the optic thalamus, which are interspersed among these tracts.

(9) A tract from the occipital and temporal lobes to the outer third of the crus cerebri, thence to the basal gray matter of the pons, thence to the cerebellum.

These tracts in their passage through the internal capsule are very liable to be compressed by tumors of the basal ganglia, and their function is then impaired. If the tumor involves the anterior part of the capsule only, no recognizable symptoms are produced. If it invades the middle portion, defective articulation, hemiplegia, or hemiataxia may be produced. If it involves the posterior portion of the capsule, hemianæsthesia, hemianopsia, and possibly slight deafness are caused.[41]

The order in which these symptoms appear will often give a clue to the direction in which the tumor is growing, whether from before backward or *vice versa.* From the symptoms it is impossible to determine whether the tumor presses upon the inner or the outer side of the capsule. But, as a matter of fact, tumors of the optic thalami and tumors growing in the lateral ventricle are much more frequently found than tumors of the lenticular nucleus. The distention of the lateral ventricles by fluid may produce outward pressure sufficient to impair the function of both internal capsules, giving rise to slight spastic paraplegia; but this is rare, as great distention may occur without such symptoms.

Tumors of the basal ganglia usually distort the brain and displace the crus and pons, stretching or compressing the cranial nerves, which then suffer in their function. Such tumors cause optic neuritis quite early in their course, and not infrequently vomiting and vertigo are more prominent symptoms than headache. Localized spasms do not occur with such tumors, but a peculiar athetoid movement of the opposite hand, somewhat choreic in character, has been noticed with tumors of the optic thalamus; and this may be accompanied by the maintenance of peculiar forced positions in the limbs of the opposite side. General convulsions are rare with tumors of the basal ganglia.

The diagnosis of tumors of the basal ganglia is more difficult than that of tumors in other regions, for the symptoms are complex, may be irregular, or may even be wanting, as has been the case in several instances.

CASES OF TUMOR OF THE BASAL GANGLIA.

REPORTER.	WHERE REPORTED.	SEX.	AGE.	TUMOR.
Pye-Smith	Neurol. Centralbl., 1884, p. 213.	M.	12	Tubercle.
Ashby	Medical Times, 1884, No. 1799.	M.	3	Carcinoma.
C. Turner	Lancet, 1885, i. 844.	F.	15	Sarcoma.
Bouchut	Jahrb. f. Kinderh., xv. 210.	M.	3½	Tubercle.
Ferrier	Mills, Pepper's System, Case 62.	M.	14	Glioma.
Senator	Charité Annalen, xiii. 323.	M.	2	Tubercle.

III. **Tumors of the corpora quadrigemina and tegmentum of the crura cerebri** are rare. Loss of pupillary reflexes, nystagmus, strabismus, vertigo, and disturbance of co-ordination with staggering gait such as occurs in cerebellar disease, are the local symptoms which have been observed. Irregular disturbances of sensation in the face and body may be produced, and also deafness, as the sensory tracts pass through the tegmentum. If a tumor is so large as to produce pressure downward upon the crura, it will give rise to third-nerve paralysis upon the side pressed upon and hemiplegia on the opposite side. This combination of symptoms is characteristic of disease of one crus cerebri, and may be produced by tumors on the base as well as by tumors in the tegmentum. Hydrocephalus is a frequent complication. Optic neuritis with blindness usually develops early in these cases. Tumors of the pineal gland produce symptoms similar to those just enumerated. These tumors by compressing the crura cerebri may cause paralysis of great extent.

The following case is cited in illustration:

Case VIII. (Sharkey: Spasm in Chronic Nerve-Disease, London, 1886, p. 26.)—A boy, aged seven years, received a blow on the head in 1879. In May, 1882, having gone to bed well, he awoke throwing himself about, and found himself partly paralyzed in all his limbs, especially in right arm and leg, and unable to walk properly. His speech was affected from the beginning, and his eyes about one week after the commencement of his illness. Tremors in the limbs were noticed from the first, and he had lost control over his bladder. He was very drowsy, but his memory was good. On admission in August to the hospital he was apathetic, but could be aroused. He had weakness in the arms and legs, and when he walked his legs seemed to drag. The most striking peculiarity of his condition was the tremor with which all his movements were accompanied. The head, neck, jaws, trunk, arms, and legs were all similarly affected. The tongue was protruded straight, and when he smiled the mouth was drawn to the right. Hearing was normal. All the muscles of the eyeballs were more or less weakened. There was ptosis on both sides, and the internal rectus of the right eye was markedly affected, as were also the superior and inferior recti. Both pupils acted normally; the right was larger than the left. No anæsthesia was present. The reflexes were normal. During the rest of his life the tremors increased, the paralysis became more marked, and attacks of spasmodic contraction of the muscles of the limbs and trunk occurred from time to time. The reflexes became exaggerated. He grew drowsy, and passed evacuations in bed. Towards the close of life optic neuritis developed. He died November 30, 1882.

Autopsy showed a large caseous mass in the region of the corpora quadrigemina, as if wedged into the aqueduct of Sylvius, which was much distended.

Case IX. (Hoppe: Neurol. Centralbl., 1888, p. 628.)—A male, aged eighteen years, had suffered as a child from cerebral symptoms, and four months before his death began to complain of occipital pain and occasional tremor-like shaking of his entire body. Examination showed double vision and loss of power of conjugate motion of the eyes upward. Pupils unequal and failed to react to light. Optic neuritis on both sides. Right ear deaf. He suffered occasionally from vomiting with the headache. All the symptoms increased in intensity gradually, and he became somnolent, lost control of his sphincters, and had difficulty in swallowing. He developed bed-sores and died.

Autopsy showed a sarcoma of the size of a pigeon's egg in the situation of the anterior corpora quadrigemina. The ventricles were greatly distended.

CASES OF TUMORS OF THE CRURA CEREBRI AND CORPORA QUADRIGEMINA.

REPORTER.	WHERE REPORTED.	SEX.	AGE.	TUMOR.
Mendel	Neurol. Centralbl., 1885, p. 512.	M.	3	Tubercle.
Henoch	Charité Annalen, 1882.	M.	3	Tubercle.
Warner	Med. Times and Gaz., No. 1542.	F.	4	Tubercle.
Senn	Mills, Pepper's System, Case 92.	M.	9	Sarcoma.
Browne	Mills, Pepper's System, Case 93.	M.	3	Tubercle.
Bristow	Lond. Path. Soc. Trans., 1886.	M.	7	Tubercle.
Finlayson	Glasgow Med. Journ., July, 1884.	M.	2	Sarcoma.

IV. Tumors of the pons and medulla, and tumors upon the base of the brain invading these parts, are quite common, and produce numerous and wide-spread symptoms. Even though the tumor be unilateral, the symptoms may be bilateral, the cranial nerves being implicated upon the side of the tumor, and the motor and sensory tracts to the opposite side of the body being at the same time invaded before they have crossed over. Such a condition is known as alternating hemiplegia or hemianæsthesia, and is characteristic of tumors in this region.

The symptoms referable to the lesion of the cranial nerves are the most important for diagnosis. A tumor in or upon the upper half of the pons and involving the crus cerebri usually affects the third and fifth nerves, producing external strabismus with dilatation of the pupil and ptosis, and also tingling, pain, and anæsthesia of the face, with ulceration of the cornea, and possibly grating of the teeth during sleep. A tumor in or near the lower half of the pons involves the sixth, seventh, and eighth nerves, causing internal strabismus with contracted pupil, paralysis of the face, including inability to close the eye, and deafness with vertigo. When the tumor lies within the pons and destroys the nucleus of the sixth nerve, there is inability to turn *both* eyes towards the side of the lesion, this nucleus presiding over the act of conjugate movement of the eyes to its own side, and being in functional connection with that nucleus of the opposite third nerve which governs the internal rectus muscle. Loss of power of conjugate motion to one side may indicate, therefore, a tumor within the pons affecting the sixth-nerve nucleus. In such a case, however, the power of convergence of both eyes is preserved. When the tumor lies in the pons or on its surface, and affects the root or the trunk of the sixth nerve, but not its nucleus, the conjugate movement of the opposite eye is not affected, and when the patient attempts to look towards the side of the lesion the opposite eye turns promptly, while the other remains fixed. This is the most valuable sign in differentiating a basal tumor from one within the pons.

A tumor invading the medulla oblongata may irritate or affect the glosso-pharyngeal, pneumogastric, spinal accessory, and hypoglossal nerves, producing difficulty in deglutition, irregular respiration, irregular or intermittent pulse, flushing of the skin, with profuse sweating, projectile vomiting, polyuria or glycosuria, retraction of the head or rolling of the head upon the pillow, and, lastly, inability to articulate distinctly or to protrude the tongue or to suck.

It is rarely that all of these symptoms appear in a single case, but when they do the probability is in favor of a tumor on the base, lying upon the side of the pons and medulla and compressing the cranial nerves after their exit. Tumors within the pons and medulla often cause remarkable combinations of some of these symptoms, but space is too limited to discuss such combinations here.[28] It is evident, however, that a tumor after affecting the nerves of one side may extend to the other side, and thus produce in the end bilateral instead of unilateral symptoms.

The invasion of the tracts passing through the pons and medulla is likely to produce paralysis and anæsthesia of the limbs. Usually the tendon reflexes are exaggerated in the paralyzed limbs, but occasionally they are lost when the tumor affects the pons. The paralysis is not usually total, but the muscles may become rigid; it is not accompanied by atrophy, and the electric reactions remain normal. The anæsthesia is not complete, as a rule. The control over the bladder and rectum is frequently impaired, the patient being unable to urinate voluntarily or to restrain the sudden emptying of the bladder when it is full.

Tumors of the pons not infrequently invade the middle peduncles of the cerebellum, which contain the tracts from the basal gray matter of the pons to the cerebellar hemispheres. In this case vertigo, with a special tendency to fall or to turn to one side, towards the side of the tumor, is observed. This tendency to fall is supposed to be an effort of the patient to save himself, the subjective vertigo leading him to think that he is falling towards the other side.

Tumors of the pons may produce such pressure on the cerebellum as to cause cerebellar ataxia, but this is very rare.

In tumors of the pons and medulla, headache, vertigo, and vomiting are the general symptoms most commonly observed; optic neuritis and psychical changes are often found, but general convulsions rarely occur.

The following cases are cited as illustrative of tumors of the pons:

Case X. (G. Middleton: Glasgow Medical Journal, April, 1888.)—A four-year-old boy had a fall, hitting his forehead and also the back of his head. When he got up he staggered, and the following night and for several days vomited frequently. On the twelfth day his right eye turned in, and he became dizzy, and often fell in walking, falling most frequently to the left side. In speaking his tongue seemed thick, and he could not put it out. It was then noticed that his face had little expression, that the saliva ran from his mouth, that he had internal strabismus of the right eye, and that he staggered in walking. Knee-jerks were increased on both sides. Pulse 100; temperature normal. A gradually-increasing weakness of both arms and both legs developed, and he died suddenly three months after the fall.

The autopsy showed a glio-sarcoma infiltrating the pons and medulla, more extensive on the right side, with dilatation of the ventricles with fluid.

Case XI. (Gibney: American Journal of the Medical Sciences, July, 1875.)—Glioma of the pons.

Male, aged six and a half, in November, 1873, fell on the back of his head. Two days after this he had a headache, and two weeks later his gait became irregular and ataxic, so that he reeled in walking. He was restless at night, and had headache frequently, but no vomiting or constipation. In January, 1874, his speech became indistinct and jerky, and his memory began to fail. In April he had become very stupid, and his speech was slow and unintelligible, though he tried to talk. There was marked ataxia of the head and all the limbs, but no paralysis or anæsthesia. There was exophthalmus, and all the motions of the eyes were performed slowly, but there was no disturbance of vision. Hearing was good. Later in the month he had involuntary evacuations of urine, and the left pupil was dilated. In May, vomiting, intermittent pulse, dimness of vision, and paresis of the facial muscles began, and an ophthalmoscopic examination showed atrophy of the left disk, and congestion of the right disk. On the 8th he became comatose, and on the 9th he died of apnœa.

Autopsy.—The pons was found to be enlarged in all directions, measuring two inches long, two and a half inches wide, and one and a half inches thick. In a depression along its centre ran the basilar artery, which was much stretched. The tubercula quadrigemina were pushed up and flattened; the anterior portion of the fourth ventricle was occupied by a rounded swelling, which was firm on the left side and elastic on the right side. The anterior pyramids at their entrance into the pons were elevated, but their point of entrance was deep and normal. Microscopic examination showed the existence of small round and polygonal cells, with granular matter in a stroma of thickened neuroglia, throughout the pons. (Glioma.) There was an atrophy of the optic nerves. Lateral ventricles were distended by serum.

Case XII. (F. A. Miles: Archives of Medicine, October, 1881.)—Tumor of one half of pons.

Female, aged seventeen; when admitted to the hospital was so stupid that no history could be obtained. On admission the following symptoms were found: complete paralysis and anæsthesia of the left half of the face; complete paralysis and partial anæsthesia of the right arm; paresis and impairment of sensation in the right leg, though she could walk; loss of smell in left nostril (tested by ammonia, which indicates anæsthesia); no paralysis of the tongue; loss of hearing in the left ear; total paralysis of the left eyeball, without strabismus, conjunctiva and cornea acutely inflamed; deglutition difficult, but appetite good; no vomiting; pulse rapid and weak; temperature varied slightly from normal; involuntary evacuations. Her mental faculties were so blunted that it was impossible to obtain reliable replies. She went into a condition of coma and died. (Duration not stated.)

Autopsy.—The pons was greatly distorted, and enlarged on the left side. Its surface was nodular, and its margins overlapped the medulla and crus. The left crus cerebri was also enlarged, and nodular. The left pyramid was compressed and indented, and the right pyramid pushed aside. The floor of the fourth ventricle was widened and bulged upward on the left side. The tumor was spherical in shape, occupied the left half of the pons, and had pushed the raphé to the right. No microscopic examination. No description of sections.

Case XIII. (J. C. Mackenzie: Cincinnati Lancet and Clinic, iv. 150.)—Tumor of one half of the pons.

Male, aged eight. November 1, 1877, he suddenly fell down, and on being helped up could not stand, and trembled greatly. Three days subsequently he had a similar attack, but this did not interfere with his going to school all the month. His teacher noticed that he was very clumsy, but did not think him stupid. For three weeks prior to December 12 he suffered from darting pains through his head, occasional vomiting, and weakness in his left hand. When examined December 12 there were found occipital headache, right facial paresis, head inclined to the left, ptosis of left eyelid, paresis of left hand, and an unsteady gait. In a week the symptoms had increased in degree; the left pupil was dilated, but the ptosis had disappeared in the left, and appeared in the right eyelid. His skin was cool; pulse 80, regular; appetite good. There was no intellectual disturbance. On December 29 vomiting was associated with the paroxysmal headache, and at the same time his bowels moved. His speech was indistinct, and he was very garrulous, talking constantly. During January his mind became much weakened; his special senses were not impaired; strabismus of the right eye appeared, the ptosis remaining; right half of face became anæsthetic; he could no longer stand or walk, and his head seemed too heavy for the muscles which supported it. The optic disks were normal. The left hemiplegia became complete, and his right foot was kept in motion constantly; the pulse became irregular and rapid, and on February 10 he died of paralysis of the pneumogastric nerves.

Autopsy.—The right side of the pons was much larger than the left; soft and white in its entire extent. The change extended along the middle cerebellar peduncle into the right hemisphere of the cerebellum. The fifth nerve could not be traced through the mass. This portion of the pons consisted of round and oval cells with few nuclei, in a granular stroma, with many vessels. The tumor was a soft glioma. No sections, and no accurate localization.

CASES OF PONS TUMOR.

Reporter.	Where Reported.	Sex.	Age.	Tumor.
H. Hun	Medical News, 1887, li. 386.	F.	6	Glioma.
Sokoloff	Deut. Arch. Klin. Med., xli. 443.	M.	5	Glioma.
Bourneville	Progrès Médical, 1887, No. 33.	M.	5	Tubercle.
Homen	Neurol. Centralbl., 1885, p. 418.	F.	10	Endothelioma.
H. Smith	Neurol. Centralbl., 1884, p. 210.	M.	3	Tubercle.
Crohn	Neurol. Centralbl., 1883, p. 471.	M.	5 mo.	Tubercle.
J. L. Smith	Archives of Pediatrics, 1888, p. 271.	M.	5	Tubercle.
Pousson	Jahrbuch f. Kinderh., xix. 249.	M.	9	Glioma.
Banham	Lancet, 1884, ii. No. 14.	M.	12	Glioma.
Seeligmüller	Jahrb. f. Kinderh., xvi. 343.	M.	2	Tubercle.
Sharkey	Spasm in Nervous Disease, p. 24.	M.	2	Tubercle.
Brown	Neurol. Centralbl., 1886, p. 151.	M.	2½	Tubercle.
Mills	Pepper's System, Case 83.	F.	8	Cyst.
Pepper	Mills, Pepper's System, Case 86.	M.	5	Glioma.
Hobson	Mills, Pepper's System, Case 87.	M.	2	
H. Jackson	Mills, Pepper's System, Case 88.	M.	12	Glioma.
Ross	Brit. Med. Jour., May 10, 1884.	M.	11	Glioma.
Whitcomb	Archives of Pediatrics, 1884, p. 205.	M.	8	Glioma.
Middleton	Glasgow Med. Jour., 1888, p. 342.	M.	4	Glio-sarcoma.
Seguin	Jour. Ment. and Nerv. Dis., 1882.	M.	9	Sarcoma.

CASES OF TUMORS OF MEDULLA.

Reporter.	Where Reported.	Sex.	Age.	Tumor.	Location.
Heubner	Archiv f. Psych., xix.	M.	1	Tubercle.	
Douty	Brain, 1885, 409.	M.	17	Villous.	Fourth ventricle.
Gibney	Mills, Pepper's System, Case 78.	M.	6½	Glioma.	" "
Lewis	Mills, Pepper's System, Case 82.	F.	6½	Tubercle.	" "
Finlayson.	Glasgow Med. Jour., 1888, p. 338.	F.	7½	Tubercle.	" "

V. **Tumors of the cerebellum** are of very great importance, as they are frequently met with in children and youth. In falls the back of the head is often injured, which may account for the greater frequency of tumors in the posterior cranial fossa. In many cases, when the tumor lies wholly within the cerebellar hemispheres and does not encroach upon the middle or vermiform lobe, there are no local symptoms of cerebellar disease, and no diagnosis of the situation of the intracranial growth can be made. Small tumors are occasionally found in this region unexpectedly at autopsies, but, as a rule, such tumors give rise to general symptoms. But when the tumor originates in the middle lobe of the cerebellum, or in its growth invades the middle lobe directly, or by pressure from a distance interferes with the circulation and function of the middle lobe, characteristic local symptoms appear. The time of the appearance of these local symptoms in relation to that of the general symptoms should be noticed; for if they appear very early the tumor is undoubtedly in the middle lobe, while if they appear late the tumor has invaded the middle lobe after growing for some time in the hemispheres.

The characteristic local symptoms of cerebellar tumor are vertigo and cerebellar ataxia.[42] The vertigo is more constant than in tumors elsewhere, is more severe, and is usually always in the same direction. The patient has a subjective sense of falling or turning about, and grasps for support, or sinks into a chair or to the floor. The sensation is so distressing that the eyes are closed, the patient is too bewildered to talk, and may be made faint and nauseated by the giddiness. The vertigo occurs in attacks with severe headache, but sometimes persists between the attacks in a lighter degree. It is not to be ascribed to disturbance in the movements of the eyes, though this symptom may be present, but is a direct symptom of some derangement in the mechanism of equilibrium. It is known that the central nervous connections of the semilunar canals of the ear, which give us the impressions of position in space, are in the cerebellum, to which they pass through the auditory nerve, and the vertigo is ascribed to the disturbance of this mechanism.

The second characteristic symptom of cerebellar disease is cerebellar ataxia. This consists of a staggering gait, which reminds one of the gait of an intoxicated person, the steps being irregular in length and position and the body swaying from side to side. It cannot be mistaken for the gait of locomotor ataxia, as in that the steps are of equal length, the feet are raised high and stamped down, and the patient watches his feet carefully in walking. Should any doubt arise, however, it can be resolved by testing the knee-jerks, which are lost in locomotor ataxia and preserved or exaggerated in cerebellar tumors; and also by testing the ataxia of the feet with the patient in a recumbent posture, for in locomotor ataxia the incoördination persists in any position, while in cerebellar ataxia the co-ordination is perfect except when the patient stands without support to the back. The patients frequently ascribe the irregular gait to the vertigo, but, as each of these symptoms may be present without the other, they must be considered as independent. The cerebellar ataxia is due to a disturbance in the mechanism of equilibrium in so far as it depends upon impressions coming to the brain from the muscles of the back and trunk and legs, all of which are received in the cerebellum. It may be so severe that the patient cannot stand alone or even sit up in bed. If the tumor invades either middle peduncle of the cerebellum, the tendency to fall to one side is very marked, though it is not yet certain whether the patient falls to or from the side of the tumor. In the large majority of cases recorded the tendency has been to fall towards the side of the tumor.

The cerebellar ataxia is not necessarily accompanied by paralysis or by loss of muscular sense in the extremities, but these symptoms are not infrequently developed when the tumor exerts pressure downward upon the pons and medulla. They appear in the limbs opposite to the side on which pressure is made. Such pressure may also cause vomiting and nystagmus and strabismus, all of which are common symptoms in cerebellar disease. Occipital headache, mental disturbances, and optic neuritis are general symptoms of cerebellar tumors, which appear early in the course of the case. Tumors in the cerebellum usually produce symptoms of disturbance of function in the cranial nerves, because in their growth they invade the base of the brain or displace the pons and medulla to one side.

The least frequent symptoms of cerebellar tumor are spasm and general convulsions, though occasionally retraction of the head and rolling movements of the head upon the pillow have been observed. They are probably indirect symptoms of affection of the medulla.

A very frequent complication of cerebellar tumor is hydrocephalus. This is explained by the pressure of the tumor directly upon the veins of Galen or on the fourth ventricle, the former producing venous congestion, and the latter preventing the free movement of fluid between the ventricles which seems to be necessary. Distention of the lateral ventricles may cause in infants great enlargement of the head, and even as late as the thirteenth year the cranial sutures have been separated by such intracranial pressure (Steffan). Such distention gives rise to headache, optic neuritis, and mental impairment: hence the frequency of these symptoms and their early appearance in cerebellar tumors.

The following cases illustrate the symptoms of cerebellar disease, and are of additional interest as they are the only cases on record at present in which an attempt at the removal of cerebellar tumors in children has been made.

Case XIV. (Bennett May: Lancet, April 16, 1887, i. 768.)—Male, aged seven, suffered in April, 1886, from headache, chiefly frontal, and vomiting; then gradual failure of sight developed, and in July he was nearly blind. In July paralysis of the right sixth nerve was noticed, and the eyes were turned to the left. Optic neuritis was then found in both eyes. At this time the gait became affected: he staggered, and tended to fall backward and to the left. His mind was clear. In August the headache and vomiting became severe, he could not stand, and the head was retracted. In October he had become totally blind, and nystagmus had appeared. There was loss of knee-jerk on the right side, and general weakness, with great emaciation.

He was then operated upon by Mr. May. The cerebellum was exposed on the right side of the median line, and appeared to be healthy, but bulged, and was felt to be hard at one spot. After incision at this spot, a tumor was felt one inch below the surface. This was dug out with the handle of a spoon. It was larger than a pigeon's egg, hard on the surface, caseating at its centre. Hemorrhage was slight, but the child died of shock a few hours afterwards.

Case XV. (Horsley: British Medical Journal, 1887, i. 865.)—Male, aged eighteen, had suffered from headache, vomiting, optic neuritis, increasing weakness of all his limbs, especially of the left arm and leg, vertigo, and typical staggering gait of cerebellar disease. He also had epileptoid attacks, with turning of the head and eyes to the right. His mental state was good, but he was much emaciated and had been in bed a year. Dr. Bastian made the diagnosis of a tumor involving the right lobe of the cerebellum, and, as a last resort, Mr. Horsley attempted its removal. After trephining, a tubercular tumor was found in the right lobe of the cerebellum and removed. It weighed seven drachms. The patient sank gradually, and died nineteen hours after the operation.

Case XVI. (Suckling: Lancet, 1887, ii. 656.)—Female, aged twelve, complained of headache and vomiting for eighteen months, and then for nine months of increasing weakness of the right arm and leg, and then for three months of severe frontal pain over the right eye. During the last month she had noticed an inability to turn the eyes to either side, and both double and dim vision. On admission to the hospital the left pupil was found to be larger than the right; there was nystagmus on any attempt at movement of the eyes; there was loss of conjugate motion of eyes to the right, and impairment of motion to the left; there was great impairment of vision, with double optic neuritis. Weakness of the right extremities with diminution of knee-jerk, staggering gait with tendency to stagger to the right and to fall forward, and marked tremor of the right arm on any motion, were found. There was a slight paralysis of the left side of the face, but the tongue deviated to the right. Headache, vertigo, and vomiting were very severe and constant. The diagnosis of cerebellar tumor was made, and an operation was considered advisable.

The occipital bone was trephined over the left side, and the cerebellum exposed. It bulged out of the wound, and its tissue appeared darker and softer than normal. A part of the cerebellar substance was cut away, and the wound was dressed. The patient went into a state of collapse, and died of exhaustion in forty-eight hours. The autopsy showed that the glioma had occupied the entire left lobe of the cerebellum and had invaded the middle lobe also.

CASES OF CEREBELLAR TUMOR.

REPORTER.	WHERE REPORTED.	SEX.	AGE.	TUMOR.
Peabody	Medical Record, xxix. 727.	M.	14	Sarcoma.
Kinnicutt	“ “ xxix. 642.	M.	12	Sarcoma.
Holt	“ “ xxvii. 342.	F.	10	Glio-sarcoma.
Lowenfeld	Neurol. Centralbl., 1885, p. 10.	F.	9	Tubercle.
Strahan	“ “ 1885, p. 11.	M.	12	Tumor.
Oliver	“ “ 1884, p. 58.	M.	4	Glioma.
Scarpi	“ “ 1884, p. 59.	M.	14	Cysts.
Laschkewitsch	“ “ 1883, p. 562.	M.	14	Glioma.
Lemcke	Jahrb. f. Kinderh., xviii. 307.	M.	7	Glioma.
E. Bull	“ “ xxv. 149.	M.	11	Tubercle.
W. Vost	Glasgow Med. Jour., July, 1884.	M.	12	Tubercle.
Drummond	Lancet, January 1, 1887.	M.	7	Tubercle.
Mackenzie	“ 1880, i. 14.	F.	$2\frac{8}{12}$	Glioma.
Mackenzie	“ 1880, i. 15.	M.	$3\frac{3}{12}$	Tubercle.
Bastian	“ 1880, i. 25.	F.	5	Glioma.
Wilks	Diseases of Nervous System, p. 190.	M.	5	Glioma.
Wilks	“ “ “ p. 192.	M.	4	Tubercle.
Sharkey	Spasm in Nervous Diseases, p. 29.	M.	4	Tubercle.
Sharkey	“ “ “ p. 33.	M.	10	Tubercles.
Heubner	Arch. f. Psych., xix.	F.	9	Tubercle.
Mills	Pepper's System, Case 71.	F.	13	Tubercle.
Goodhart	London Path. Soc. Trans., 1886.	M.	9	Glioma.
Taylor	“ “ “ “ “	M.	9	Glioma.
Butlin	“ “ “ “ “	F.	8	Glioma.
Finlayson	Glasgow Med. Jour., March, 1885.	F.	10	Tubercle.
Zenner	Cincin. Lancet, xxi. 61.	F.	16	Sarcoma.

Tumors upon the base of the brain, including aneurisms, produce local symptoms referable to disturbance of function in one or more of the cranial nerves, and to pressure upon the crura cerebri, pons, and medulla. The symptoms of tumor in the anterior fossa will, therefore, be those of tumor in the frontal lobe; in the middle fossa, of tumor of the basal ganglia and crura cerebri; in the posterior fossa, of tumor of the pons and medulla. It is often impossible to differentiate a tumor pressing upon these parts of the brain from a tumor within these parts; and for such finer distinctions of localization, which are, after all, of no practical importance, the reader is referred to special works on neurology.

DIFFERENTIAL DIAGNOSIS.

The diagnosis of the *existence* of a brain-tumor is to be made from the presence of the general symptoms, taken in connection with any local symptoms present, and studied carefully as to their mode of development and progress. The diagnosis of the *situation* of the tumor may often be made from a consideration of the local symptoms,—their order of appearance and method of extension, and their combination, being sometimes sufficient to make this diagnosis certain. It is to be remembered, however, that tumors in certain locations may not give rise to recognized disturbances of any cerebral function, and hence that local symptoms may be absent. It is also to be remembered that some local symptoms may be produced indirectly, by pressure or disturbance of circulation by a tumor at a distance from the area affected, and hence caution is to be exercised in studying the local symptoms. The diagnosis of the *variety* of tumor present may be arrived at by a consideration of the facts mentioned in the section on pathology. The diagnosis of a brain-tumor should not be made without considering the possibility of the presence of brain-abscess, of tubercular meningitis, of chronic hydrocephalus with atrophy of the brain, and of cerebral hemorrhage.[7]

The symptoms of *brain-abscess* may be the same in kind as those of brain-tumor, since both produce an increase of intracranial pressure and a progressive destruction of brain-tissue. In their origin, mode of development, progress, and termination, however, there are not infrequently marked differences. Brain-abscess develops very frequently in conjunction with suppurative affections of the inner ear and of the nasal and orbital cavities, and with caries of any of the cranial bones which lie in contact with the membranes. These rarely occur prior to the development of brain-tumor. The symptoms common to tumor and abscess may develop after a blow on the head, but when the condition is one of abscess the symptoms appear in more rapid succession, with greater severity, and more frequently with fever than when the condition is that of tumor. Furthermore, the symptoms of abscess after appearing suddenly often subside, the abscess becoming latent and all symptoms disappearing for months or years, and then break out again with suddenly fatal termination. This course contrasts markedly with that in a case of tumor, where a gradual progress with slowly-increasing intensity of all the symptoms is found. The constant addition of new symptoms is usual in tumors, and a temporary remission rather than intermission of the symptoms is the rule when the progress is not continuous. There may also be some points of distinction found in the individual symptoms. Thus, headache is more severe and paroxysmal with tumor; optic neuritis is much more commonly found with tumor; mental changes are more gradual and constant with tumor; and local symptoms are slower in onset and more apt to develop with tumor. A complication of tumor, not infrequent, is cerebral hemorrhage. Meningitis is the usual complication of abscess. Lastly, a duration of from one to two years with symptoms constantly present points directly to tumor.

Tubercular meningitis is under certain circumstances easily mistaken for cerebral tumor. This is not true of the ordinary cases of tubercular meningitis with acute hydrocephalus, which develop marked symptoms rapidly and terminate fatally within four or six weeks. But there are a number of cases of tubercular meningitis which present a chronic course with gradual progress, and in which the diagnosis from tubercular tumor is almost impossible. It is true that the symptoms often develop rapidly in these cases, and yet this is sometimes apparently the case in tumor; for, unless a patient is carefully watched, the early symptoms of tumor may escape notice for some time. The symptoms of chronic tubercular meningitis may be the same as those described as general symptoms of brain-tumor; but the headache is less severe in meningitis, and more continuous; there is more likely to be hypersensitiveness to light, sound, or touch in meningitis; and optic neuritis develops less frequently, less rapidly, and with less intensity than in tumor. Tubercles upon the choroid are found more frequently in meningitis than in tubercular tumor. It is, of course, understood that a localized meningitis may give rise to the same symptoms as a small tumor, and then the differentiation is impossible. This is more common about the base of the brain, in the region of the cranial nerves, than elsewhere. It is also to be remembered that a chronic progressing meningitis may develop in the vicinity of a tumor.[43] Here, again, the diagnosis will be impossible. A gradual subsidence of the symptoms, with recovery, will point to meningitis rather than to tumor.

Chronic hydrocephalus, while not infrequently the result of tumor or meningitis, may be due to a chronic inflammation of the ependyma of the ventricles. It then advances, and the fluid within the ventricles, producing pressure upon the brain, causes atrophy. The course is chronic, and the general symptoms are those of cerebral tumor. The local symptoms, however, differ in some respects from those of tumor. Spastic paralysis develops with chronic hydrocephalus without localized spasms, and is always bilateral; the lower limbs are affected more intensely than the upper. The child presents the extended, adducted, stiff legs, with overlapping knees, rigid muscles, increased tendon reflexes, and the spastic gait, so familiar as a sequel of Pott's disease; and, in addition, the hands move without proper co-ordination. As the disease progresses, the crura cerebri and pons may be displaced by the pressure of the fluid, and irregular symptoms due to stretching of the cranial nerves may appear. These, with the paraplegia, may lead to a suspicion of a tumor of the pons or base of the brain, and only by the order of development of the symptoms can the differentiation be made.

The diagnosis between cerebral tumor and cerebral hemorrhage will be necessary only in a few cases in which the onset of the symptoms has been very sudden. There are a few cases of tumor, chiefly glioma, in which the growth has been latent for some time, and has then given rise suddenly, after a blow on the head, or exposure to the sun, or some other accidental influence, to well-marked symptoms. These are usually both general and local, more noticeably the latter. The suspicion of a tumor will be aroused if after such an apoplectic stroke the symptoms persist and increase instead of subsiding, and if headache, convulsions, and optic neuritis appear. Hemorrhage alone never gives rise to the last-named symptom.

The diagnosis between cerebral tumor and cerebral syphilis needs little attention in patients below the age of fifteen, as we have seen that cerebral syphilis is rarely found in youth. The history of the case, the nocturnal pain, and the signs of syphilis will in any case aid in the differentiation.

It is necessary to mention that chronic Bright's disease, chronic lead-poisoning, and anæmia with hypermetropia have been mistaken for brain-tumor ; but a knowledge of this fact is sufficient to warrant such appropriate investigations as will enable any careful physician to avoid similar mistakes.

PROGNOSIS.

It is necessary in all cases of brain-tumor to give an absolutely unfavorable prognosis. Spontaneous recovery does not occur. The case goes on with greater or less rapidity to a fatal termination. The average duration is two years from the onset of the general symptoms. The only prospect of recovery lies in the possibility of the removal of the tumor by surgical interference. The only exception to this rule is in the case of syphilitic growths, which are, however, rare in childhood.

The manner of death is usually as follows. The general symptoms increase in intensity and wear out the strength of the patient, who gradually becomes emaciated and sinks into a state of stupor or coma, in which a series of convulsions occur and terminate fatally, or in which there occurs a paralysis of the respiratory mechanism, the heart continuing to beat for ten or even thirty minutes after breathing has ceased. Occasionally death occurs suddenly from cerebral hemorrhage, and rarely sudden death without known cause has been known to occur. The danger of sudden death in these cases should always be revealed to some member of the family of the patient, lest its occurrence after the adoption of some harmless therapeutic procedure should lead to mistaken accusations regarding the effect of remedies.

TREATMENT.

In syphilitic tumors or in cases where syphilis is a possibility, an active course of mercurial inunction with gray ointment, followed by the continued use of large doses of iodide of potassium, beginning with ten grains three times daily, and increasing a grain a day until sixty or eighty grains three times a day are being taken, is indicated.

In tubercular tumors the prompt employment of forced fatty diet, with cream, or cod-liver oil ; country air and life out of doors in a proper climate ; and the continued use of iodide of potassium in ten-grain to twenty-grain doses three times daily, together with tonics of all kinds, may be tried.

In all other forms of tumor it is customary in this country to administer iodide of potassium in large doses, and in the experience of Seguin and others favorable results have occasionally occurred. It affords the only means of hope in those tumors which are inaccessible to surgical interference.

The surgical treatment of tumors of the brain is a subject too large for extended discussion here, and is also a subject upon which facts are so constantly and rapidly accumulating that statements made now are likely to need revision in a very short time.[26] At the present time it seems possible to remove tumors from any part of the cerebral convexity or any part of the great longitudinal fissure. This includes tumors of the cortex and tumors of the centrum semiovale which lie just beneath the cortex and do not invade the basal ganglia. When the diagnosis of a tumor in these regions is arrived at with any degree of certainty, an attempt at removal is indicated. In many cases of tumor in the central and occipital regions an early diagnosis is possible; and in such cases the operation should be done as soon as the diagnosis is reached, provided the case is one considered suitable for operation. The question of removing tubercular tumors is an open one, German authorities disapproving[41] and English authorities urging the procedure.[45] The difficulty of removing all parts of a tubercular growth, and the liability of recurrence, as well as the unfavorable condition existing in a tubercular patient for any operation, must be admitted; yet successful operations are on record.

A more important question to be decided is in regard to the existence of a tumor so clearly defined from the brain-tissue as to allow of removal. An infiltrated sarcoma or glioma might be reached by the surgeon which could not be cut out without great destruction of brain-tissue, and whose limits could not be determined. As a matter of fact, such infiltrating tumors are usually quite vascular,—at any rate, much more vascular than the hard, encapsulated sarcoma or glio-sarcoma or cerebral cyst. The evidence of vascularity in a tumor is marked variability of cerebral symptoms, dependent on or in evident connection with those causes which alter the blood-supply of the brain or the general vascular tone. For example, if hot baths, warm or cold douches to the spine, stimulants, the changes of weather, such drugs as produce marked changes in arterial tone, have very marked effect upon the symptoms, either in decreasing headache, cerebral sensations, vertigo, vomiting, or spasms, or in increasing them, it is evident that the symptoms vary with the state of the blood-supply. It may then be concluded that the new growth is vascular, and such a vascular tumor is more likely to be an infiltrated tumor difficult of removal. All tumors, it is true, produce increase of intracranial pressure and variations in the cerebral circulation, but in hard encapsulated tumors the symptoms are much more constant and less liable to variations of the character described. Such tumors may be removed easily and safely when accessible.[46, 47]

When the table on page 551 is studied, it appears that fifty-six of the tumors tabulated were situated in the cortex and centrum semiovale of the cerebral hemispheres.

Since tumors in these parts are the only tumors which can be reached by the surgeon, it becomes a matter of interest to analyze these fifty-six tumors, with a view to ascertain how many of them could have been removed. A careful study of the histories of these cases[26] showed that in sixteen of them the tumor could not have been diagnosticated during life, or have been reached by a surgeon. Of the remaining forty cases, nineteen could have been successfully diagnosticated and removed. The remaining twenty-one could not have been accurately located, from the symptoms present, and hence in these the necessary indications for an operation were wanting. It is therefore evident that of the two hundred and ninety-nine cases eighteen per cent. were in a part of the brain open to surgical interference, and only six per cent. could have been successfully removed.

The question arises whether tumors in other locations, constituting the remaining eighty-two per cent., are inaccessible to the surgeon. It must be confessed that it seems impossible at present to remove tumors of the cerebral axis (thirty-four per cent. of the cases). These tumors lie too deep to be reached by the surgeon, or they lie in and about important cerebral tracts whose division during their removal would be absolutely contra-indicated, as dangerous to life, or as entailing permanent disability or paralysis. The most sanguine of surgeons might well refuse to interfere in such cases.

In regard to tumors of the cerebellum it is as yet too soon to make any absolute statements. It has been shown that, while the diagnosis of a tumor in the cerebellum is comparatively easy, it is very difficult to determine in which hemisphere of the cerebellum the tumor lies. This must be settled, if at all, through a study of indirect local symptoms, such as those of distant pressure on the pons and cranial nerves, which often mislead. Again, the cerebellum presents but one of its three surfaces to the cranial bones, and lies in such a small space under the tentorium that any displacement for exploration is impossible. If an operation for cerebellar tumor is undertaken and no tumor appears, the tumor may be searched for by incision of the hemispheres, which was done successfully in one of the cases already cited. How far such incisions are safe is as yet uncertain. It is probable that tumors of the middle lobe of the cerebellum can be reached, and can be safely removed if the middle lobe be not injured in the process. Its injury is always followed by permanent cerebellar ataxia in animals, and there is no reason why the same should not be true of man. And, finally, it must be noted that up to the present time operations for removal of cerebellar tumors have been uniformly followed by a fatal termination, the patients failing to rally, and dying of shock or of exhaustion with respiratory failure. With our present imperfect knowledge of the functions of the cerebellum, it is impossible to assign any reason for the fact that operations upon it are more dangerous than those upon the cerebrum. It must remain, therefore, undetermined whether or not it is justifiable to attack tumors of the cerebellum surgically. Yet this question is of great importance in cases of tumors in children, which, as the table shows, are found in this location more frequently than in any other (thirty-two per cent.).

The remaining cases of multiple tumor (fourteen per cent.) require but passing notice. The day may come when a surgeon may venture to operate upon several tumors at once in succession. As a last resort this might be attempted. But it seems that the existence of multiple tumors is likely to baffle both diagnostician and surgeon, and certainly the liability to recurrence or to the existence of other tumors of unexpected location would make an operation inadvisable.

The result of this review shows that but a small percentage of tumors of the brain are at present subject to surgical interference.

For a discussion of the methods of surgical procedure in the removal of tumors of the brain the reader is referred to the article upon cerebral surgery.

Since the removal of the tumor is impossible in a large proportion of the cases, treatment must be largely devoted to combating individual symptoms. Rest and quiet are always indicated, to prevent cerebral congestion. The majority of the general symptoms are due to an increase of intracranial pressure, and measures which may decrease this pressure may relieve the symptoms. Such are free watery purgation, hot baths, especially mustard foot-baths, cups to the temples or nape of the neck, leeches behind the ears, bromide and ergot internally. Any of these may be used when symptoms are distressing, with prospect of relief. When the headache is severe, antipyrin in fifteen-grain doses, or phenacetine in fifteen-grain doses, repeated in one hour (once), antifebrin three grains and morphine one-sixth of a grain, are the best remedies, the doses to be reduced according to age, those mentioned being for adults. The same remedies sometimes quiet vertigo. Insomnia may be treated by bromides, paraldehyde, or sulphonal. Cerebral vomiting is best arrested by bromides or by hypodermic injections of morphine. If general convulsions occur frequently, bromide and chloral will have to be given, as in a case of epilepsy. In any case such measures are merely palliative, and, when there is doubt regarding the diagnosis of the tumor, and it is known to be inaccessible to the surgeon, it is justifiable to give the patient all the comfort possible by establishing an opium-habit.

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AFFECTIONS OF THE NERVOUS SYSTEM IN HEREDITARY SYPHILIS.

By ABNER POST, M.D.

Our knowledge of the manifestations of syphilis in the nervous system is far from complete; in the congenital forms it is even less complete than in the acquired. This article is not intended to be in any sense a complete account of nervous syphilis in the child, but rather to record so much of our present knowledge—chiefly from the clinical stand-point—as will be of practical use in the detection of other cases.

There have been recorded a comparatively large number of cases of congenital syphilis that have shown nervous symptoms of various sorts. In fact, so various have been the manifestations that it is probable that every form of nervous syphilis known in the adult may appear in the heredito-syphilitic child, and these various manifestations may take place in the youngest infant or among those late symptoms which occur in childhood or even in adult life, and which constitute syphilis hæreditaria tarda, so called.

Although many cases have been reported, they are as yet comparatively infrequent as regards the total number of syphilitic children.

As to the relative frequency of nervous symptoms among syphilitic children, Rumpf[1] has some interesting figures.

Of his patients with nervous diseases of a syphilitic origin there are forty-two married, of whom he has exact knowledge. Of these forty-two marriages fourteen are without children, and there was apparently no suspicion of pregnancy even. Of the remaining twenty-eight marriages all have children. In thirteen cases out of these twenty-eight, syphilis was not recognizable in the children. In fifteen families hereditary syphilis showed itself in the most various forms. In these fifteen families there occurred seventy pregnancies, and of the children thirty-two were born dead before term. Of the remaining thirty-eight children, seventeen died in the first month, in part of convulsions, one died at the age of three years, of inflammation of the brain, and the remaining twenty are still alive. Of these twenty still living, perhaps three may be regarded as healthy. (They all belong to one family, in which nine miscarriages were followed by eight children who are still alive,—five more or less heredito-syphilitic, the youngest being less diseased than the eldest, while the three last born present no certain sign of syphilis.) In four the syphilitic manifestations are doubtful, and in thirteen they are evident, more particularly in diseases of the bony system. In two cases there is disease of the nervous system. Seven cases having already died of diseases of the nervous system, the proportion of nervous affections in the total of seventy syphilitic births stands at about thirteen per cent.[1] That is to say, the early forms of nervous syphilis amounted to thirteen per cent. The possibility that the apparently healthy may have later forms cannot be shut out. Whether the nervous system was affected in some of the still-births cannot be determined.

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[1] Die syphilitischen Erkrankungen des Nervensystems, von Theodor Rumpf, Wiesbaden, 1887.

Whether there was a special tendency in the disease to attack the nervous system because of a nervous heredity cannot, of course, be determined; but thirteen per cent. must either be an unusually large proportion or the syphilitic origin of nervous disease in children must be often overlooked.

There is the same history of prodromic symptoms and gradual progress towards the most serious forms of disease to be found in many of the cases of hereditary nervous syphilis as in the acquired. Headache, persistent or intermittent, and often accompanied with dizziness, occurs in elder children, while in infants wakefulness and convulsions occur with frequency.

Clinically there are a number of cases in which death takes place with the appearance of severe brain-disease. Usually convulsions set in, occasionally attended by paralysis, enlargement of the head, or abnormally small head. Of Rumpf's seventy-two cases ten died in this manner. These cases went on rapidly to death, and the most different syphilitic changes were found.

It is probable that in most of the alleged recoveries from tubercular meningitis the disease has been syphilitic. The more favorable opportunity for treatment offered by a syphilitic origin makes it exceedingly important to recognize heredito-syphilis in such cases. "A general indefiniteness of symptoms and slowness of progression should arouse suspicion, especially if the absence of pulse-retardation or the presence of any characteristic symptoms indicate that the vault rather than the base of the cranium is involved," is the caution given by Dr. H. C. Wood.[2]

Fournier thinks the diagnosis between tubercular meningitis and syphilis may usually be made, and that it rests upon the following points. In cerebral syphilis there is, save in very exceptional cases, a constant absence of fever, while in tubercular meningitis, on the contrary, the rise of temperature commences with the initial symptoms and undergoes later curious and almost characteristic oscillations. Certain symptoms which are constantly observed in tubercular meningitis are not met with in cerebral syphilis, such as the sudden alternations between redness and pallor of the face, the retraction of the abdominal wall, the variations in the pulse-rate, and the curious association of a raised temperature with a pulse of normal or diminished rapidity.

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[1] Rumpf's method of reckoning is a little obscure. Apparently six of the cases said to have died of convulsions are counted as cases of nervous disease; one died of inflammation of the brain, and two are still living: these nine cases are practically thirteen per cent. of the seventy pregnancies.

[2] Syphilis of the Nervous System, 1889, p. 15.

E. Mendel believes that chronic hydrocephalus is often of syphilitic origin.

In a digest of the literature on chronic hydrocephalus, by M. Armand Ruffer, M.D., in *Brain*, part ii., 1890, the author states that he has found hereditary syphilis mentioned in twenty per cent. of the cases examined by him or mentioned in medical literature, though this is a larger percentage than is given by other writers. Hasse reports the case of a woman who had a chancre, the husband being evidently syphilitic. She had three stillborn children, but the fourth was hydrocephalic, hemiplegic, with purple spots on the skin, and died when six months old. The other children, born in later years, were evidently syphilitic.

Sandoz believes in the existence of a syphilitic hydrocephalus which may be congenital or may develop in the first few months of extra-uterine life. According to this author, it is most probably caused by inflammation of the ventricular ependyma and plexuses.

In speaking of hydrocephalus, Fournier says, "I have too often met hydrocephalus in connection with hereditary syphilis not to recognize a connection entirely different from simple chance. On the other hand, for the sake of controlling my own observation by the experience of others, I have searched the annals of science, and I have found such a number of cases identical with my own that it is truly impossible not to consider syphilitic heredity as one of the better-proven causes of hydrocephalus." But he distinctly states that it is but one of several causes that give rise to hydrocephalus.

The cause of an enlargement of the head that passed for a hydrocephalus was found by Heubner (in a child of nearly five months) not in an extraordinary enlargement of the ventricle, which was but slightly enlarged, but in a pachymeningitis hæmorrhagica.[1] Waldeyer and Köbner found a similar case.[2] They found double-sided periostitis with changes similar to those in pachymeningitis hæmorrhagica interna. Heubner considers the enlargement of the ventricle secondary to the disease of the skull, and believes that in this way a confounding with hydrocephalus takes place.

Epileptic convulsions in various forms have long been recognized as occasionally the result of inherited syphilis. They constitute one of its most frequent manifestations, it may be as the only manifestation or associated with other symptoms of cerebral affections. Sometimes, though beyond question rarely, the epilepsy is free from all associated symptoms, and cannot be distinguished from essential epilepsy. More often the attacks, at least in the opinion of Fournier, are associated with pain in the head and symptoms of a congestive order, such as buzzing in the ears, sudden and passing obscurations of vision, dizziness, and modifications in habits and character and failure in intellect. A case of Fournier's illustrates so well this class of accidents that it seems worth while to give it in brief:

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[1] Virchow's Archiv, lxxxiv. 267.

[2] Ibid., lv. 368.

An infant born of parents recently syphilitic presented in infancy unmistakable syphilitic lesions on the skin and mucous membranes. It was treated and apparently cured. It reached the age of seven and a half years in apparent health. At that time it had a convulsion while at play. Two other attacks succeeded in the same week. At this time Fournier saw the case. The parents described minutely the convulsions, but no mention was made of any other symptom. Fournier succeeded in drawing out a series of symptoms which served for diagnosis. 1. For about three weeks the boy had at intervals complained of his head, and sometimes severely. At such times he could not bear the light. 2. During the same time he had experienced strange symptoms, which he had told his mother. At certain moments he could not see, or his head turned round or everything turned about him, he heard bells in his ears, and once thought he was going to fall. 3. The child's governess gave important information in regard to his intelligence. For some weeks he had been inattentive, incapable of application, and forgetful. For some seconds he seemed to comprehend nothing, and could not answer questions put to him. Such a collection of symptoms excluded the hypothesis of simple epilepsy, and showed the convulsions to be symptomatic of a most serious cerebral affection, which would be most naturally syphilitic from the antecedent history.

Gowers[1] has seen eight cases in which patients with epilepsy were the subjects of well-marked inherited syphilis. In all these cases the attacks had the aspect of idiopathic epilepsy, cases in which there were symptoms suggestive of local brain-disease being excluded. In only two of these cases did the attacks begin in infancy. In all the others they commenced towards the end of or after childhood.

A case of my own illustrates the attacks of less marked character. In this case the syphilis is undoubted, but the heredity cannot be fully established, except from the evidence of Hutchinsonian teeth and hazy corneæ. She is seldom free for any length of time from some manifestation of syphilis, which yields to appropriate treatment. When twelve years old, she had a series of "nervous spells." In these attacks, which always occurred at night, everything seemed to be turning round, and at the same time the child felt as if she were in a boat which was rocking violently and was going to tip over. These attacks lasted about an hour, and were followed by nausea and vomiting. There was no loss of consciousness. The attacks occurred as often as three times a week, and disappeared after the use of iodide of potassium.

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[1] Epilepsy and other Chronic Convulsive Diseases, etc., by W. R. Gowers, M D., F.R.C.P., 1881.

Hemiplegia, more or less complete, is an occasional manifestation of inherited syphilis. It seems to be often preceded by other symptoms of disease within the cranium, but is sometimes the first sign of nervous trouble to attract attention.

In a clinical lecture on hemiplegia in children, Dr. John Abercrombie, assistant physician to the Great Ormond Street Hospital, speaks of some fifty cases of which he has notes. There was evidence of congenital syphilis in four of these cases, and there was good reason to attribute the paralysis to this cause. Syphilis may have had a share in some of the other cases also, as two of the children in whom the paralysis followed measles were certainly syphilitic, and consequently may have had disease of the cerebral vessels, while it was also a possible factor in other cases.[1]

Dr. A. B. Marfan gives, in the *Annales de Dermatologie et de Syphiligraphie* of September 25, 1887, the following history:

The father had chancre with secondary lesions six months before marriage. Being without symptoms, he believed he was well. Three or four months after marriage the wife had a sore, evidently a chancre, on the lower lip, being at that time already pregnant: she took iodide of potassium, and her baby was born apparently healthy, but at the age of one month had a syphilitic eruption.

The second child, a daughter, the subject of this sketch, was born in January, 1887. On the 1st of May the little one, up to that time healthy, fell from a height of fifty centimetres, and on examination after the fall moved all her limbs spontaneously. The 3d of May the mother found she did not move the left leg. The following day the left arm was almost without movement, and the mouth was deformed when she cried; the child did not appear to suffer, and nursed well. Examined on the 8th of May, it was possible to recognize the following condition. The left lower limb was nearly inert. There was not complete abolition of motion; it would be more proper to call it paresis than paralysis, since the limb maintained itself in the position that was given it, but had no tendency to return to the original position. When the child was held up by the fingers in the axillæ, the right leg was animated by very active movements, while the left leg remained motionless. The left arm was motionless, though the right was very active. When crying, there was marked deviation towards the right. The orbicularis was intact, and the pupils showed no inequality. No contractures in the paralyzed limbs. Sensibility intact.

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[1] British Medical Journal, June 18, 1887, p. 1326; Braithwaite's Retrospect, January, 1888, p. 37.

Mercurial inunction was employed upon the child, and to the nursing mother two grammes of iodide of potassium were given daily. At the end of eight days amelioration was manifest: movements had in part returned. Fifteen days after, it was impossible to tell any difference between the two sides.

Any possible doubt as to the hereditary syphilis was removed a month later. On June 22 the child showed moist papules.

The author says, "At the beginning we could not be absolutely certain that the hemiplegia was due to syphilis. The undeniable syphilis of father and mother and the certain hereditary infection of the elder brother spoke for it; but the absence of all tegumentary lesions and of all other index of hereditary syphilis, the perfect health of the child, and the rarity of specific hemiplegia in the new-born, left us in doubt. Hesitation disappeared in part with the effect of treatment, and totally with undeniably syphilitic cutaneous lesions."

Dr. Marfan speaks of an arterial lesion, a *partial* obliteration, as the only explanation of the symptoms.

Dr. J. Hughlings Jackson, in his pamphlet on "Nervous Diseases in Inherited Syphilis," gives a case of a girl of ten years under his care with hemiplegia of the left side. The paralysis of the arm was complete, the leg was so weak that the child could not make an approach to standing, and the face deviated to the right. The signs of congenital syphilis were satisfactory. Two years before, she was said to have had St. Vitus's dance. The mother was quite positive that the movements affected only the left, the subsequently paralyzed, side. On getting out of bed one morning nearly two years later, she fell, and could not stand when raised. She was put to bed again, and quickly afterwards she screamed and went into a fit. She foamed at the mouth and her mouth was bloody, and the fit was followed by the hemiplegia already described.

This case is particularly interesting because of the preceding chorea. A few other cases have been reported in which chorea has occurred in syphilitic children, but the relationship of cause and effect cannot be considered established.

Paraplegia depending upon inherited syphilis is rare, but differs in no essential particular from the paraplegia attending acquired syphilis.

The paralysis may be the result of spinal lesions that manifest themselves externally and that affect the cord only secondarily, or of lesions that attack the cord or its envelopes directly. Fournier relates a case of the first class in a boy of seven years, who was attacked with a gradually increasing paresis of the legs. Fournier found the reason for that symptom in a massive swelling of several dorsal vertebræ which constituted a sort of gibbosity. The specific nature of that bony lesion was inferred from the family and personal antecedents of the child, and from the coexistence of other manifestations undoubtedly syphilitic, and notably tibial periostitis. Treatment by iodine produced a notable diminution in the size of the spinal tumor and coincidently in the paralysis, but the child was obliged to leave Paris prematurely, and its subsequent history is unknown.

Laschkewitz relates a similar case occurring in an heredito-syphilitic girl of thirteen years, in which a hyperostosis of the second cervical vertebra gave rise to paralysis of all four limbs. Recovery took place in two months under antisyphilitic medication.

Of the second class, Keyes gives a case of a boy of four or five years, who was affected with complete paraplegia on two separate occasions, the attack lasting only one day each time, and who habitually suffered from too frequent desire to urinate, especially at night. He had headache, and was irritable, pallid, run down, dejected, and miserable. At eight years of age he had had no return of his nervous symptoms.

The relationship between syphilis and disease of the cord is still a subject of grave discussion, and but few attempts have been made to establish a connection between disease of the cord and hereditary syphilis. Remak drew attention in 1885 to a possible connection between tabes in children and syphilitic heredity.[1] His first case was that of a twelve-year-old girl whose symptoms consisted in a girdle-like constriction, lancinating pains, paræsthesia with a reduction of the sensation of taste, absence of the reflexes, ataxia and gastric crises, one-sided ptosis and double vision, and, later, atrophy of the optic nerve.

Affections of the peripheral nerves in consequence of hereditary syphilis are frequent, and have been particularly noticed in the cerebral nerves, but usually in conjunction with other symptoms. Atrophy of the optic and auditory nerves is probably not unusually to be referred to this cause.

Dr. Hughlings Jackson, in his pamphlet on "Nervous Diseases in Inherited Syphilis," published in 1868, said that he had seen but one case of marked nervous symptoms (except convulsions) in an infant who at the same time showed signs of syphilis. In this case, seen in Mr. Hutchinson's practice, there was spasm of the muscles supplied by the portio dura, and paraplegia.

A peculiar form of paralysis has been occasionally met with, affecting the anterior branches of the brachial plexus and causing more or less complete motor paralysis of the upper extremities, the sensibility and temperature remaining normal. In two cases of this affection noted by Henoch the upper extremities were almost completely paralyzed, the flexor muscles of the fingers alone retaining a slight trace of contractility. Signs of syphilis were present, and the paralysis soon disappeared under the influence of specific treatment. In connection with some of these cases a peculiar twisting of the head backward has been mentioned.

Paralysis of the ocular muscles is very common among nervous manifestations of acquired syphilis. In congenital syphilis it has been reported but rarely.

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[1] Berlin. Klin. Wochenschrift, 1885, No. 7.

There is a report of a case by Mr. Nettleship,[1] in which the patient, a girl of fourteen, had partial numbness of the right forehead, cheek, and side of the nose, and of the eyelid, conjunctiva, and cornea. She could feel, but less distinctly than on the other side. The numbness appeared greatest on the eyeball itself. There was complete paralysis of the external and superior recti, and paresis of the inferior and internal recti and levator palpebræ: the superior oblique was probably paralyzed. In this patient the father was known to have had syphilis before the child's birth, and the evidence of syphilis in the child herself was satisfactory.

Mr. J. B. Lawford reports two cases of paralysis of the ocular muscles in congenital syphilis in the London *Ophthalmic Review* for 1890, page 98. These cases had both reached adult life. In one case, at least, the paralysis was probably due to peripheral nerve-disease. In looking for recorded cases through a large amount of literature Mr. Lawford has found only three cases. The first is one recorded by Von Graefe: a child aged two years had paralysis of all the branches of the left third nerve; the right eye had also been lost from syphilitic iritis. At the post-mortem examination there were found gross changes in the intracranial portion of the third nerve, described as a gummatous interstitial neuritis and perineuritis. The second case was that of Mr. Nettleship, already mentioned; and the third is contained in Hutchinson's book on syphilis, and is that of a boy aged sixteen, who had partial bilateral ophthalmoplegia externa, and, in addition, complete atrophy of the optic nerves.

Ptosis and double vision were symptoms in the case of Remak just mentioned.

Idiocy does not seem to be a frequent result of syphilis, according to Ireland, in his "Idiocy and Imbecility," though he has met with a few apparent instances of the kind.[2] Such cases are rare, according to Fournier, because the cerebral lesions which result in such a loss of intellect are usually fatal; but, though rare, they are nevertheless well authenticated. Fournier has himself met with five cases.

Syphilis leads more often to juvenile dementia after infancy than to congenital cerebral deficiency. In an extremely valuable article on this subject in *Brain* for 1883, Dr. Judson S. Bury concludes that hereditary syphilis is a more frequent factor in the production of mental disturbance than has hitherto been recognized, and that congenital deficiency of mind from inherited syphilis is probably rarer than mental failure coming on in childhood. He thinks it is questionable whether juvenile dementia and insanity have been properly separated by writers on insanity.

In the matter of diagnosis, it must be remembered that there are no symptoms pathognomonic of syphilis of the nervous system. The same general principles hold good in nervous symptoms in hereditary syphilis as in acquired. Disorderly grouping of symptoms and the coexistence of several foci of disease—that is, the simultaneous affection of several points of the nervous system at the same time—are circumstances that should awaken suspicion. It is the antecedent history of the patient and of the family that should then be considered. If there is a reasonable suspicion of the disease, the child should be given the benefit of the doubt and be put upon specific treatment. The iodide of potassium may be given in relatively large doses to quite young children without harm. It is particularly indicated in nervous syphilis, where it shows its greatest influence. It should be used first and freely. Mercury may well be added; but the relative value of these two drugs is, in nervous disease, the reverse of that in other and more common forms of congenital syphilis.

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[1] Transactions of the Pathological Society of London, 1881, vol. xxxii.

[2] See also Fletcher Beach, Cases of Idiocy and Imbecility due to Inherited Syphilis, American Journal of Insanity, January, 1888.

TOXIC AFFECTIONS FROM ARSENIC AND LEAD.

By J. J. PUTNAM, M.D.

ARSENICAL POISONING.

DEFINITION.

It is the intention of this paper to deal mainly with the subject of chronic arsenical poisoning, especially as arising from those causes to which children are liable to be exposed, such as the use of arsenic as a medicine, and for the purpose of coloring papers and fabrics. Acute arsenical poisoning will be described only so far as it is necessary to give completeness to the general clinical picture, and for the sake of calling attention to certain matters of pathological interest.

ETIOLOGY.

Sources of Poisoning, and Relative Poisonousness of the Different Arsenical Preparations.—The sources of poisoning to which children are mainly exposed—not to speak of intentional poisoning—are, for the acute forms, the mixtures used to destroy vermin (such as "Rough on Rats," "Paris Green," "German Fly-Paper," and "Buffalo Carpet-Moth Annihilator"), against the manufacture and sale of which our laws afford us inadequate protection; for the more chronic forms, the incautious use of arsenic as a medicine, and, above all, many of the pigments employed in coloring articles of domestic use, and certain glazes. Of some of these pigments arsenic is a fixed component; while it is used in the manufacture only of others, and can be removed from them by a proper purification, which, of course, somewhat increases the cost[1] of the product. Of the latter class are many of the aniline dyes; and this may suffice to call to mind the general fact that numerous colors besides green are to be suspected.

The following list, condensed from those given in the papers of Dr. Jabez Hogg,[2] and the reports by the Committee of the English National Health Society,[3] and by Draper[4] and Wood, will indicate the range of materials containing arsenic which might be sources of danger to children: fancy colored papers, both in sheets and as used for covering or making boxes, toys, and confectionery, lamp-shades, labels, books; artificial flowers and leaves; wax ornaments; clothing, and curtains printed or woven; furniture-covering (perambulators); colored toys; oil-colors for decorative purposes; japanned goods; Venetian and other blinds; linen-glaze (rarely), and glazed paper collars; flannel and calico shirts; the linings of boots, coat-sleeves, and hats; colored chalk; stockings; and foulard cambric.

Green tarlatan contains enormous quantities of arsenic, applied so loosely that it readily flies off, and both workers and weavers of this fabric have frequently been actually poisoned. This material, though now rarely made into dresses, is still largely used for decorative and other purposes.

A recent investigation in England, by A. W. Stokes,[5] shows that the danger from these various sources is not a matter of the past. The external use of arsenic on raw surfaces, and even on the sound skin, whether in the form of powder (see *infra*, Violet-powder Cases), germicide washes, or ointments, may give rise to poisoning. Some of the papers used for kindergarten purposes (especially the glazed green and red) contain very large quantities, so that a piece a few inches square might, if chewed or sucked, cause serious or even fatal poisoning, and such results have followed the sucking of wall-paper or, in one case, of a green band surrounding a package of envelopes.

With regard to the vexed subject of wall-paper and carpet poisoning, however much overrated the danger from these sources may be thought to have been, no candid person can thoroughly study the evidence without becoming convinced that the danger exists. The arsenic occurs not only in the pigment of the paper, but sometimes also in the paste or sizing with which the pigment or the paper is attached.* In some cases, especially with papers of an older date, but still largely on our walls, as much as forty or fifty grains of arsenic have been found to the square foot of paper.† We are not, however, as yet in position to assert how much arsenic a paper must contain before it is to be pronounced dangerous. This question can be answered by extended clinical observations alone, and it is already certain that various conditions not fully understood may make a paper dangerous which would have been thought harmless.

The greatest danger of poisoning from this cause is doubtless from the dust blown off by currents of air, or detached by the brush or cloth, which settles on the floor, or on books and furniture, thence to be wafted off again in small or large quantities. Besides this source, however, the facts that poisoning seems to occur sometimes from an arsenical paper over which another, free from arsenic, has been laid, and that heat and moisture favor its occurrence, suggest that some volatile compound of arsenic may be formed.

Toxic Dose.—The maximal therapeutic dose of arsenious acid for adults is usually placed at from five to eight milligrammes (from one-twelfth to one-eighth of a grain, equivalent to from ten to fifteen minims of Fowler's solution). Much larger doses are occasionally taken after gradual habituation, and the arsenic-eaters of Styria, it is said, become inured to doses of twelve or even twenty centigrammes.

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* A small, and probably insignificant, amount of arsenic is said to come from the imported hides, preserved with arsenic, from which the glue-size is sometimes made.

† A case has recently come to the writer's notice where almost all the papers of a patient's house, fifteen in number, which had been put on by one of the best dealers in Boston twenty years ago, contained arsenic, and most of them in large amounts.

Doses of six centigrammes (one grain) are liable to cause alarming symptoms, and from twelve to eighteen centigrammes (from two to three grains) to cause death (Taylor and others), though the fatal result occasionally fails to occur after enormous doses, both of arsenic in substance and of the solutions of its salts.

Children differ less from adults in their susceptibility to arsenic than in that to many other poisons.

Doses of ten and even twenty minims of Fowler's solution two or three times daily are sometimes reached in the treatment of chorea, but the patients need close watching while taking them. Taylor[6] reports a case where a child of sixteen months was given about one-third of a grain of arsenious acid in solution, and recovered after prolonged vomiting. The same writer[7] was personally consulted with regard to three hundred and forty children in an industrial school who were accidentally poisoned in consequence of the dilution of milk with water from a boiler containing a quantity of arsenical cleansing fluid. Each child was believed to have taken the equivalent of about one grain of arsenious acid, yet all recovered after one or two weeks. In cases of wall-paper poisoning, moreover, affecting a whole family, it is by no means always the children who suffer most. Children differ greatly, however, as regards their susceptibility, and occasionally show slight symptoms of poisoning from a dose as small as two minims, perhaps even less. I have cited in another place[8] a number of instances to show that even small doses are not without danger, and that apparent habituation is not a perfect safeguard.*

PATHOLOGY.

Arsenic is a poison to all forms of animal life, with the unexplained exception of certain bacteria and low fungous growths. The changes which it sets up in the human body are partly due to its local action, partly secondary to alterations in the blood and nervous system and other organs.

Although not regarded as a corrosive agent, it is a powerful local irritant, and may eventually cause the destruction of the mucous membranes, and, less rapidly, of the skin, when applied in solution or even in substance.

It is important to note that many, though not all, of its local effects closely resemble those which it excites in the same parts after absorption. It will therefore be profitable to consider in conjunction these two modes of action, noting the differences between them when necessary.

The direct action of arsenic on the skin varies greatly, according to the preparation, its concentration, and the length of exposure, as well as to the susceptibility of the patient and the peculiarities of his reaction, the latter factors being dependent in part upon age and constitution. In general, it may be said that the arsenical eruptions cover a large range of inflammatory conditions and typical dermatoses. These cases are often difficult of diagnosis. Thus, Dr. J. C. White[9] reports the case of an infant with extensive and severe papular eczema, with excoriation of the face and scalp, and intertrigo of various parts of the body, apparently due to arsenical wall-paper.

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* Even the peasants of Styria are said to fall victims occasionally to their habit. See also a case reported in the Dublin Quarterly Journal, vol. xxxvi. p. 474.

A cutaneous affection which occasionally results from arsenic when given for a long time is herpes zoster, seen by Bokai * in three out of one hundred and thirteen cases of children, all treated in a similar manner for chorea. This small number might well be due to coincidence, but other practitioners have made the same observation,[10] and the writer has recently seen an apparently similar case in an old woman. It is possible that in such cases the arsenic acts by causing localized neuritis, as it is known sometimes to do, and that the herpes is a secondary result.

Another interesting effect is a general bronzing of the skin, simulating the appearance seen in Addison's disease, and lasting for a number of weeks after the cessation of treatment.[11]

An important series of cases illustrating the local and general effects of arsenic applied in substance to the skin of young infants are the "violet-powder" cases reported by W. H. Power and by several gentlemen who had observed the patients.[12] Of the twenty-nine cases reported, thirteen were fatal. The powder contained about thirty-eight per cent. of arsenic, and was dusted on repeatedly for toilet purposes. The skin became intensely congested, and in some instances dark-colored and brawny. Acute inflammatory reaction and sloughing soon set in in some of the cases.

Arsenic is liable to be absorbed through the skin, especially from abraded surfaces; and severe general symptoms and even death have resulted, in the case of children, from ointments and washes, and even from clothing, such as stockings, colored with arsenical pigments.[13]

It is still more true of the changes set up by arsenic in the mucous membranes, than of those excited in the skin, that they may occur independently of the local action of the poison.

These changes in the stomach and intestines have been the subject of much study. They may be entirely absent even in acute cases of poisoning where arsenic has been swallowed. When present, they consist essentially, as regards gross appearances, in ecchymoses, or larger and smaller hemorrhages, and ulcerations. Although this is often given the name of hemorrhagic gastritis, the signs of true acute inflammation are usually wanting. Under the microscope, fatty degeneration of the gland-cells is found, and this sometimes occurs with great rapidity, as in a case described by Virchow,[14] where the patient died within a few hours after swallowing a large

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* Compare J. C. White, loc. cit.

dose with suicidal intent. Analogous changes occur in the conjunctiva, mouth, pharynx, and gums.

An interesting article on the "Anatomical Appearances resulting from Acute Poisoning by Arsenic" has been recently published by Dr. F. W. Draper,[15] of Boston.

After absorption, no matter from what surface, arsenic distributes itself through the body, and, if in sufficient quantity, sets up degenerative changes in the kidneys, liver, heart and other muscles, in various other organs, in the bones, and in the blood, analogous to those found in the gastric glands, besides causing ecchymoses in the pericardium, endocardium, and elsewhere. These changes, together with those induced by phosphorus, the action of which is similar to that of arsenic in this as in several other respects, have recently been studied with especial care by Ziegler and Abolonsky,[16] for the liver and kidney in particular, and incidentally also for the muscles. The action of arsenic and that of phosphorus were found to be similar, but that of arsenic was the less intense.

The first action of both is found to be on the gland-cells, causing degeneration, usually described as fatty, but more strictly a progressive granular necrosis, formation of vacuoles, necrosis, and destruction. Next the epithelium of the gall-ducts and endothelium of the vessels, especially the capillaries, and finally the connective-tissue cells, become involved. The nuclei of the liver-cells divide and multiply, and even a slight degree of fatty degeneration does not seem to prevent this change. This process of new formation seems to be due to the direct action of the arsenic after penetrating the cells. The changes, both in these organs and in the muscles, occur in foci.

It is important to note that the term "degeneration" does not strictly apply to the whole of the process observed in these experiments, but that an active cell-division goes on, within the parenchyma of the organs as well as in the connective tissue. It may be that the influence which leads to this change is in some way related to the therapeutic action of arsenic and phosphorus.

The careful experiments of Vrijens[17] (Amsterdam) in 1881 make it probable that no department of the nervous system is exempt from the influence of the poison, though not all are equally involved. The hemorrhages so widely found are thought to result in part from paralysis of the vasomotor nerves, in part from the changes in the constitution of the blood and vessels.

The probability is that the paralytic and sensory disorders of the limbs are due to multiple neuritis. This view was first suggested by Jaeschka in 1882,[18] and has since then been confirmed[19] by anatomical as well as clinical evidence.

To what extent the central nervous system is responsible for these paralyses is still uncertain. Changes in the spinal cord, perhaps even etiologically antecedent to the neuritis, might be anticipated in the case both of lead and of arsenic, and in fact Popow[20] in 1882 described such changes. Doubt was afterwards expressed as to the validity of his observations, but their author has recently[21] reaffirmed his opinion, supporting it by an account of the examination of the spinal cord from a case of acute arsenical poisoning. The matter cannot as yet be regarded as definitely settled, but it has been shown that arsenic injected subcutaneously is quite capable of destroying the adjacent nerves, independently of any change in the nerve-centres. Inasmuch, however, as some of the cases of muscular atrophy of arsenical as well as of saturnine origin are permanently incurable, it is highly probable, judging from what we know of the reparative power of the peripheral nerves, that the central nervous system is sometimes seriously affected.

The occasional occurrence of profound anæmia would seem to indicate that the damage is sometimes done to the blood-making organs. Arsenic is sometimes found in the bones, and in one case, reported under Elimination, traces were found in the bones many months after cessation of the exposure. In this case the arsenic had been used as a medicine for a long time and in small doses.

According to Filehne,[22] the erosions seen in the stomach are probably not due to the direct action of the arsenic, unless where it is present in substance, but to the peptonic digestion for which the degenerative changes in the gastric glands prepare the way. He found that if the stomach was kept steadily alkaline by magnesia, or carefully freed from acid by frequent washing, these changes did not occur.

Dogiel[23] has recently shown the important fact that arsenic and its salts form chemical compounds with the albuminoid tissues (gelatinous substance when heated with egg albumen), although this is contrary to what has been universally believed.

The anatomical changes observed in the case of children are, for the most part, the same as those seen in adults. Cohn has, however, found œdema of the brain in a child of five years who died from acute poisoning, and suspected the same condition in the case of another patient whose symptoms were of similar character, but who eventually recovered.

ELIMINATION.

Arsenic is found in all, or almost all, the secretions and excretions of the body, especially the bile, the fæces, and the urine.

The secretion most important for present consideration is the milk. Brouardel and Pouchet[24] gave Fowler's solution, in the dose of from two to twelve drops, to nursing mothers, without unpleasant results, but found in the case of one woman, who took the maximal dose daily for six days, one milligramme of arsenic in each one hundred grammes of milk. Experiments with animals gave similar results. The young in many cases died, and arsenic was found in their tissues. The same writers cite the following case. A man was accused of an attempt to poison his wife with arsenious acid. The woman lived, but her nursing child of two months died after forty-eight hours. The whole body, weighing two kilogrammes, was analyzed for arsenic, and five milligrammes were found.

On the other hand, Ewald[25] could not find arsenic in thirty grammes of the milk of a woman who had been taking thirty drops of Fowler's solution daily for four days.

The time usually considered sufficient for the complete elimination of arsenic is from twelve to twenty days, but exceptionally it is found in the urine after five or six weeks. Gibb[26] reports a case, however, where traces were found in the liver and bones of a female patient six months after cessation of a long course of medicinal treatment which had led to her death. In some of the wall-paper cases it has been found as late as seven or nine months after the apparent removal of the source of poisoning.[27] This class of cases is obviously not well suited for determining the point, but the facts are stated here on account of their clinical value.

It is not yet known whether elimination goes on more rapidly with children than with adults. The process is, however, undoubtedly favored by a healthy condition of the kidneys and other organs, and it is possible, though not probable, that the rarity (see below) of arsenical paralysis in children is due to their eliminating the poison before this symptom, which is usually a relatively late one, has time to make its appearance.

Frequency with which Arsenic is found in the Urine.—Dr. C. P. Worcester has examined, at my request, more than fifty specimens of urine for arsenic. The persons from whom the specimens were mainly taken were patients presenting themselves at the out-patient department of the Massachusetts General Hospital, those cases being selected, as a rule, which did not present any symptoms that seemed referable to arsenical poisoning. The specimens showed traces of arsenic in forty-three and a half per cent. of the whole number taken. This result seems to me one of decided interest, apart from its clinical importance.

The fact to which I have repeatedly called attention, that lead is to be found with great frequency in the tissues of healthy persons, is, for two reasons, less surprising than that the same should be true of arsenic: first, because lead is less easily eliminated than arsenic, so that it may accumulate little by little; second, because the sources of possible lead contamination are extremely numerous.

The sources of arsenical contamination, on the other hand, are comparatively few. The coloring-matter of wall-papers, and of cotton and woollen stuffs, constitutes by far the most important of them; and even if, in estimating the significance of the figures I have given, we make large allowances for coincidence and for exceptional causes, it is impossible to avoid the conclusion that from these materials arsenical compounds are frequently given off, under conditions which render them liable to be absorbed, and of such a kind that they occasionally cause more or less severe symptoms of poisoning.

On the other hand, the clinical inferences which the figures primarily suggest is, that while these coloring-matters are evidently a source of frequent contamination of the tissues, they are not a frequent cause of poisoning, and that the discovery of arsenic in the urine cannot be taken as a conclusive proof, or even as very strong evidence, taken by itself, as to the origin of doubtful symptoms.

Nevertheless, if the results of these analyses are anything like typical of those which would be obtained from a larger number, it is evident that the danger, such as it is, is a wide-spread one, and that even if only a very small proportion of the persons in whose urine arsenic could be found should be injuriously affected, the absolute number might be quite large.

SYMPTOMATOLOGY.

The symptoms of arsenical poisoning vary according as the patient has been exposed to (1) large toxic doses; (2) medium doses increased gradually to beyond the limit of tolerance; (3) minute doses continued for a long time; and according to the nature of the preparation.

Very large doses may be rapidly fatal in the course of a few hours or even half an hour,[28] sometimes with convulsions, delirium, and other signs of serious involvement of the nervous system in all its parts, and sometimes without causing any other symptom than collapse. Vomiting and diarrhœa may be entirely wanting or present in only moderate degree, and this especially in the cases of acute collapse.

Cohn[29] reports two cases of acute poisoning in young children, five and six years old respectively, in order to show that severe cerebral symptoms—loss of consciousness and delirium, apparently due to hyperæmia and œdema of the brain—may occur not only in cases of rapid collapse, where gastro-intestinal signs are wanting, but also when they are present. In the "violet-powder" cases above referred to, some of the infants died with symptoms of general prostration. Abdominal signs occurred, but were not, as a rule, prominent.

When the prostration of the nervous functions has not been so great as here indicated, vomiting and purging generally make their appearance, and, from their violence as well as from the character of the discharges, sometimes suggest true cholera. These symptoms are usually associated with excessive thirst, burning sensations along the œsophagus and in the throat, intense pain in the abdomen, with tympanites, cramps in the extremities, and retention of urine. The latter secretion, as well as the alvine discharges, may be bloody. The action of the heart and the respiration are profoundly affected. Consciousness is usually preserved, but attacks of syncope and convulsions sometimes occur, especially with young children.

In the following interesting case of poisoning in a young boy, reported by Silliman,[30] the symptoms for a time suggested belladonna poisoning. The first symptoms were thirst, nausea, and retching, but there was no vomiting; these were the only gastric symptoms, and they rapidly subsided. In from one-half to three-fourths of an hour there was profound stupor, with excessive dilatation of the pupils, the pulse was rapid (130) and small, respiration from 50 to 60 per minute and shallow, and there was great muscular debility. Emetics were given, and the vomitus contained no blood nor mucus. The patient was revived by stimulants. Two and a half hours after the first symptoms the pupils had become normal, and the pulse and respiration had improved. Five hours later there was a relapse, ending in collapse, coma, and death eight hours and three-quarters from the time of onset. During the whole course there was no complaint of pain and no disturbance of the bowels.

It is noteworthy that there are sometimes temporary lulls, even of several days' duration, which give false assurance of recovery. Recovery may, however, occur in a case that has long seemed hopeless, and this is as true of children as of adults. Thus, in one of the instances above cited (reported by Cohn), of a child six years old, in spite of the fact that delirium and loss of consciousness were present, and increased up to the tenth day, the patient eventually recovered, and at the end of four weeks was nearly restored, though somewhat emaciated. Likewise, in the interesting case of a girl eleven years old, reported by Donelly,[31] in spite of severe symptoms of acute poisoning, and although as late as the fourth day the body was greatly swelled, the breasts hard and painful, and the secretion of urine almost suppressed, there was eventual recovery.

Even after the acute symptoms of the early stages of poisoning have passed away, the patient has still much to fear from the subsequent outbreak of paralytic symptoms, supposed to be due to generalized neuritis. These may follow even a single dose, and may occur after an interval of days, weeks, or even months.

These paralytic symptoms consist in greater or less loss of power over the extremities, usually associated with severe rheumatoid pains, tenderness, and a variable amount of cutaneous and muscular sensory impairment, especially of the hands and feet. The lower extremities are commonly affected more than the upper, the distal more than the proximal groups of muscles, and the extensors rather more than the flexors; but exceptions to all these rules occur.[32] The electrical irritability is usually diminished to both currents, but may be normal or even exaggerated. Atrophy of varying degree generally occurs, and in adults is occasionally incurable. Ataxia, also, probably of neuritic origin, is occasionally seen.[33]

These paralytic symptoms are of infrequent occurrence, relatively to the other symptoms, even with adults, and apparently still more rare in the case of children, although a number of instances have been reported in which there were cramps, pain, and tingling in the extremities. An interesting case is reported by McCready where paralysis of the legs, followed by death, occurred in a young child in consequence of rubbing arsenic mixed with gin on the head to cure an attack of favus. The death, however, occurred so soon after the exposure (less than forty-eight hours) that it may be questioned whether the paralysis was really of neuritic origin, though this is not impossible. Kirchgasser has also seen paralysis of the legs from poisoning with wall-paper. A case was cited in a recent paper by the writer where ataxic symptoms showed themselves in a child of about twelve years, in whose urine arsenic was found, probably of wall-paper origin. I have found no report of a typical, well-marked case of generalized neuritic paralysis in an infant or a young child. Several cases of lead paralysis of this character are, however, on record.

Even in the first stages of poisoning, marked signs of irritation of the peripheral nerves may be present, consisting in severe pain and paræsthesia and muscular cramps in the extremities, and these symptoms are met with in children.

These paralytic (neuritic) symptoms occur, in adults, both after large single doses of arsenic and, though but rarely, after long-continued repetition of small doses, and even in cases of poisoning from wall-papers and fabrics.

The Effects of Moderate Doses of Arsenic gradually increased to the limit of tolerance, as when it is employed as a medicine with the object of obtaining its full physiological action, and pushed too far.—The first signs of poisoning under these circumstances are, usually, disorders of the digestion, consisting in coated tongue; redness, followed by lividity, of the gums, with varicose enlargement of their vessels, and, eventually, ulceration and bleeding; salivation; loss of appetite; gastric pain; nausea; vomiting; and offensive discharges containing undigested food. The urine is liable to be diminished in quantity, and is often turbid and offensive. Albuminuria may be present. The eyes soon come to have a glistening look, the pupils enlarge, the sclerotic becomes yellowish in color, the lids swelled and painful, the conjunctiva congested, and the cutaneous glands of the lids inflamed. The metabolic changes (urea, etc.) increase and then diminish, or oscillate. The nervous functions are all liable to be deranged. Among the more prominent symptoms of this class are signs of cerebral irritation, and prostration, and the sensory and motor disorders of the extremities which later deepen into the paralyses characteristic of arsenical neuritis. (This is, essentially, a digest of the experiments of Vaudrey upon himself and others.)

Poisoning from the too long continued Use of Arsenic in Small Doses.—The cases falling under this head have been already discussed under Dose. It only remains to be said that the symptoms usually make their appearance in the order and manner described in the last section, but that almost any one of the symptoms may be unusually prominent or may be wanting.

Occasionally, though very rarely, as has already been mentioned,[34] after long use of arsenic and apparent habituation, a violent, almost acute attack may occur.

It is especially noteworthy that the first symptoms of any prominence may be signs of localized nervous irritation in the extremities or abdomen, such as numbness and prickling, or cramp-like pains, usually associated with more or less general indisposition. One digestive sign often referred to as occurring early is a silvery-whitish coating on the tongue. The occasional occurrence of herpes zoster and of bronzing of the skin is spoken of in an earlier part of this paper.

Poisoning from Arsenical Wall-Papers, Paint, and Fabrics.—Fortunately, the time has almost gone by when the idea of danger from these sources of poisoning can reasonably be scouted, and it is now only through neglect that the physician fails to take them into account in obscure cases of failure of health, even without specific symptoms.

The following remarks are based, so far as possible, on observations[35] in which arsenic was actually found in the urine. The illustrations will be drawn largely from the cases of children, but such other facts will be added as may throw light upon this obscure subject.

It is not equally true for chronic as for acute poisoning that the symptoms are the same in children as in adults, inasmuch as the effect of the poison is, in part, to call out latent tendencies to disease, and in determining these age is probably an important factor.

The local and special symptoms do not differ from those seen in poisoning from larger doses, except in the fact that they are only in faint traces, which have to be carefully sought for.

The cases may usually be classed, respectively, in one of five groups: 1. Those in which the symptoms mainly concern the general nutrition and health, without signs of local irritation. 2. Those in which the symptoms are mainly due to irritation of the conjunctiva, mouth, or pharynx. 3. Those in which the symptoms mainly concern the digestive tract. 4. Those in which the symptoms mainly concern special departments of the nervous system. 5. Those in which the symptoms mainly affect the nutrition of some special part of the body other than those mentioned. To these there might fairly be added a sixth class, containing cases in which latent tendencies to nervous affections of various sorts are brought out by the debilitating influence of chronic arsenical poisoning, just as lead poisoning causes hysteria.

Most cases present several of these sets of symptoms.

The cases of the first class are probably less common than those of the second and the third, since signs of local irritation are usually present in some degree. It is, however, to be remembered that it is just in regard to cases of this group that more accurate means of diagnosis are needed, and perhaps we think them rare only because they are overlooked. Two or three cases are known to the writer where arsenic, apparently of wall-paper origin, was found in the urine, and where few or no symptoms were observed besides anæmia, debility, and perhaps (especially frequent in the case of children) irritability and restlessness. One of the twenty-five cases referred to in the note[36] is that of an infant, reported by Dr. Francis, of Brookline, who, though previously healthy, became languid and refused food, but recovered promptly after removal from exposure. Arsenic was found in the urine.

If suspicions are aroused, they may properly be strengthened by even slight signs of local irritation either of the mucous membranes or of the peripheral nerves. If slight prickling or numbness of the hands and feet is complained of, careful examination for diminished electrical irritability of suspected muscles should be made. If these signs are absent, the diagnosis can often be only guessed at; but the probability of arsenical poisoning is increased, though not converted to a certainty, if arsenic is found in the urine on analysis by a skilled chemist.

Anæmia and general impaired health are constantly met with in connection with other symptoms, such as disorders of digestion, etc., without being wholly secondary to them.

Cases under Class 2 are very common. The conjunctivitis is frequently purulent and associated with inflammation of the Meibomian glands, and even with nebulæ and ulceration of the cornea.

The changes in the pharynx may assume a diphtheritic appearance, and Osborne Reynolds reports the case of four children, where the diagnosis of diphtheria was made, who eventually died. The father and mother also showed signs of arsenical poisoning, and the sickness of the children was traced, with great probability, to the same source. Kirchgasser, also, speaks of diphtheritic changes in the pharynx. The pharyngitis is apt to be associated with bronchitis and cough; these symptoms are worse in the morning, when the arsenical paper is on the patient's bedroom, and, like other symptoms of poisoning, both local and general, are worse when the windows of the rooms are closed and when the furnace is lighted. The heat of a lamp or gas flame, acting upon an arsenical shade, is liable to produce similar results.

The digestive disturbances (Class 3) may occur either alone or combined with the other symptoms, and may affect either the stomach or the bowels most severely. Morris reported the cases of two children of an English surgeon, who died with symptoms of enteritis. Occasionally, though rarely, constipation occurs instead of diarrhœa.

Icterus may occur, and tube-casts with small quantities of blood are occasionally found in the urine, together with albumen.

I have seen no statement that casts and blood have been found in the urine in children, but albuminuria is not very uncommon with them where arsenic is given as medicine, as in chorea.

Class 4.—The nervous disorders most frequent in children are headache, insomnia, restlessness and nervous irritability, neuralgic pains, and, rarely, signs of the slighter degrees of neuritis.

Kirchgasser reports the case of a boy, three and a half years old, who suffered from convulsions, followed by paralysis of the legs, possibly traceable to arsenical paper, and passing away after removal from exposure. The convulsions showed a tendency to daily periodical recurrence and were followed by feverishness.

The same writer has observed that other nervous symptoms may show this same tendency to periodical recurrence, simulating malarial affections; and something of the same sort was reported by Dr. Nichols, of Cambridge.[37] Here the diagnosis was strengthened by the discovery of arsenic in the urine. Other instances of convulsions in young children have been noted in cases of this class.

Class 5.—Affections of the skin due to poisoning from arsenical wall-paper are noted, although but rarely. There is some reason to think that inflammation of the external and middle ear may be due to the same cause.

The neuralgia is sometimes so sharply localized as to suggest neuritis of a single nerve, and two or three cases have been reported where the nutrition of one limb has been seriously impaired.

The symptoms are sometimes much worse in the morning, the patient often waking with a severe frontal headache, bad taste, and irritation of the eyes and the digestive tract.

The apparent immunity enjoyed by a certain proportion of the persons under the same exposure is often commented on as throwing doubt on the cause of the symptoms in the rest. But, while this doubt is entirely justifiable in a given case, the general fact that there is great difference in the susceptibility of different persons towards many other poisons besides arsenic is too well known to need comment.

The symptoms of the second class may be mainly looked for where the arsenic is present in the form of dust, but this cannot be stated as a universal rule.

Arsenical paint, though less injurious than many papers, is, nevertheless, capable of mischief; and the same may be said of papers containing arsenic, over which other papers, free from arsenic, have been pasted, or which have been coated with varnish.

It is worthy of notice that what might be called a therapeutic action of arsenic—namely, an increase in weight and fat during exposure—is seen in cases of exposure to arsenical wall-paper.

Kirchgasser has spoken of the same effect in relation to one or two cases of children where other members of the family had shown signs of poisoning.

I have not met with any account of salivation in children, though in the case of adults it is somewhat characteristic.

DIAGNOSIS.

Acute arsenical poisoning presents itself under two general types, both of which, however, are subject to many variations. The first is that of rapid collapse, which follows the ingestion of large quantities of the poison, and may be unattended by any symptoms of local irritation. The second is the gastro-intestinal type, characterized by intense burning at the epigastrium, along the œsophagus, in the fauces, etc., and by the cholera-like vomiting and purging.

Both these forms might possibly be mistaken for poisoning by phosphorus or other irritant poison, or even for cholera, under certain circumstances. In case of doubt, the stomach may be washed out, and examined for the characteristic odor of phosphorus, and the attempt made to develop the garlic smell of arsenic by heating.

It should be remembered that, even in cases of subacute poisoning, where death is delayed for some days, gastro-intestinal signs may be inconspicuous. In the special case of children, the possibility of the association of acute cerebral symptoms, due to œdema of the brain, with the gastro-intestinal signs, as in the case described by Cohn, will be borne in mind.

With regard to subacute and chronic arsenical poisoning, there are certain signs which, when present, are characteristic; but the diagnosis is frequently very obscure, and in the absence of an evident cause cannot always be made out with certainty.

The most characteristic symptoms are the œdema of the eyelids, and ulceration of the gums and in the fauces; the occurrence of continuous or intermittent, and apparently causeless, attacks of acute indigestion, with nausea, and with epigastric pains, or with salivation; and, finally, the paralyses; but as yet only a few cases of paralysis, and these not in a fully-developed form, have been reported as occurring in children. For the sake of completeness, we give the most important features of this symptom as met with in adults. The arsenical paralyses are not to be absolutely distinguished from those due to neuritis from other causes, but it is relatively characteristic of them, as compared with those due to lead, that the lower extremities are affected more than the upper, and the extensors more than the flexors, while at the same time both flexors and extensors are involved.

In the majority of cases the paralysis is associated with signs of marked debility and restlessness.

Ataxia has been observed in neuritis of various origins, but more often in arsenical than in lead neuritis. Furthermore, an acute onset is very much more common in arsenical than in lead paralysis.

Although it is more true of paralyses from arsenical neuritis, to a certain extent, than of those from lead neuritis, that the distal muscular groups of the limbs suffer more than the proximal, still a distribution varying from this is sometimes met with.[38]

With regard to the extremely chronic forms of arsenical paralyses, those resulting from arsenical wall-papers and the like, it is only, in many cases, by weighing the evidence of every sort that a conclusion can be reached. Characteristic symptoms are sometimes absent, or represented only by traces, and an almost infinite variety in grouping may be expected. Under Symptomatology the diagnostic points have been given which will be found especially useful.

When neuritis is present, the electrical reactions are usually diminished to both currents. They may, however, be nearly normal, and may even be increased.[39] Sometimes the electrical reactions suffer before any loss of voluntary power occurs. This is a point of great practical importance, because a careful examination will occasionally enable us to make a diagnosis in a case otherwise very obscure.

An excellent test for slight weakness of the extensor muscles of the hand consists in letting the patient try to maintain the carpus fully extended and to spread the fingers.

In doubtful cases the urine should be analyzed by a professional chemist.

It is not, of course, to be concluded from the finding of small quantities of arsenic in the urine, that the symptoms from which the patient is suffering are due to that cause;* but the weight of other evidence is obviously strengthened thereby.

With regard to the significance of other urinary signs, the presence of indications of renal irritation is suggestive, but not pathognomonic.

A few cases are on record which would suggest that acute and chronic nephritis may be due to this cause. One of these, reported by Dr. T. M. Rotch, is cited in the paper already referred to[40] (Case 19). This patient was a child's nurse, forty years of age, and it is interesting to note, as illustrative of the different kinds of personal susceptibility, that, while the nurse suffered from a subacute nephritis, the child himself showed the general and the mucous-membrane symptoms of arsenical poisoning, but no nephritis, in spite of the fact that he was attacked with scarlet fever at the time of exposure, and that, too, while the urine still contained arsenic. The fever ran its usual course. Both patients recovered promptly on removal from exposure.

PROGNOSIS.

The prognosis of acute arsenical poisoning is always grave, but even the most unpromising cases sometimes recover. A favorable issue is not necessarily to be expected on account of a lull in the symptoms, even though this may last several days.

With regard to the chronic forms, the prognosis varies with the duration and severity of the symptoms. In the light cases, and in most of the moderately severe ones, complete recovery may be expected under favorable conditions.

In peripheral neuritis the heart's action is liable to be affected, and death occasionally results from this cause.

TREATMENT.

The treatment of acute poisoning consists in removing the poison from the stomach and bowels by washing and active cathartics, and in giving, even previously to this measure, large quantities of freshly-precipitated sesquioxide of iron. This is most conveniently prepared by neutralizing the tincture, or other solution, of chloride of iron with carbonate of sodium or magnesia. The quantity given should be large and the doses frequent.

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* Recent researches by the writer have shown that it is not uncommon to find traces of arsenic in the urine of persons in apparent health.

For further details the reader is referred to works on toxicology and to Wood's "Therapeutics."

For a case of chronic poisoning there is no specific remedy, other than removing the cause. Where this is arsenic used for therapeutic purposes, it is not always enough to withdraw the medicine when the first symptoms of poisoning show themselves, partly because several weeks must elapse before the whole of the arsenic can be eliminated, and partly because the symptoms occasionally go on increasing in severity without continued ingestion. It is, therefore, safer to anticipate trouble by discontinuing the treatment for a time every few weeks, especially if the doses are large.

Iodide of potassium is usually given, under the belief that it increases the rapidity of the elimination of arsenic. It is quite possible that this effect is produced, but there is not the same reason to expect benefit from this remedy as in the case of lead poisoning. There is, however, good reason to believe that saline diuretics in general increase the rapidity of the elimination of arsenic, as of poisons in general.

The arsenical paralyses are to be treated on the same principle as those of other origin—namely, by rest, sustaining regimen, and anodynes—through the acute period; later, by massage, carefully-measured exercises, and electricity. By a seeming paradox, arsenic in proper doses is one of the remedies recommended in the treatment of chronic neuritis, and one may readily conceive that it might be useful, in late stages, even where the neuritis was of arsenical origin. On account of the heightened susceptibility to arsenic, however, which is occasionally developed, it would undoubtedly be safer to avoid this remedy and trust to other alteratives, such as iodide of potassium, cod-liver oil, and iron.

Where the poisoning comes from arsenical wall-papers, these should be wholly removed, and not merely covered in by varnish or another paper.

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LEAD POISONING.

INTRODUCTION.

The history of lead poisoning is now in an important phase. Many of the masterly descriptions written in 1837 by Tanquerel des Plauches sound, as far as they go, as fresh and adequate as ever. But since that remarkable treatise was written, on which later descriptions have so strongly leaned, facts have gradually been accumulating which indicate that the subject is a far wider one than then appeared, and especially that long-continued exposure to minute quantities of lead causes symptoms which are in some respects quite different from those observed among the workmen at Clichy, and in the Paris hospitals.

Lead poisoning now ranks as one of the causes of chronic nephritis, and recently a few cases of endocarditis have apparently been traced to this origin. Tissue-changes have been discovered in the brain, and many chronic derangements of the nervous system, grouped under hysteria, neurasthenia, and the psychoses, find in lead poisoning one of their many sources of origin.

The important advances in other departments of pathology, and especially in the study of other poisons, both mineral and zymotic, have thrown new light upon the action of lead. Thus, through the important discovery of toxic multiple neuritis, and its relation to diseases of the spinal cord on the one hand and to the primary myopathies on the other, our insight into the nature of lead poisoning has had its share of gain. Still more important is our better recognition of the part played by poisons in general in the production of structural disease. To speak only of the spinal cord, we now know that locomotor ataxia and other of the spinal scleroses are due in a large proportion of cases to syphilis, while chronic ergot and lathyrus poisoning lead to analogous forms. No one can to-day doubt that if the tissues and organs of excretion could be kept free from inorganic, organic, and zymotic poisons a long step would be taken towards insuring their health.

Furthermore, it has for some time been known that alcohol increases the toxic action of lead, and it is worthy of inquiry whether the same may not be true of other general tissue-poisons, such as arsenic.

Under these circumstances, and in view of the fact, which will be dwelt upon later, that both lead and arsenic can frequently be found in the excretions of persons not exposed to them by their occupations, the study of these poisons, as acting alone or conjointly, should be approached in a spirit at once expectant and critical. It may be that habituation removes most of the danger, but our rules for determining whether the safety-line is being overstepped need constant revision.

The justification for the present article is found not in the abundance but in the meagreness of the present accumulation of facts relating to children, and the importance of taking steps towards increasing it. The table at the end gives most of the cases which the writer could find in the literature at his command that were reported in sufficient detail to be of value. Additional cases are referred to in the course of the article.

ETIOLOGY.

The most important of the sources of poisoning to which children are exposed are contaminated drinking-water, and coloring-matters. These will be studied more at length farther on. Less frequent but still important sources are glazed cards, acid fruits and vegetables put up in soldered tins, chocolate and other articles wrapped in spurious tin-foil,[1] articles cooked in vessels with lead glaze, contaminated cider, the vapor of freshly-painted rooms (see Table, Cases 15, 16, 18), and, in the case of infants, the milk of nurses poisoned with lead from the use of cosmetics, etc. The prolonged use of lead washes and ointments on excoriated surfaces has repeatedly given rise to poisoning, and the medicinal employment of lead compounds has occasionally had the same effect. A case is quoted by Taylor from Dr. Letheby,[2] of a child who died from taking for nine weeks one-fifteenth of a grain of lead acetate two or three times daily. The first symptoms were colic, constipation, and fetid breath. The child then lost flesh and the limbs became paralyzed. Death occurred two days after the nine weeks, in convulsions and coma. Another case showing great susceptibility to acetate of lead in medicinal dose is reported by Pick. Taylor[3] cites the case of an infant, previously healthy, who was bathed daily for a long period in the distilled water obtained from a leaden pipe connected with a steam-boiler. The child grew weak and "almost paralytic." Neither final result nor reference is given.

Red lead enters largely into the manufacture of articles of rubber, especially of the heavier sorts, and has occasionally been found in the rubber nipples of nursing-bottles. The stoppers of nursing-bottles are also occasionally made in part of lead, and this is said to have given rise to contamination of the milk.

The children of lead-workers often inherit seriously degenerated nervous systems, and it is probable that a careful search would discover analogous cases among the children of artisans, such as painters, whose trades furnish cases of serious poisoning, and even among those of persons poisoned with drinking-water and the like.

Constantin Paul[4] reported in 1860 a careful analysis of one hundred and twenty-three cases of pregnancy among female lead-workers, showing the extreme frequency of miscarriages, premature births, and still-births, and the great mortality among the living children in the first few years. Sometimes the mother, and sometimes the father, of these children was a lead-worker, but the influence of the mother was much the greater of the two.

The interesting report by D. D. Stewart,[5] of Philadelphia, on the cases of poisoning by lead chromate used to color cake and buns, contains the following statement: "All of the five infants born of mothers exhibiting symptoms of lead poisoning during gestation had convulsions,—four, within two months after birth. Three of the five died in them. Another infant, born in July, 1888, of a mother who had pronounced lead poisoning during the early months of pregnancy, died in the fourth month in convulsions." The researches of Roque[6] and Berger[7] give further evidence that children of lead-workers who survive the early months are apt to be idiotic or epileptic.

It has not been positively shown, so far as I am aware, that life-long epilepsy of the ordinary type originates in this way, or through acquired lead poisoning; but there is reason to think that it might do so, if the opinion which has been expressed by Gowers and other writers is correct, that epilepsy induced by lead poisoning is liable to last for a long time after the removal of the cause. Further evidence on this point will be given under Symptomatology.

Of great practical interest, as furnishing a suggestion for future observations, are three cases reported by Swann,[8] where sterility and abortion were thought to be due to lead poisoning from drinking-water. There were scarcely any other signs of poisoning, but lead was apparently present in the tissues, and the patients bore healthy children after removal from exposure.

By far the most important of all the sources of domestic poisoning is drinking-water, the danger being greatest in country places where force-pumps with lead pipe are used, the end of the pipe resting in the well, and where, as is still often the case, the water is conveyed in lead pipes from wells or springs thirty, forty, or fifty feet distant from the house.

Even in our large cities it is possible to find traces of lead at any time in water that has stood for some hours in the lead pipes of private houses, the coating of insoluble salts forming but a partial protection. The danger is increased if the pipes siphon each other and suck air, as is almost always the case to some extent; or if a softer water is introduced; or if the supply runs through bogs or marshes yielding vegetable acids or other organic compounds, a condition which a rainy season sometimes temporarily produces.

Several English towns have been visited by sudden and severe epidemics of lead poisoning in this way. In tracing out the cause of a recent epidemic at Bacup, Dr. John Brown[20] found reason to believe that free sulphuric acid was the dangerous ingredient of the water, and suggests the manner in which it is formed.

It is true that the danger attending the use of lead pipes is largely removed if the contaminated water is entirely run off, but it is not commonly realized how long a time is required for this (probably not less than fifteen minutes for a pipe fifty feet long), nor how soon traces of lead may again be found. It is highly probable that it is from this source that the greater part of the lead comes which, as will be shown later, is to be found in traces in the tissues of a considerable proportion of the community.

The next source of domestic poison of greatest importance is from the chromate of lead used in coloring-matters. This substance has long been used to color cloths and papers, toys, sweetmeats, butter, and many other substances, but cases of poisoning from it have been considered rare, the greater number affecting weavers working with yellow cloth or yarn. In cases of acute poisoning the symptoms seem mainly due to the action of the chromium element; in the chronic cases, to the action of the lead.* Several cases of the former kind, occurring in children, are on record in the older literature (see below, and in the Table).

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* See W. B. Hills, Wood's Hand-Book of the Medical Sciences, article "Chromate of Lead."

A notable impulse has been given to the subject within the past few years by several important investigations and reports from Philadelphia, by Dr. D. D. Stewart and others. Seventy-nine cases of poisoning were traced to lead chromate used by a number of bakers to color cake and to give a glaze to buns. These cases will be studied under Symptomatology.

PATHOLOGY.

The Frequency with which Lead is Found in the Tissues.—Some of the earlier chemists of this century (Devergie and others) believed that lead was a regular ingredient of the tissues of men and animals. This is not the case, but, as has been pointed out recently by Gautier,[9] who gives a formidable list of the domestic sources of poisoning, there are so many ways by which small quantities of lead may find entrance into the body that it is almost impossible to keep wholly clear of it, though, as a rule, no more is absorbed than is eliminated.

In the course of the past few years, Dr. A. M. Comey and Dr. C. P. Worcester,[10] both of Harvard University, have analyzed, at my request, the urine of more than one hundred and fifty persons not presenting symptoms of lead poisoning, and living mainly in the neighborhood of Boston, and have found traces of lead in about twenty-five per cent. of them. It is not, of course, asserted that these figures would apply to the community as a whole, but any one attempting to control them should use a process as delicate as that followed in these researches. In an appendix to this article there will be found a description of the method of analysis formerly used, and also a paper by Dr. Worcester, pointing out certain chances of error, and the means adopted by him, after long and careful study, to obviate them. The number of children represented in these analyses is as yet small, but it will be increased in further investigations.

One group of cases, the patients being a father, a mother, and three children, who were under the care of a colleague of the writer, is especially interesting in this connection, as showing the importance of taking lead poisoning into consideration in obscure cases, and as indicating the difference in susceptibility between different members of the same family. The water used by this family at their country house, where they spent a considerable part of the year, ran for some distance through lead pipes, and was found to contain a large quantity of lead. Repeated analyses showed that lead was present in considerable quantities in the urine of two of the children, the father, and the mother, though, for some reason not easily to be explained, not in that of the third child. All these children were in fair health. One of them, however, had been very subject to sharp attacks of gastric indigestion, pain in the left side, and vomiting, preceded by sore throat, and also to constipation, which was worse during the attacks of indigestion. When the lead was found, small doses of iodide of potassium were given, but, as they seemed to precipitate the attacks above mentioned, they were, after a number of trials, discontinued by advice of her physician, who believed her attacks to be due to imperfect power of digestion. A possible cause for this imperfect digestion was, obviously, the lead. The water was abandoned, and she became, finally, perfectly well, but she still has to be careful with her food. Another child, a sister, in whose urine, also, lead was found, is said to have had a few attacks of indigestion and occasional clay-colored stools. A third child, a boy, who was thought to have drunk even more of the water than his sisters, had no lead in his urine, according to two analyses, and presented no symptoms. The father and mother were perfectly well, though their urine contained a considerable amount of lead.

The above statistics, taken as a whole, would seem to indicate not only that lead is often found in small quantities, but also that it is usually harmless. When, however, it is remembered that out of a whole family exposed to the same source of poisoning—drinking-water, for example—one may be severely poisoned and the rest not at all, that we are wholly without knowledge of the grounds for this great difference in susceptibility, and only imperfectly acquainted with the manner in which lead acts, and that symptoms, or groups of symptoms, are occasionally met with for which lead poisoning seems to be responsible, yet which differ widely from the classical types, it is fair to conclude that the wide-spread presence of this subtle poison is a real danger to the community and one calling for further investigation.

It is known that an outbreak of acute symptoms sometimes occurs after long exposure to minute doses, and without apparent cause. This is sometimes attributed to the fact that the organs which eliminate the poison fail in their efficiency; but another cause, met with among adults, is a concurrent poisoning with alcohol, and perhaps other poisons.

ABSORPTION, ELIMINATION, AND ACTION ON THE TISSUES.—Lead, whether in a metallic state or as a salt, may be rendered soluble and absorbed by the fluids of the digestive and respiratory tracts. It may also penetrate the skin, when rubbed on in the form of ointment, as for cosmetic purposes, or applied in solution to abraded surfaces; and there is a fair amount of evidence to show that even when handled for long periods in metallic form it makes its way through the epidermis, perhaps after decomposition by the sweat, sufficiently to exert local action on the skin and its nerves. It remains stored up in the body, mainly in the liver, but in the central nervous system and a large number of other organs as well, sometimes more in one, sometimes more in another, this being probably in part dependent upon the period at which the examination is made and the character of the exposure.

It readily combines with albuminoid substances to form an albuminate, but Marshall[11] thinks it must be stored up in the tissues in some less soluble form than this, or it would be more rapidly eliminated. Whether it is entirely inert, except when circulating with the blood or when in process of elimination, is a point which is in need of further study.

It is eliminated mainly with the urine and the bile, but to some extent with other secretions and excretions, including the milk, which may contain enough to be a source of danger to infants at the breast.

The anatomical changes due to lead have as yet been studied mainly in cases of severe poisoning. It is not yet known whether they occur from prolonged ingestion of quantities too small to give rise to symptoms of recognizable character. There is, however, a fair amount of evidence that important series of changes in one organ, as the kidney, may be set up and progress without corresponding lesions in other organs.

The general character of the changes which it produces in the body is well indicated by the recent investigations of Cöens and D'Ajutolo.[12] These experimenters find that there is scarcely an organ or a tissue that remains unaffected. The action of the lead is first on the parenchyma of the organs involved, then on the blood-vessels; and it eventually leads to new formation of connective tissue. Its action is very slowly progressive, and attacks the different structures in foci.

The changes in the liver-cells, and in the glands of the digestive tract, are described as a granular degeneration of the protoplasm. The nuclei undergo a vesicular degeneration. The apparent atrophy of the liver-cells which had been described by Friedländer and others was not observed. The changes in the kidney were, first, parenchymatous degeneration, with glomerulitis and hyaline degeneration of the vessels; finally, interstitial nephritis. The changes in the muscles were analogous to those described for the liver and kidney.

The able researches of Maier[13] indicate that, besides many other organs, the cortex of the brain is liable to suffer from this process of sclerotic degeneration.

Lead has been found by Raimondi[14] in the marrow of the long bones; and this is of special importance in view of the fact that lead poisoning gives rise to changes in the blood very similar to those met with in pernicious anæmia. (Malassez, Gowers.) This anæmia seems to underlie the complex state known as cachexia, which forms one of the prominent symptom-groups of lead poisoning. Another cause of this state is impaired digestion, the anatomical basis of which has been fruitfully studied, especially by Maier (*loc. cit.*), who found parenchymatous changes in the mucous and submucous coats of the stomach and intestines, and sclerotic degeneration of the submucous and mesenteric ganglia.

Another important series of changes are those affecting the peripheral nerves. These are sometimes undistinguishable, except by their distribution,—and not always in this way,—from the ordinary so-called "multiple neuritis," such as is due to alcohol, arsenic, and anæmia, and many other causes, as well as to lead. It has been suggested that these changes may be secondary to an impairment of the trophic functions of the spinal cord. This is, however, as yet wholly unproved.

The experiments of Gombault upon guinea-pigs point to the occurrence of changes characteristic of lead, and forming what has been called a "peri-axillary neuritis," in which the medullary sheath is affected more than the axis-cylinder, and that, too, in a segmental manner, relatively healthy portions of nerve intervening between the diseased portions. It is a significant fact, though one not to be utilized without caution, that the guinea-pigs which showed this form of neuritis induced by chronic lead poisoning were not paralyzed. It is worthy of inquiry whether these changes in the myeline, if they occur in man also in anticipation of actual paralysis, may not account for the fact that the electrical reactions of the nerves of persons poisoned with lead often suffer before any sign of paralysis has made its appearance. Another explanation that suggests itself, however, for this fact is that in such cases only a portion of the fibres going to a certain muscle may be destroyed; and this is more in harmony with the observation of Erb, confirmed by Gowers,[15] that pre-paralytic changes in the muscle-reactions (partial degeneration-reaction) are sometimes met with. It is to be remembered also that, according to Harnack's[16] researches, the typical paralyses of lead are usually and primarily due to the direct action of the lead upon the muscles.

Besides these neuritic changes, there is reason to believe that subacute processes occasionally occur in the spinal cord; but it is probable that these are not the cause of the typical "extensor-paralysis," but represent one of the later manifestations of serious poisoning (Schultze).

A number of cases have come to my knowledge pointing to the possibility that chronic lead poisoning may give rise to typical system scleroses of the spinal cord. But many additional observations are needed before this can be considered as certain.

Lead also affects the nutrition of the brain, both directly, and indirectly through the blood and the blood-vessels.[22]

Another highly important series of changes are those which affect the kidney, the character of which has already been indicated.

Lead poisoning is held by most writers of the present day to be a cause of chronic nephritis (see above), and it is not impossible, considering the great frequency of this disease and the difficulty of tracing it to any other origin, that long-continued exposure even to minute quantities of lead may sometimes account for it. The acuter forms of renal degeneration noted occasionally in experiments on animals (Ollivier and others) have not been much observed clinically, except that albuminuria is known to occur as a temporary symptom.

Chronic interstitial nephritis is scarcely met with in children, and we do not as yet positively know anything about the action of lead upon their kidneys.

Among the most important of the observations of the last few years are those pointing to valvular diseases of the heart as sometimes directly traceable to lead. Duroziez[17] pointed out, in 1885, the frequency of mitral disease among painters and other persons employed in lead, and

Stewart (*loc. cit.*) devotes some space to its discussion, and reports two cases.

It is a remarkable fact that, in spite of the accumulation of lead in the liver, and the degeneration of the liver-cells and increase in the connective tissue, chronic cirrhosis has not been traced to this source, in spite of the fact that various other poisons (malaria, alcohol, phosphorus) are believed to cause it. It should be said, however, that the etiology of hepatic cirrhosis is by no means easy to make out in every case.

In the foregoing descriptions lead has been spoken of as causing more or less permanent changes in the nutrition of the different tissues and organs, but it should not be forgotten that it acts also, in all probability, as a temporary poison, without necessarily giving rise to gross tissue-change (Harnack), and it is perhaps in this way that the occasional rapid onset and the almost equally rapid disappearance of paralysis and other symptoms are to be explained.

SYMPTOMATOLOGY.

Relative Susceptibility of Children.—Children of every age have so often been seriously poisoned by exposure to small quantities of lead that their sensitiveness to single doses of moderate size may be considered as greater than that of adults. On the other hand, it is noteworthy that the reports of certain of the epidemics, as they may be called, of drinking-water poisoning which have occurred in England and elsewhere state that the proportion of children affected severely enough to require medical care was very small.

Thus, Dr. Brown,[19] in reporting on the epidemic at Tredegar, expressly says that he saw no serious symptoms in any person under eighteen years of age, although he observed fifty-two cases of well-marked poisoning, and two hundred cases with lead-line on the gums, in persons older than this. He does not say whether any children were seen with the lead-line. This seems in general to be rare, owing, no doubt, to the fact that the teeth of young children rarely collect tartar; but forty-two cases where this line was seen in children under fifteen years of age are reported by Dr. John Brown[20] out of three hundred and three cases collected during the past year in an epidemic at Bacup. Five of these children were under five years of age; eighteen were between five and ten. The remaining cases are tabulated as follows, as regards the frequency of the lead-line:

10 and under 15 years of age . . .	20	25 and under 50 years of age . .	144
15 " " 25 " " " . . .	68	50 years and upward	48

Of thirty-eight persons belonging to the household of the royal family at Claremont, of whom thirteen were severely poisoned through the drinking-water, no children were attacked, though they were present to the number of eight.

If these facts really indicate a relatively slight liability on the part of children to poisoning from minute doses often repeated, it is a matter of great interest, possibly indicating a more active power of elimination on their part. Several cases of drinking-water poisoning in children are, however, upon record, and the subject must be considered as requiring further study.

The *Lancet* for January, 1889, contains the report of a case of paralysis of all four extremities from the fruitful source of the Sheffield drinking-water in a girl of eight, and a similar case has been reported by Seeligmüller.

The history of the group of three children cited under Pathology is interesting in this connection. It will be noted that in the urine of two of the children lead was found in large quantities, while in that of the third it was absent. Possibly this was due to the fact that this patient, being a boy, lived a more active life than the others, who were girls, and that the perspiration helped to eliminate the lead.

There is a certain amount of evidence indicating that with children, as well as with adults, previous exposure to poisoning by lead in minute doses, insufficient to cause definite symptoms, acts as a contributive cause for an outbreak of acute symptoms on further exposure. The cases reported by Chapin (see Table) were perhaps an instance of this. The final exposure, leading to prolonged and serious sickness, consisted only in a few days' stay in a freshly-painted room; but this had been preceded by an exposure of two years to the small amount of emanations arising from a pot of fresh paint which was kept in a room adjoining that in which the children mainly lived. It has frequently been noticed with regard to workers in lead that they become more susceptible after one attack of poisoning.

ACUTE AND CHRONIC POISONING.—There is no material difference in character between the symptoms of acute and those of chronic poisoning, except for the fact that in the former case the local irritation of the lead compounds on the gastro-intestinal tract may cause an important series of direct and indirect symptoms.

Seeligmüller, in drawing up the differential diagnosis between lead poisoning and arsenical poisoning, says that paralysis does not occur from acute lead poisoning. This is certainly incorrect except as a matter of degree, as several cases cited by Taylor, and the physiological experiments of Harnack, sufficiently indicate.

In the following description of symptoms special reference is had to cases of chronic poisoning; and those symptoms are chiefly considered which are apt to occur in children.

GENERAL CACHEXIA, with earthen hue of the skin, icteroid discoloration of the conjunctiva, loss of flesh and of appetite, etc., is, in a large proportion of the cases of chronic poisoning of both adults and children, the first sign of lead poisoning, and, combined with a variety of obscure nervous symptoms and a moderate amount of indigestion, such as might easily be overlooked, may remain for a long period, or even permanently, as its sole manifestation. An interesting group of cases of this sort has been reported by Dr. Robertson.[18] Twenty young girls, in an institution which had borne the highest reputation for the care and good health of its inmates, became, one after another, languid, pale, and sick in various ways, and did not recover until—after one or two had been attacked with severe colic and constipation—it was discovered that they were in the habit, while at play, of drinking the water from a lead-lined cistern, ordinarily used for washing. Absolute proof that these cases were due to lead poisoning is not furnished, but the water contained a large amount of lead, and after removal from this exposure the patients recovered and no new cases of illness occurred.

Occasionally the emaciation and general failure of nutrition are of an extreme degree, yet either without other signs of poisoning or out of proportion to them.

It is impossible to define with accuracy the clinical symptoms of lead cachexia in the very chronic cases which may occur from exposure to lead in minute doses. It has its source in a variety of disordered functions.

Disorders of Digestion.—The most common symptom of this class is colic. In spite of the opinion expressed by Soltmann,[30] that lead colic is rare in children, I find it frequently noted, and see no reason to think that its absence is characteristic. It was, however, absent in most of Stewart's lead-chromate cases. It is also important to note that it must in many cases be difficult to distinguish lead colic from the intestinal colic to which children are so largely subject. The colic in adults is usually most severe about the umbilicus, but often spreads over the whole abdomen. Constipation, for which diarrhœa is occasionally substituted, is an important confirmatory symptom. The abdomen is often retracted, but this need not be the case. Vomiting frequently occurs, and the vomitus may be yellowish in color even when other salts of lead than the chromate are the cause of the poisoning, though oftener in the latter case. In regard to the less definite kinds of indigestion we have, as yet, no satisfactory means of making the diagnosis, but if no other cause is discoverable, and if lead is found in the excretions, it will be worth consideration whether it may not be acting as at least a contributive cause.

Numerous cases might be cited from among adults to show the great variety of symptoms of indigestion for which lead is at least partially responsible. Thus, in a case reported by Tunzelmann,[21] vomiting, especially at night, was for some time the only symptom. Typical symptoms finally appeared, and eventually another patient in the same house likewise came down with paralysis. Blue line was present in both cases. In the *Lancet* for 1884, several cases are noted where the signs were thought to point to cancer of the stomach, and another where a distressing indigestion of years' duration, apparently due to lead, was the only symptom. Where the poisoning is due to chromate of lead and is acute, the dejections and vomitus are especially likely to be yellowish in color. Yellowish vomitus is, however, common in poisoning from other salts of lead besides the chromate, as Dr. Stewart points out in connection with his cases. The character of the indigestion in a group of children possibly poisoned by drinking-water, and in whose urine lead was found, is described under Pathology.

It is earnestly to be hoped that the character of the indigestion in cases of chronic poisoning in children will be carefully studied in the future. In the important group of lead-chromate cases reported by Stewart, anorexia, nausea, and vomiting were prominent, but the constitutional effects of the lead may have been complicated by the local action of the chromium, though in most of the cases the exposure had been going on for some time. Of the seven cases described, constipation, not of a high degree, had been present in four only, and slight colicky pains in only two.

SYMPTOMS AFFECTING THE NERVOUS SYSTEM.—**Motor Symptoms.**—The motor paralytic symptoms due to lead poisoning are, so far as we know, almost wholly of peripheral origin. It is possible that spinal disease sometimes plays a part in their production, as will be shown later; and occasionally, in the case of adults, cerebral hemorrhage and œdema, or cerebral degeneration due directly to lead, cause paralytic symptoms.

The peripheral paralyses may be divided, broadly, into (1) those where the loss of power is a direct consequence of disease of the nerves corresponding to the affected muscles; (2) those where the loss of power is the result of disease of the muscles themselves (Gowers's "primary atrophy:" compare Pathology).

The first of these groups may again be divided into those where the distribution of the paralysis is of the usual type,—that is to say, where the extensors of the fingers and hands are the parts mainly affected, the supinator longus and the extensor ossis metacarpi pollicis usually escaping,—and those where the symptoms suggest forms of generalized neuritis such as are due to alcohol, arsenic, and many other poisons.

The "primary atrophy" (hitherto studied only in adults) affects pre-eminently the small muscles of the hands, but often other groups as well. Its course is chronic, and the prognosis less favorable than in the neuritic or "degenerative" form.

The clinical analysis of these different forms of poisoning might be carried much farther if space permitted. For an excellent account of them the reader is referred to Gowers's "Manual of Diseases of the Nervous System." It is now generally recognized that the selective preference of lead for certain special nervo-muscular groups is relative only, many nerves and muscles apparently unaffected being really diseased; and a mistake in diagnosis might easily be made by attaching too much importance to the presence or absence of extensor paralysis. Sometimes the shoulder-muscles are first or mainly affected.

It is a striking fact that whereas in adults the paralysis in the vast majority of cases affects the upper extremity, and especially the extensors of the hand, either alone, or far more than those of the foot, this appears not to be the case with children. In every instance that the writer has found described or has seen, the legs have been affected as much as the arms, or more, as in paralysis from alcohol and arsenic, the symptoms generally appearing first in them. The type of the paralysis seems in other respects to be the same as in adults; that is to say, the extensors are affected more than the flexors, as occurs in multiple neuritis of every origin, except sometimes in cases of arsenical poisoning, where the flexors may be affected nearly or quite as much as the extensors.

The details of the physical examination in the reported children's cases are not sufficiently exact to enable us to say whether the particular muscles which usually escape in adults are unaffected in them or not.

It would be premature to offer an explanation of this relatively greater tendency of lead poisoning in children to affect the lower extremities, since the cases on which the observation is based are still few in number. It will, however, be an interesting matter for further inquiry, and, if the observation is confirmed, it may lead to conclusions of general interest.

Ataxia of the extremities, which has been noted a few times in adults, has not as yet been met with, to my knowledge, in young children, except in the form of chorea.

In adults, and also in certain animals, paralysis of the larynx is occasionally met with (Seifert and others), and this may occur without other symptoms of note. I am not aware that this symptom has been seen in childhood.

Sensory symptoms are met with, both of the type of those due to neuritis and of the cerebral type (especially hemianæsthesia), and, furthermore, local anæsthesia is occasionally due to the direct action of lead upon the skin. No cases of either of these sorts have as yet been described as occurring among children.

Symptoms affecting the Visual Apparatus.—A number of cases in adults are on record where neuritis or atrophy of the optic nerve was observed, and where the motion of the eyes was affected. I am not aware that any case of neuritis or atrophy has been reported as affecting a young child, although unexplained cases of neuritis are now and then met with which might possibly be of this origin. Dr. Miller, of New Orleans, has reported a very interesting case, of drinking-water origin, where double vision and amaurosis, associated with serious and prolonged paralysis, occurred in a "young lady," whose age is not given. I have myself seen and reported a case of partial paralysis of the ocular muscles of the same origin, associated with dizziness and ataxic symptoms involving the extremities, in a girl of fifteen. It is probable that this, like the corresponding cases of arsenical origin, is one of those instances where lead acts as a cause of generalized neuritis.

Encephalopathic Affections.—The four forms of cerebral symptom-complex adopted by Tanquerel—the delirious, convulsive, comatose, and mixed forms—have been essentially accepted by all later writers. The whole subject has recently been reviewed by Dr. A. Westphal,[22] of Berlin, and a pathological classification offered, showing the various ways in which the brain may suffer injury. So far as children are concerned, we have to deal mainly with the direct toxic action of the poison, and with the effects of impaired nutrition. Among infants and very young children the tendency to convulsions as a result of acute lead poisoning is, as might be expected, greater than with adults. I find also a record of two cases of children, exposed by their occupation to lead, where death occurred, preceded by a series of rapidly-recurring epileptic attacks, besides other cerebral symptoms. (See Case 28 of Table.)

A case is reported by Kersch[23] where convulsions occurred in a child of two years from eating food served in a dish made of an alloy containing fifteen per cent. of lead. A large number of the children in Dr. Stewart's series of lead-chromate cases died in convulsions, and one of them had mild delirium.

One of the most practical and important questions connected with this group of symptoms seems to be whether true epilepsy of the usual type may originate in lead poisoning, unconnected with other symptoms sufficiently marked to indicate the presence of the poison. Gowers gives the opinion, without adducing the facts upon which it was based, that epileptic attacks originating in lead poisoning may continue for a long time after the cause has been removed. Gaillard[24] also says that epileptic attacks starting in this manner may become habitual.

I have myself seen and reported a case which is very important in this connection. It is that of a young woman of twenty-seven, poisoned by drinking-water drawn through twenty feet of lead pipe from a sunk well, the end of the pipe resting in the well. Her first symptoms consisted in failing health and severe pains in various parts of the body, especially the abdomen, the cause of which was not recognized. After a time she began to have "fainting-spells," characterized by loss of consciousness of two or three minutes' duration, and preceded by slight dizziness. She suffered also from diffuse headaches and from dizziness. After these symptoms had been present for two years or more, she had a violent cerebral attack, with prolonged loss of consciousness, convulsions, and amaurosis. This was followed by another attack, after which came characteristic lead palsy of the arms, and finally localized atrophy, from which she never entirely recovered. The epileptic attacks did not, however, recur, so far as I know, after the cause was removed and treatment instituted; and this is certainly the usual experience.

The question arises whether, in such a case as this, if the character of the exposure had been less severe, or if the patient had accidentally been removed from exposure before the final outbreak occurred, the case might have progressed, by virtue of habituation, like one of ordinary epilepsy, and the real cause passed unsuspected.

In the epidemic at Tredegar, already cited, one case of temporary epilepsy associated with other signs of poisoning was met with out of fifty-two cases of serious poisoning. Similar cases are reported by Leidesdorf[25] and by Inman.[26] Kronig[27] gives an analogous case, and a few others are on record.[31] It has been stated that in the epileptic attacks due to lead the loss of consciousness occurs suddenly, without aura, but this, even if invariably true, is not pathognomonic.

I have seen three cases of chronic epilepsy where lead was found in the urine, but, for the reasons stated under the head of Diagnosis, I do not consider this a sufficient indication of their origin.

I shall now note, without dwelling on them at length, a number of symptoms of rarer occurrence which have been mainly observed among adults, but which might readily occur among children.

Hysteria.—A number of the French writers, following Charcot, believe that lead poisoning must count as at least an exciting cause of hysteria, bearing the same relation to it, for example, as traumatism, psychical influences, and the like; and that the hemianæsthesia and some of the paralyses, apparently of cerebral origin, which have been described in cases of lead poisoning, are induced in this way.

Insomnia has been noted frequently in lead poisoning of adults, and sometimes forms an obstinate symptom and one difficult to trace to its origin, especially as it is not necessarily associated with marked signs of poisoning.

Neuralgic Pains.—It is well known that pain in various parts of the body besides the abdomen is a common symptom in the early stages of lead poisoning. This pain is, in the great majority of cases, dull in character and probably neuritic in origin. Now and then, however, cases are met with where the pain, instead of being of this dull sort, is frankly neuralgic in character and follows the distribution of the peripheral nerves. This is important, as its origin might not be suspected.

Tremor is an important symptom, and one which sometimes occurs alone. It is especially noticeable in the hands, and is, with adults at least, either fine or coarse in character.

Chorea has occasionally been reported in cases of lead poisoning both of adults and of children. It is probable that its relation to lead is an indirect one, though Harnack observed movements which he considered to be choreiform in animals poisoned experimentally with lead.

CUTANEOUS AFFECTIONS.—The skin of patients with chronic lead poisoning is usually dry and harsh. Cases have been noted, however, where distinct inflammatory affections of eczematous character were present.

FEBRILE CONDITIONS.—Gowers notes the fact that a febrile state is occasionally seen in lead poisoning, especially with the onset of acute nervous symptoms, and that this is especially true of children. In Case 4 of the Table the temperature ran up to 100° F. as the paralysis of the extensors came on.

RENAL AFFECTIONS.—In the chronic poisoning of adults, renal changes play, as is well known, an important part. Published records with regard to this point in the case of children do not exist, and observations bearing on the subject are greatly needed. The renal changes hitherto considered characteristic as regards adults are those described under Pathology as leading to chronic interstitial nephritis. This is a rare form of disease in children, although the first changes leading to this condition might perhaps occur in childhood.

Some of the encephalopathic symptoms observed in adults have been considered as indirectly due to the renal and associated arterial changes. It is, indeed, more than probable that hemorrhage and well-marked uræmic symptoms are of this origin. The same cannot be said, however, of the greater number of cerebral symptoms due to lead, and this view is borne out by the fact that similar symptoms are observed in children and young persons. It should be said, to be sure, that the theory has been advanced that uræmia due to lead may perhaps occur in consequence of spasm of the renal arteries without gross structural change; but proof of this is wanting.

LEAD-LINE.—This is probably observed less often in children than in adults. Nevertheless forty-two such cases, where it was observed during an epidemic in Bacup,[20] are on record. Here it was associated with a peculiar form of caries of the teeth. Two of the children were under five years of age. Separation of the gum from the tooth by tartar is less common in children than in adults, even where the nutrition of the teeth is bad, and it is probably to this fact that the usual absence in them of the blue line is due.

DIAGNOSIS.

For the details of the diagnosis of *acute lead poisoning* the reader is referred to special works on toxicology. In general, it may be said that the pathological conditions do not differ much from those of chronic poisoning, except that the results of irritation of the digestive tract are often superadded, and that, partly as a consequence of this irritation, and partly from the toxic action of the lead, the cerebral symptoms and general prostration are liable to be prominent and serious. It sometimes happens that acute cerebral symptoms come on suddenly in the course of chronic poisoning.

The possibility that epileptiform attacks due to lead may precede by a long time other well-marked signs of poisoning should be borne in mind.

The commonest sequence of symptoms in cases of *chronic lead poisoning* is, first, a general languor, with a certain degree of anæmia and debility; then, constipation (or diarrhœa) and colic, and the dull muscular "arthralgic" pains; later, the typical wrist-drop or other form of paralysis.

The encephalopathic symptoms, the primary myopathies, and the signs of subacute myelitis commonly belong to a relatively late period and imply advanced degrees of saturation. In bad cases, like those of the two children described by Chapin (Table, Nos. 15 and 16), ulceration of the gums and caries or loosening of the teeth may make their appearance after a time. Last of all, the chronic sclerotic affections, of which interstitial nephritis, arteritis, and valvulitis are the best known to us, appear, and may progress entirely independently of the continuance or non-continuance of the exposure. It is possible that some of the neuroses also—the psychoses, hysteria, neurasthenia, epilepsy, etc.—to which the poisoning occasionally gives rise may over-last the exposure, for a time or permanently, either because of the establishment of a morbid habit, or through the persistence of chronic changes of the nervous centres, or general impairment of the nutrition. The "blue line" may be present throughout, but is not necessarily accompanied by noticeable symptoms of poisoning.

The "typical" sequence is subject to striking and manifold variations. Almost any one of the symptoms may appear out of place, or may dominate the scene for a longer or shorter time; and this is true of children as well as of adults.

A greater or less degree of cachexia is almost invariably present, but many adult cases are on record where this sign was not present in any marked degree, the wrist-drop, the cerebral symptoms, or some other of the later signs appearing almost without warning. A number of the children with lead-chromate poisoning reported upon by Dr. Stewart showed convulsions and delirium * very soon after the first symptoms of intoxication.

Digestive Disorders.—Typical constipation and colic may be highly characteristic (see Symptomatology), and if obstinate and protracted should always excite suspicion. It is doubtful whether any description of the obscurer forms of indigestion can at present be given that would not tend as much to mislead as to aid the physician, and often the only safe course is to search for other signs. It is certain that the outbreaks of indigestion may temporarily cease, or recur paroxysmally, and that diarrhœa may occur instead of constipation.

If lead is found in the urine by a competent chemist, it is a confirmatory sign of great value; but it should be remembered that, with adults at least, the presence of traces of lead in the tissues is not incompatible with health. The "blue line" is oftener absent with children than with adults.

Paralytic Affections.—Wrist-drop, so far as our present evidence goes, is not to be expected in children so often as in adults as an isolated symptom; and, conversely, paralysis of the extensors of the feet appears to be relatively more common in them. In this respect the cases of lead-paralysis in children simulate those of multiple neuritis, as from arsenic and other causes, and the diagnosis would have to depend on other signs and on the clinical history.

Cases of lead poisoning with wide-spread muscular atrophy have been mistaken for acute poliomyelitis, and for progressive muscular atrophy, and *vice versa*. The former doubt is the only one of importance as regards children, and it would rarely, if ever, happen that a careful investigation into the history and surroundings of the patient, and a careful scrutiny of the nutrition, the gums, and the distribution of the paralysis, would fail to reveal the true state of the case. In case of doubt, the urine should be analyzed.

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* Oliver (British Medical Journal, 1885, vol. ii. p. 730) calls attention to the fact that encephalopathic symptoms, though usually of late occurrence, may occur very early, especially among women. The same fact is noted in the British Medical Journal, 1885, vol. i. p. 496, and elsewhere, and, indeed, was long ago pointed out by Tanquerel. The case of one of the workmen at Clichy is also on record, who suddenly became insane, while his history presented clear evidence of lead poisoning. I have seen a drinking-water case of great severity, where the encephalopathic symptoms were not, to be sure, the first to appear, but were preceded only by pains in the abdomen and elsewhere, and general signs of failing health, the cause of which was not recognized. (See above, under Epilepsy.)

PROGNOSIS.

The prognosis in a case of lead poisoning is usually fairly good, provided the patient does not die from an acute attack, and provided destructive changes have not already taken place in tissues incapable of much regeneration, such as the spinal cord, nor degenerative processes set in motion which have a tendency to spontaneous progression. These provisos are, however, of much importance. A guarded prognosis should be given at the outset of an attack of acute symptoms, whether these have occurred in the course of chronic poisoning or as the result of a single severe exposure. Paralysis of the neuritic type is usually recovered from, but the "primary muscular atrophy" is more often permanent, and it is not always easy to say, at first, with which condition we have to deal.

When the general nutrition has suffered deeply, absolute recovery cannot be counted on, though the patient is likely to improve very much. The prognosis is probably much better for children than for adults, but in Case 18 of the Table the strength of the limbs was not perfect at the end of two years. The prognosis of the convulsive attacks is considered under Symptomatology.

TREATMENT.

The treatment of an attack of acute poisoning is governed by the usual toxicological rules concerning the removal of the poison and the protection of the mucous membrane from irritative action. There is no special antidote. For further details the reader is referred to toxicological works.

In the treatment of a chronic case the most important indication is the removal of the poison from the tissues. The most efficient eliminating organ is the kidney, and, since it is known that the tissues gradually, though very slowly, free themselves, as a rule, from the poison, and that its presence may frequently be demonstrated in the urine, even when no special means have been taken to promote its elimination, it is reasonable to believe that this, like all other poisons given off in the same manner, is eliminated more rapidly if means are used which promote diuresis.

The iodides and bromides appear to increase the elimination of lead even more than can be accounted for by their diuretic properties. This is so markedly the case that it is often necessary to be cautious in giving iodide of potassium, lest the lead should be thrown into the circulation in large amounts and thus exhibit its poisonous action with increased violence. The action of iodide of potassium in promoting the elimination of lead is not, however, a uniform one. Experiments of Pouchet[28] and of Swete[29] have shown that a very rapid elimination takes place shortly after the drug is first administered, but that the amount steadily falls, to increase again if the remedy is suspended for a time and then renewed.

The symptomatic treatment needs no especial description, except so far as the paralyses are concerned. In the treatment of paralysis the use of strychnine in increasing doses, carried, if necessary, to the limit of tolerance, is thought to be of great value.

We have also at our command, in the different forms of electricity, massage, local exercises, and hydropathic applications, means for materially improving the nutritive conditions in the muscles and peripheral nerves. The general cachexia and anæmia call for the best treatment that can be devised to raise the general tone of the nutrition, but it is not necessary to specify these methods in detail.

The use of sulphur baths as a means of elimination has been practically abandoned. Where the skin contains lead, a sulphide will sometimes be formed on the surface, and the applications have, therefore, a diagnostic value; but the amount of lead got rid of in this way is unimportant.

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APPENDIX A.

CHROMATE OF LEAD CASES.

Reference.	Sex, Age.	Exposure.	Symptoms.	Result.	Remarks.
1. Leopold, Vierteljahresschrift für gerichtliche Medicin und öffentliche Sanitätswesen, Band xvi. S. 29.	9 weeks.	Dust from cloth colored with chrome yellow, the parents being weavers and working in the room where the child lived.	After three weeks' exposure the following signs appeared: fever, restlessness, several yellow fluid stools daily, redness of skin over chest and abdomen, parched lips, and, just before death, short respiration.	Death six or eight days after first symptoms.	Autopsy revealed marked signs of corrosive action in the stomach; 3.6 mm. of lead chromate were found in the lungs, none elsewhere. Symptoms probably were largely reflex.
2 and 3. Van Linstow, ibid., Band xxi. 60.	Male, $1\frac{3}{4}$ years and $3\frac{1}{2}$ years.	Both children ate several small objects made of gum tragacanth colored with lead chromate.	Vomiting, thirst, prostration, fever, congestion of skin of abdomen and breast; collapse; no diarrhœa or pain: in the case of the younger child diarrhœa and convulsions.	Death of the younger child in forty-eight hours; of the older in five days.	Marked signs of local irritation of gastro-intestinal tract; congestion of various organs; beginning fatty degeneration of liver. Symptoms probably mainly reflex, or due to decomposition of the lead chromate within the body. (W. B. Hills.) (*Loc. cit.*)
4. D. D. Stewart, Phila. Medical News, June 18, 1887.	Girl, $5\frac{1}{4}$ years.	Had eaten regularly and largely of buns the glazing on each of which contained about seven grains of chromate of lead.	Anorexia, nausea, and vomiting. After three days, violent general convulsions, followed in the course of eight hours by thirty-four more.	Death.	

5. D. D. Stewart, ibid.	Girl, 3¼ years.	As above.	Vomiting of greenish-yellow fluid, biliary in character; convulsions, which occurred at intervals for twelve hours.	Death.	This and the following four cases were in the same family.
6. D. D. Stewart, ibid.	Boy, 7½ years.	As above.	Breath offensive, tongue furred, epileptiform attack, followed the next day by a series of convulsions occurring at short intervals.	Death after twenty-eight hours.	
7. D. D. Stewart, ibid.	Girl, 12 years.	As above.	Listlessness and languor for several days, then nausea, vomiting, and headache, followed by two severe and general convulsions,—later by a larger number. Temperature never higher than 102½° F.	Death forty-two hours after first attack.	
8. D. D. Stewart, ibid.	Girl, 13¾ years.	As above.	Failing health and strength for several months; headache, general pains, nausea, and vomiting. Skin pale, with yellowish tint Slight colicky pains; constipation; finally mild delirium; blue line on a part of one gum.	Recovery.	
9. D. D. Stewart, ibid.	Boy, 9 years.	As above.	As in last case.	Recovery.	

INFANTS.

Reference.	Sex, Age.	Exposure.	Symptoms.	Result.	Remarks.
10. Bouchut, Gaz. des Hôp., 1873, vol. i.	8 days.	"Eau de Delacour" on nipples of nurse: child nursed for a few days.	Colic, constipation, pain on pressure on abdomen; prostration.	Death after twelve days.	
11. Cited by Anst, Arch. für Kinderheilkunde, 1886, viii. 73 (Deut. Chem. Zeitschr., 1886, vols. i. and ii.).	Infant.	Cosmetics in large quantity on face of nurse.	Severe colic, great debility; skin of bluish color.	Recovery.	
12. Löwy, Wiener Med. Presse, 1883, p. 1542.	5 weeks.	Nurse poisoned by cosmetics on face.	Colic, persistent crying; livid appearance.	Recovery.	
13. Löwy, ibid.	5 weeks.	Goulard's water on breast of nurse.	Colic.	Recovery.	
14. Löwy, ibid.	. . . . .	Lead attachment to stopper of nursing-bottle.	Colic.	Recovery.	

CHILDREN AND ADOLESCENTS

Reference.	Sex, Age.	Exposure.	Symptoms.	Result.	Remarks.
15. Chapin, New York Medical Record, 1884, vol. v. p. 17.	5 years.	A pot of white lead and turpentine was kept during two years in room adjoining that in which children mainly lived; finally the room in which they lived was freshly painted and they were exposed to the emanations for eight days before symptoms began.	Pain in abdomen, which intermitted, worse at night, lasting four weeks; then pain in legs, with weakness; finally almost complete paralysis of legs. Improvement after a few days; after four weeks more, return of abdominal pains and increase of paralysis of legs, followed again by improvement; the extensors weak. After three months, symptoms worse again, and blue line in case of oldest, not of youngest, child. Gums became soft, spongy, and ulcerated; teeth greatly loosened: condition at end of a year even worse than described. Lead in urine.	At last report, partial recovery and steady improvement.	
16. Chapin, ibid. (brother of last patient).	7 years.				
17. Chapin, ibid.	2¼ years.	Cut thumb bathed for a number of days with solution of lead acetate ʒ ii to Oi.	Complete paralysis of legs.		
18. Records of Children's Hospital, Boston, July, 1886.	Girl, 6 years.	Interior of house painted throughout. Five canary-birds died shortly afterwards.	Almost immediately after this, vomiting, which continued intermittently for two weeks; weakness of legs, toe-drop, waddling gait, coming on after vomiting. About a month later, weakness of fingers and wrist noted; extensor muscles most affected in both arms and legs; knee-jerk absent. Faradic reaction of left tibialis anticus much diminished, and contraction feeble; no marked increase of galvanic irritability; still some weakness at the end of a year. Re-examined two years later: no paralysis, but legs easily fatigued.	. . . . . .	Six months after painting of the house and sickness of this child, an older child was attacked with acute nephritis and died. Some wall-paper obtained recently, and which at that period covered the walls, is found to be arsenical. The patient poisoned by lead had been somewhat rachitic, and had suffered much from protracted attacks of vomiting. When four years old she had a convulsion.

CHILDREN AND ADOLESCENTS.—*Continued.*

REFERENCE.	SEX, AGE.	EXPOSURE.	SYMPTOMS.	RESULT.	REMARKS.
19. Miller, New Orleans Med. and Surg. Jour., 1873–74, i. 198.	4 years.	Drinking-water.	"Paralytic state."	Death after four months.	Both of these cases were in one family. Three other children in the same family were not affected. Many of the symptoms in the last case came on after removal from exposure.
20. Miller, ibid.	3 years.	Drinking-water.	Much impairment of general nutrition.	Death after eight years, in spite of removal from exposure.	
21.	Young lady.	Drinking-water.	Serious impairment of general nutrition; diplopia; "paralysis" complete for three years.	Recovery.	
22. Letheby, Pharm. Jour., Dec. 1845; cited by Taylor, On Poisons, 3d ed., p. 409.	6 years.	Took lead acetate, gr. $\frac{1}{15}$, two or three times a day, for nine weeks, in a quack medicine.	Loss of flesh; colic; constipation; black and offensive stools; paralysis of limbs; convulsions; coma.	Death two days after ceasing to take the medicine.	
23. L'Union Méd., 1857. (Cited in Bull. Gén. de Thérap., vol. lii. p. 381. Author not given.)	Boy, 12 years.	Drank habitually of cider containing about 0.25 of lead in each 1000.	For two weeks loss of appetite, bad taste in mouth, constipation, violent pain at epigastrium, eventually spreading to the whole abdomen, retraction of belly, coated tongue, blue line. No vomiting or pains about joints; no paralysis.	Recovery.	The father and mother of these children had both been severely poisoned in the same manner.
24, 25, and 26. Ibid.	Three other children, from 4 to 14 years old.	As above. Less degree of exposure.	Colic and constipation.	Recovery.	

27. Duchenne fils, quoted by Seeligmuller; cited in Handb. der Kinderk., Gerhardt, vol. i. p. 116.	3 years.	Drinking-water.	Paralysis simulating poliomyelitis.		
28. Charteris, Lon. Lancet, 1881, vol. i. p. 331.	Boy, 16 years.	Worked for five years in a pottery, where he was obliged to handle freshly-glazed vessels.	Health fairly good until seven weeks before death; then pain, constipation, wrist-drop, cramps in thighs; finally epileptic attacks, recurring to the number of nine and ten in two hours; dilated pupils.	Death in four hours after onset of final symptoms.	In the London Lancet for 1888 a case similar to this is reported, affecting a girl of fifteen years, but not with sufficient detail to make it of great value.
29. J. J. Putnam, Bost. Med. and Surg. Jour., Dec. 22, 1887.	Girl, 15 years.	Drinking-water.	Headaches, dizziness, unsteadiness of gait. Diplopia from paresis of both third pairs. Slight but well-marked ataxia in both upper and lower extremities.	Recovery.	In the Brit. Med. Jour. for 1888, vol. i. p. 471, a case of ataxia in a brass-worker is reported, and reference is made to the same symptoms in zinc-workers.
30. Suckling, Brit. Med. Jour., 1888, vol. i. p. 647.	Boy, 14 years.	Had worked in zinc and lead for eighteen months, until within a few weeks before onset of symptoms.	Typical case of so-called "multiple neuritis," without the usual symptoms of lead poisoning.		The diagnosis of lead poisoning is not certain, but the case is interesting as pointing a line of future study. These symptoms correspond, I believe, with one type of lead poisoning.

APPENDIX B.

I.

The method used by Dr. A. M. Comey, and usually employed in the Medical Department of Harvard University, for the detection of lead in the urine, is as follows :

The urine is first evaporated to dryness, then fused in a crucible, with the addition of a little pure nitre, till it becomes white. The crucible is then cooled, and dilute HCl added, hot, to extract the residue after ignition. It is then filtered, and the filtrate treated with ammonia to alkaline reaction, to precipitate the phosphates and iron. Sulphide of ammonium is added at the same time, which throws down the sulphide of iron and lead. This is washed three times by decantation with hot water, then water is added, and the whole is acidified with HCl, and allowed to stand until the next day. It is then filtered through a small filter and the residue washed. A little pure (free from iron) nitric acid is then added, drop by drop, by which the sulphide of lead, if present, is dissolved and carried through as nitrate of lead. This is collected in a watch-glass, evaporated to dryness, and the final test made by the addition of a drop of water and a crystal of iodide of potassium. Finally, to eliminate bismuth, the iodide of lead is again dissolved in hot water, filtered, and re-precipitated in a perfectly clean test-tube with dilute sulphuric acid. The test-tube is allowed to stand twenty-four hours, and is then twirled gently between the fingers. The sulphate of lead, if present, rises in a delicate spiral.

II.

NOTE ON THE DETECTION OF LEAD IN URINE.

By Dr. C. P. Worcester,

Assistant in Chemistry, Harvard University Medical School.

Having had occasion to examine for Dr. J. J. Putnam a large number of specimens of urine for lead, I undertook some experiments to satisfy myself as to the accuracy as well as the delicacy of the usual methods of analysis. These experiments have resulted in some slight modifications which others perhaps may find useful.

Of the various methods tried, including the electrolytic process and that depending upon the solubility of the sulphate of lead in some of the ammonia salts, the one giving me much the best results was as follows :

The urine is rapidly evaporated over a free flame in a casserole with a large excess of nitric acid. After ignition, it is re-ignited with more nitric acid until the residue is perfectly white ; this is taken up with a few drops of strong hydrochloric acid and the least possible quantity of hot distilled water, and the resulting somewhat turbid solution is transferred to a small flask, and then sulphide of ammonium is added in excess, and the precipitate, which forms a thin, pasty mass, is well shaken. The object of keeping the solution of the ignited residue so concentrated is to insure the precipitation of the lead as sulphide in masses large enough to be caught by a filter, for although solutions of lead salts of a dilution of one part in forty thousand are readily darkened by the addition of sulphide of ammonium or of sulphuretted hydrogen, yet the closest filter will fail to separate the sulphide of lead even after long heating and standing. Solutions of somewhat greater concentration, however, are readily precipitated and perfectly cleared by filtering. It will be seen, therefore, that if the amount of urine taken (usually one quart) contains one milligramme of lead (a large amount to be found in that quantity of urine), all may be lost by making up the solution at this point to forty cubic centimetres.

If the precipitate at this point is very bulky, it is washed by decantation, but if not too heavy, it is at once thrown on a filter and washed once or twice with hot water. It is then treated with cold dilute hydrochloric acid of a strength of one part of the ordinary concentrated acid in fifty of water. This, of course, may be done either in the flask or on the filter, but washing on the filter with both the water and the dilute acid has given me the better results with urines containing known amounts of lead. When the readily soluble elements of the precipitate are dissolved by the dilute acid, the remaining precipitate, of which the sulphide of lead forms a part, is thrown on a filter, if not already there. If, as sometimes happens, the precipitate contains an appreciable amount of sulphate of calcium, it is washed with hot water containing a little sulphide of ammonium. Finally, a few cubic centimetres of hot dilute hydrochloric acid (equal parts of concentrated acid and water) are poured through the filter, collected in a watch-glass, and evaporated to dryness.

The residue on the watch-glass is taken up with a few drops of warm water and passed through a small filter one-half inch in diameter into a glass cell made in the following manner. A short piece of one-quarter-inch glass tubing is ground flat at one end, and this surface is warmed and touched with a mixture of solid paraffin, beeswax, and rosin, and applied to the centre of a microscope cover-glass. When cool, the cover-glass makes for this little jar a firm base which can be easily removed at pleasure. To the solution filtered from the watch-glass into this cell is added a drop of sulphuric acid. After several hours the resulting sulphate of lead will have settled on the cover-glass bottom as a thin adherent gray film. The supernatant liquid is withdrawn from the cell by means of a little roll of filter-paper, and the cover-glass is then detached. A drop of water is placed upon it and removed with filter-paper in order to wash off any traces of iron which the solution here often contains, and then a drop or two of sulphuretted hydrogen solution is added. Then when the cover-glass rests upon white paper, the least possible trace of lead can readily be recognized by the dark stain caused by the addition of the sulphuretted hydrogen. This final reaction with sulphuretted hydrogen proves much more reliable than that with the iodide of potassium, on account of the frequency with which traces of iron are found at the very end of the analysis as ordinarily conducted, having obstinately resisted complete elimination by treatment with dilute hydrochloric acid, and the iodide of iron has much the same appearance as traces of iodide of lead. The final application of sulphuretted hydrogen proves also of importance in distinguishing a very slight deposit of sulphate of lead from other matters not lead, which not infrequently are deposited on the bottom of the cell as a faint whitish film.

By the foregoing method a seventh of a milligramme of lead proves not difficult to detect in a quart of normal urine.

MYELITIS, SPINAL MENINGITIS, AND HEMORRHAGE INTO THE SPINAL CORD OR MEMBRANES.

By MARY PUTNAM JACOBI, M.D.

Though anatomically distinct, these three affections are clinically closely associated, and may therefore be suitably discussed together.

Myelitis, as here understood, does not embrace all the inflammations of the spinal cord observed in childhood, but only a certain portion of the diffused or interstitial class. Even from this are excluded multiple sclerosis, syphilitic lesions, and epidemic cerebro-spinal meningo-myelitis. These are treated in separate sections of this work, as are also all the systematic diseases of the cord:[1] anterior polio-myelitis,—by far the most frequent medullary disease in childhood; primary lateral sclerosis, and that, infinitely more frequent, which follows upon cerebral lesions; locomotor ataxia, which is almost unknown in childhood;[2] together with the combined form of posterior and lateral sclerosis known as hereditary ataxia or Friedreich's disease, and of which about sixty-five cases have, so far, been described.[3] Among the forms of meningitis, again, the inflammation of the dura mater, known as hypertrophic pachymeningitis, also receives special consideration in another part of this Cyclopædia.

Thus there remain for discussion in this article acute and chronic transverse myelitis; compression myelitis; acute diffuse myelitis; tubercular meningitis; simple meningitis, acute and chronic; primary hæmatomyelia or hæmatorrhachis; secondary hæmatomyelia or hæmatorrhachis; and, finally, hemorrhage into the cord or its membranes by extension from hemorrhage of the brain.

The foregoing, like all systems of classification, corresponds to the mental analysis by means of which certain groups of symptoms and lesions have gradually become isolated from one another; but it does not immediately serve to guide the clinician who is confronted with actual cases of disease. The problem then resolves itself into three stages: 1st, decision that disease of the spinal cord or its membranes exists; 2d, exclusion of the systematic diseases of the cord, or of the special diffuse forms which have been above mentioned (multiple sclerosis, cerebro-spinal meningitis); 3d, distinction between myelitis, meningitis, and intra-spinal hemorrhage.

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[1] Vulpian, Maladies de la Moelle épinière, vol. i.

[2] Gowers, Diseases of the Nervous System, p. 287.

[3] Ibid., p. 344.

There are, however, no hard-and-fast lines in nature, and the boundaries between the different forms of spinal-cord disease are far from ineffaceable. Sclerosis of the lateral columns, whose combination with posterior sclerosis constitutes the anatomical peculiarity of Friedreich's disease, is imminent in every case of chronic transverse myelitis. It is then attended by all the spastic symptoms which characterize primary or cerebral descending sclerosis. The same transverse myelitis, and especially that form of it which is due to compression, also determines an ascending or systematic degeneration of the columns of Goll, which occasionally extends into the root zone and causes ataxia. It has been recently shown that the morbid process in acute infantile paralysis is far more diffused than was at one time supposed. The lesion is not precisely limited to the anterior cornua nor to the ganglionic cells in them, nor to the segment of the cord corresponding to the muscles finally paralyzed. But there is an interstitial inflammation of the cornua accompanying and perhaps determining the conspicuous lesion of the cells, and this diffuses to a greater or less extent throughout the cord, and may even invade the posterior horns and root zones.[1] This most typical systematic disease, therefore, grades by imperceptible degrees into the acute ascending diffuse myelitis, whose clinical termination is usually so different. Again, the syringo-myelia, which hitherto has been usually considered as a lesion apart, and even as the result of a disintegrating glioma,[2] has been described by Joffroy as a periependymatous myelitis, for which he proposes the special name of "myélite cavitaire."[3] The group of diseases which we are here considering is roughly distinguished from all the others mentioned, except cerebro-spinal fever, by one important practical circumstance,—they are all immediately dangerous to life. The danger in the systematic diseases of the cord is, on the other hand, quite remote. When, therefore, a child is seized with fever, convulsions, and paralysis, and brain-disease has been excluded, it is of the greatest interest to determine whether the spinal-cord disease is about to prove an anterior poliomyelitis, with its favorable prognosis, or a common myelitis or meningitis, with its much more serious outlook.

COMPRESSION MENINGO-MYELITIS.

The general picture of non-systematic or diffuse disease of the spinal cord in children may be obtained from that form of it which is in them the most frequent,—namely, the compression meningo-myelitis caused by caries of the spine, or Pott's disease.[1]

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[1] Eisenlohr, Deutsches Arch. für Klin. Med., Bd. xxvi. S. 543; Drummond, Brain, vol. viii. p. 18; Angel Money, Transactions of the Pathological Society, 1884, p. 45; Förster, Jahrbuch für Kinderheilkunde. Bd. xv. Heft 3 u. 4.

[2] Allen Starr, American Journal of the Medical Sciences, May, 1888.

[3] La France Médicale, October, 1887.

In the first case we shall quote, symptoms of myelitis preceded the appearance of pathognomonic symptoms of spine disease:

Case I.—Girl eight and a half years old. At the age of three she was attacked by a pain in her throat, and then rather suddenly lost the use of her legs. She lay in bed for several months, then entirely recovered, but with a slight prominence of the spine in the upper dorsal region. Remained well until seven and a half years old; when she began to suffer from pains in the back and abdomen, and the curvature of the spine increased. Six months later, sudden loss of power in both legs, and in the right hand, then, after a few days, in the left hand also. The paralyzed muscles contracted slightly to the faradic current, and those of the arms showed an increased excitability to the galvanic current. Sensation intact. The right pupil was larger. Profuse sweating; occasional incontinence of urine and fæces. Treated by absolute rest in bed, tonics, and cod-liver oil. The condition remained unchanged for two months; then some motor power returned to legs and left hand. In four months control of the sphincters was regained. In five months she could move right arm, then walk, and by six months could run about the ward.[2]

Case II.—Paraplegia in a girl five and a half years old, with dorsal spondylitis and scoliosis, cured in twelve weeks, the deformity remaining, by extension of the spinal column and daily galvanism.[3]

Case III.—Girl of fifteen. Four months before admission, pain between shoulders, then motor power of legs diminished, until there was complete paralysis. On admission, paraplegia persisted, with loss of tactile sensations in legs, and analgesia as high as the third dorsal vertebra. Legs were wasted, flexed at knees, and drawn up,—marked rigidity. Patellar reflexes were exaggerated, ankle-clonus existed. The breathing was entirely diaphragmatic. There were bed-sores, and paralysis of the bladder, with incontinence. Angular curvature in upper dorsal region, and great pain on moving head. After admission, rigidity of legs at first increased; but in six months there was return of sensation, in nine months ability to move toes, rigidity disappeared, and bladder was controlled. In sixteen months patient could walk by the aid of a chair.[4]

Case IV.—Boy of six. An angular curvature of spine began at four or five years, after a fall. Power over legs and bladder impaired for three weeks. Counter-irritation applied to neck; in three weeks boy could stand. Then attack of scarlatina, and paraplegia returned. Recovery at end of nine months; fresh relapse after pneumonia; final recovery.[5]

Case V.—Child four years old. Admitted to St. George's Hospital with well-marked symptoms of caries of cervical vertebræ. Respiration unimpaired. Soon hydrocephaloid symptoms set in, and child died under symptoms of increased brain-pressure. Ventricles found much distended with fluid. Transverse ligament of second cervical vertebra had given way, and odontoid process projected into spinal canal. Dura mater entire. Pressure on cord had not impaired its functions, but had apparently occasioned an irritation resulting in the ventricular effusion.[6]

Case VI.—Patient aged sixteen, with cervical caries, died in a similar manner.[6]

Case VII.—Girl aged twelve. Caries of spine. Under treatment by tonics and the leather corset. During course of treatment had an attack of pain in the scalp and back, which extended to ribs, accompanied by spasms in the legs. After a week, the pain was relieved, but there remained stiffness of the legs and entire trunk. Numbness occurred, and was followed by loss of sensation, and complete paraplegia, though with intact sphincters. The patient recovered in about six months.[1]

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[1] Wharton Sinkler, out of one hundred and forty cases of paralysis among children observed during a period of four years, had five cases of spinal meningitis and three of myelitis. Two of the former and all of the latter depended on caries of the spine. American Journal of the Medical Sciences, April, 1875.

[2] Gowers, Medical Times and Gazette, November, 1876, p. 517.

[3] Soltmann, Neurologisches Centralblatt, 1882.

[4] Glynn, British Medical Journal, September 22, 1883.

[5] Faure, Lancet, April 6, 1861.

[6] Brodie, Injuries to the Spinal Cord, Medico-Chirurgical Transactions, 1837

Case VIII.—Child two years old, with caries and sclerosis, without angular curvature, of third, fourth, and fifth dorsal vertebræ. Paralysis and atrophy of both lower extremities. Death. At autopsy tumor found, consisting of a sac filled with cheesy contents and pus, extending from fourth dorsal to first lumbar vertebra. The dura mater distended with flocculent serum, itself thickened and hyperæmic; the arachnoid adherent to the pia, thickened, leathery, and containing osseous plates. Cord quite normal.[2]

The liability to compression myelitis varies with the seat of the caries. In the lumbar region, according to some statistics, it is rare: Bouvier found only five paraplegias out of thirty-eight cases of lumbar caries;[3] Coudroy de Lauréal, only one paraplegia out of fifty-nine similar cases.[4] Among fifty-six cases of dorsal caries, on the contrary, Bouvièr observed thirty-eight cases of paraplegia, and Coudroy nineteen cases out of seventy-seven in the same locality. The special predominance of medullary accidents with dorsal caries is attributed to the narrowness of the spinal canal in the dorsal region, which renders the cord more exposed to compression, and less able to slip out of reach. The upper dorsal region of the spine is the most liable to aggravated forms of carious disease, the most liable to severe deformity, and the most liable to abscess with accumulations of pus that fail to become liberated from the spinal canal. It is, finally, the most difficult to treat by immobilizing apparatus. For all these reasons, it is easy to understand why paraplegia should occur more frequently with Pott's disease in the upper dorsal region of the spine than with caries of any other locality.

Compression may, by exception, be suddenly produced, if the body of a carious vertebra break down suddenly, so that fragments of bone are dislocated backward and impinge upon the cord. The accident, though occurring in the course of a chronic disease, then, in its effect on the functions of the cord, precisely resembles traumatic fracture. In the cervical region, sudden death may result if the odontoid ligament, softened by tuberculous infiltration, give way, and allow the odontoid process to be driven against the cord,—so sensitive in this region, from its proximity to the respiratory centres and to the origin of the phrenic nerve. In the dorsal and lumbar regions a similar impact of bone may be followed by paraplegia so sudden and so complete as to resemble the effect of intraspinal hemorrhage,—and, indeed, to suggest this, unless the diagnosis has been plainly indicated by the previous existence of an angular curvature or by the sudden development of one coincidently with the paraplegia.

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[1] Elliott, Dublin Monthly Journal, 1886, vol. lxi. p. 7.

[2] Kahler, Meningitis Spinalis, Leipsic, 1861, p. 80.

[3] Gazette des Hôpitaux, 1858, p. 286.

[4] Quelques Considérations sur le Mal vertébral chez l'Enfant, Coudroy de Lauréal, Thèse de Paris, 1874.

In the great majority of cases, however, even when the lumen of the canal has been suddenly diminished by the collapse of a vertebra, the cord slips behind the projecting bone, and thus escapes compression from bone.[1]

Compression of the cord in caries of the spine is due to another agent, as was first demonstrated conclusively by Michaud.[2] Irritation of the spinal dura mater, from contact with the caseous contents of the bone-abscess or with minute spiculæ of carious bone, gradually develops an external pachymeningitis. Abundant inflammatory vegetations form a thick mass around the cord, which tends to compress it more and more. The mass frequently contains tubercle. Not infrequently there is also an internal pachymeningitis, and a grayish pseudo-membrane, a vascular net-work with embryonic connective tissue, spreads over the internal surface of the dura.[3]

Submitted to the pressure of these inflammatory products, the cord becomes altered, chiefly through an excessive proliferation of its neuroglia. A peculiar interstitial myelitis is developed, complicating the meningitis which is the primary lesion.[4]

The proliferating connective tissue at first contains cells, but later becomes converted entirely into a dense reticulum, which greatly increases the consistency of the cord. The nerve-elements degenerate, the myeline wastes, although for a while many nerve-fibres persist with a narrowed myeline sheath. In extreme cases all the fibres seem to be destroyed; the ganglion-cells of the gray substance become shrunken and atrophied, and finally disappear; and at the point of greatest intensity of the lesion, all distinction between white and gray substance is obliterated. The walls of the blood-vessels are thickened by a growth of spindle-cells in them.[5] Beyond the principal focus of myelitis lies an ascending sclerosis in the columns of Goll, and a descending sclerosis in the lateral columns of the cord.[6] Occasionally the medullary lesions are preceded by neuritis of the nerve-roots which have been compressed in the thickened dura mater. The first lesions in the cord may then appear as streaks of sclerosis in the posterior columns. It is thence distributed with the irregularity which is the anatomical characteristic of all cases of compression myelitis. (Michaud.) The cord may be greatly reduced in volume, being not larger than a goosequill, and yet preserving many of its functions, while all are susceptible of restoration.

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[1] Vulpian, Maladies de la Moelle épinière; Follin, Traité de Path. externe; Grasset, Traité des Maladies nerveuses; Puel, Thèse de Paris, 1878, Du Mal vertébral. This escape of the cord from compression in the roomy spinal canal was pointed out by Cruveilhier.

[2] Thèse de Paris, 1871. This thesis was written under the inspiration of Charcot, who himself has written on the same subject,—Gazette des Hôpitaux, 1874. These essays constitute an epoch in the history of Pott's disease.

[3] Vulpian, loc. cit.; Puel, loc. cit.

[4] Gowers, Diseases of the Nervous System, 1888, p. 249.

[5] Charcot, Archives de Physiologie, 1868, p. 735.

[6] Bouchard's researches on the secondary degenerations of the cord were made chiefly upon cords affected with compression myelitis.

The irritative process sustained in the spinal meninges extends to the nerve-roots as well as to the cord, exciting an interstitial neuritis. This, indeed, usually precedes the myelitis, and is indicated by a characteristic group of symptoms, which Gowers especially emphasizes as "root symptoms." These consist chiefly in extremely acute pains, which radiate along the nerves of the limbs or trunk, from a level corresponding to the seat of a manifest or latent vertebral caries. The pains are increased by movement, and accompanied by tenderness on pressure along the track of the nerves, followed by anæsthesia distributed in irregular patches along the same track, and, in severe cases, by atrophy of the muscles to which the nerves are distributed.

When irritation and inflammation of the nerve-roots are thus indicated, the existence of a pachymeningitis is at the same time demonstrated, since the neuritis is always secondary to this. Myelitis is then known to be imminent or already commencing, and the occurrence of paraplegia is to be feared.

The myelitis is ushered in by a second group of symptoms, whose sequence upon the root symptoms is highly characteristic of compression disease. These symptoms are chiefly motor. The limbs—in the great majority of cases the lower extremities—become paretic; the paresis deepens to complete paralysis, either gradually, or with great rapidity or even suddenness. The muscles of the paralyzed limbs may be affected with spasms.

The paresis and paralysis are due to invasion by the myelitic process either of the anterior cornua or of the lateral columns. The sensibility of the paralyzed limbs often remains unimpaired. On the other hand, it is often perverted in various ways. Sometimes the conduction of sensory impressions is delayed, possibly as long as for thirty or forty seconds, or the patient suffers from numbness, formication, tingling, or dull, obtuse pains, which are in marked contrast with the violent pains excited by irritative compression of the nerve-roots.

The frequent preservation of tactile sensibility in the midst of complete motor paralysis depends on two circumstances. First, it is the anterior or motor portion of the cord, lying in immediate proximity to the diseased vertebræ, which is primarily and predominantly exposed to the encroachment of the pachymeningitis excited by the caries. The posterior, sensory portion of the cord, more remote from the focus of irritation, remains for a long time, and possibly always, unaffected. In the second place, conduction of sensory impressions will persist after a much more extensive destruction of nerve-fibres than would be compatible with persistence of motor functions. Hence sensibility may often remain intact, even when the sensory segment of the cord has been invaded by the myelitic process. For the same reason, even when sensibility has been lost, it may be recovered in a patient in whom absolute paraplegia, nevertheless, persists.

The condition of the sphincters and of the reflexes of the lower extremities depends both on the situation and on the extent of the spinal disease. The sphincters will remain intact, the reflexes normal or but slightly exaggerated, whatever the locality of the lesion, provided this consist only in meningitis or is limited to the cortical zone of the cord.[1] When the lesion is situated anywhere above the lumbar enlargement, the portions of the cord below the lesion are affected by the secondary descending sclerosis of the pyramidal tracts. The reflexes of the lower extremities are then exaggerated, there is ankle-clonus, and the tone of the sphincters is increased. On this account, and because the paths of voluntary impulse in the pyramidal tracts are simultaneously impeded, there is apt to be retention of urine and fæces.

If the lumbar enlargement itself happen to be the seat of the myelitis, then the reflexes of the lower extremities, far from being exaggerated, will be abolished,[2] and the sphincters will be paralyzed, from destruction of their spinal nerve-centres. This, however, happens only if the central gray substance has become completely involved.

Several other important variations of symptoms are observed, according to the situation of the spinal caries, and of its consecutive meningo-myelitis. Thus, in disease of the three upper cervical vertebræ there is cervical paraplegia, which may either follow or precede the paraplegia of the lower extremities, which is itself produced by lesion of any part of the cord. This sequence is readily explicable; but much less so is the more exceptional occurrence of paralysis of the arms while as yet the legs are unaffected. This remarkable fact has been explained on the hypothesis that the motor tracts for the arms may lie more superficially in the cord than those for the legs, and are thus earlier involved in a lesion advancing from the periphery. (Brown-Séquard.) Vulpian, however, claims that the anatomical reverse is the truth: that in the cervical region the nerve-fibres for the arms have already entered the gray matter of the cord, while those for the legs are yet confined to the lateral columns. But he considers that on this very account should the arm fibres be submitted to a greater compression, owing to the less resistance of the gray substance than of the white columns to compressing agencies.

Disease in the upper cervical region is distinguished from lesion in the lower by absence of the muscular atrophy of the paralyzed upper extremities which is so characteristic a feature in disease at the latter locality.[3] Muscular atrophy occurs only when the trophic centres of the supplying nerves have been affected,—thus, with compression myelitis of the cervical enlargement,—either from idiopathic pachymeningitis, or from that which is secondary to vertical caries. The characteristic "claw hand" then results. The absence of the claw hand, when other symptoms point to cervical myelitis, suffices therefore to limit the disease to the region of the three upper vertebræ.

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[1] The entire cord may be encircled by a narrow zone of interstitial myelitis, and the disease is then called cortical myelitis.

[2] Reflexes are abolished by lesions of the posterior roots of the postero-external columns (columns of Burdach) of the central gray matter, anterior root-fibres, or anterior roots. "It is probable that the superficial reflexes pass directly into the posterior horns, the deep reflexes through the postero-external column." (Bramwell.)

[3] Kahler, Prager Med. Wochenschrift, 1883, p. 458.

It is for the upper cervical region that there is the special danger already mentioned of sudden death in case the odontoid ligament rupture, and the odontoid process be driven abruptly into the spinal cord.

Vomiting is a frequent symptom in disease of this region. It may be due to an ascending myelitis which has reached the nuclei of the pneumogastric nerves, or it may be caused by direct irritation of the fibres of the spinal accessory branch.

From the same cause may be observed slowing of the pulse, or even syncope. Irritation of the phrenic nerve,[1] direct or by a descending myelitis, will cause hiccough, or sometimes a peculiar form of dyspnœa, due to tetaniform spasms of the diaphragm. If the nuclei of origin of the nerve be destroyed, diaphragmatic breathing will be arrested, and the movements of the thorax will be carried on exclusively by the intercostal muscles. The dyspnœa then becomes still more severe.[2]

Caries of the lower cervical region tends to excite a pachymeningitis similar in form to the pachymeningitis hypertrophica of adults.[3] The roots of the cervical nerves become compressed in the meningeal exudation; radiating pains in the arms occur, followed by muscular atrophy and paralysis. The arm reflexes are at first exaggerated, and percussion of the lower end of the radius or ulna causes reflex contraction of different muscles of the forearm or upper arm. In Kahler's five cases (all, however, in adults) there was neither abolition of the reflexes nor loss of electrical contractility in the paralyzed muscles. These phenomena are, however, often observed in the advanced stages of the disease.

As in the idiopathic form of cervical pachymeningitis, compression limited to one or more separate roots of the cervical plexus will result in atrophic paralysis localized at different segments of the arms. Analysis of these localized paralyses may therefore sometimes lead to more precise localization of the lesion in the cord, since, by the experiments of Ferrier, the functions of the different nerve-roots have been approximately determined.[4]

Thus, the intrinsic muscles of the hand are innervated from the first dorsal nerve, and weakness and atrophy beginning in them, associated with localized tenderness on pressure over the first dorsal vertebra, should lead to probable diagnosis of incipient caries with pachymeningitis. The presence of pain in the affected muscles is an important distinction from progressive muscular atrophy, which in this form, indeed, scarcely ever attacks children. The innervation of the remaining muscles of the limb is distributed between the five lower cervical nerves. Flexion of the wrist and fingers is effected by means of the eighth, seventh, and sixth roots; extension of the wrist and fingers by the fifth and fourth. The same roots (the three lower) which flex the wrist extend the forearm, and those (the upper) which extend the wrist flex the forearm. The eighth cervical root has no influence over the upper arm, which is adducted by the seventh and sixth roots, and raised by the fifth.

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[1] Arising from the fourth and fifth cervical region, hence at the limits between the upper and lower cervical regions.

[2] Michel, quoted by Treves, International Encyclopædia of Surgery, vol. iv. p. 941.

[3] Kahler, loc. cit., 1883, p. 58. See Joffroy, Archives de Physiologie, 1873. Kahler quotes from Proust an adult case where there was excessive atrophy of the muscles of the ball of the thumb and the interossei, without paralysis of the arms.

[4] Localization of Atrophic Paralyses, Brain, vol. iv. p. 223.

A diffuse process, like the pachymeningitis of cervical caries, would usually extend over several of these roots, adjacent to one another; so that, for instance, the intrinsic movements of the fingers, flexion of the wrist, and extension of the forearm, might all be lost, and thus a lesion in the lower part of the lower cervical region indicated; or the localization could be made for the fourth and fifth roots,—thus the upper part of the lower cervical region. Such localizations would be chiefly important if tenderness on pressure and stiffness of movement were found over a vertebra corresponding to the nerve-root implicated. In the cervical disease tactile sensibility remains unimpaired, and so long as the effects of the pachymeningitis are confined to the nerve-roots the lower extremities are not affected. Paraplegia proper sets in, if at all, as the symptoms of root irritation subside and the cord itself becomes compressed.

Characteristic of the meningo-myelitis of this region are the pupillary symptoms from irritation of the oculo-pupillary centre. With irritation of the centre the pupils, one or both, are dilated; with paralysis they are contracted. These symptoms are by no means always present.

In the meningo-myelitis of the upper dorsal caries, which, as already stated, is by far the most frequent,[1] the characteristic paralysis occurs in the lower extremities.

But disease in the upper dorsal region may also be followed by paralysis of the arms,[2] due to an ascending degeneration that has extended beyond the limits of the columns of Goll and invaded the antero-lateral columns.[3] The cervical paraplegia is then always consecutive to the lumbar paralysis.

Several other symptoms, that are often enumerated as characteristic of dorsal caries, really belong to the meningo-myelitis which has been produced by it. Among the earliest are pains radiating towards the abdomen, and often mistaken for pains in the stomach and bowels. These precede paraplegia. A characteristic advanced symptom, seen only after paraplegia has become confirmed, is paralysis of the intercostal muscles, so that the breathing is sustained exclusively by the diaphragm.[1]

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[1] *Ut supra*, p. 646.

[2] *Ut supra*, Case I.

[3] Erb, Ziemssen's Cyclopædia; also Gowers, loc. cit., p. 225. Leyden says he has never seen a compression myelitis progress upward. But positive observations of such ascension are recorded by Vulpian and by others.

Again, characteristic of dorsal myelitis are the maintenance of the reflexes, of electrical reactions, and of rigidity in the paralyzed limbs, and the absence of sphincter paralysis and trophical lesions, such as muscular wasting and bed-sores. These peculiarities, both positive and negative, depend on the fact that the transverse cord lesion is seated above the lumbar enlargement; the latter is affected only by the descending lateral sclerosis, thus not in its central gray substance at all. Disease of the lumbar enlargement, therefore, contrasts with that due to dorsal caries, in all the above particulars. The limbs soon become flaccid, the reflexes abolished, electrical reactions lost. If the gray substance be invaded, the sphincters are paralyzed, the muscles waste, and even bed-sores appear, although these are rare in compression myelitis.

If the caries affect the spine below the second lumbar vertebra, it will be below the termination of the cord. When nervous symptoms develop, they are those of neuritis, due to inflammation of the nerves of the cauda equina.[2]

Muscular atrophy is then an early symptom ; it is partial and irregularly distributed, the muscles usually being flaccid or free from contracture. The limbs are paretic, but are rarely paralyzed completely. The reflexes are exaggerated, the sensibility normal.

Attention to the above combination of symptoms will serve to distinguish a neuritis of the cauda equina from a meningo-myelitis, and conversely; and will aid in detecting an incipient caries of the lumbo-sacral spine before any angular curvature has become perceptible.

Although the combination of root neuritis and interstitial myelitis be the most common result of pachymeningitis from caries, and hence the association or sequence of root and cord symptoms the most characteristic, yet the nerve-roots are not necessarily involved. The cord symptoms may appear primarily, and usually develop in gradations as above described. Sometimes, however, a child known to be affected with spinal caries will become paraplegic in the course of a few minutes. Sometimes an angular curvature appears coincidently and for the first time, or, when already existing, becomes greatly increased. The paraplegia is then attributable to the shock caused by the impact of the fractured bone upon the cord, and may pass away with the shock of the accident, even though it return later after a meningo-myelitis has been lighted up. When, however, a suddenly-developed paraplegia is both complete and permanent, it is probable that a hemorrhage has occurred among the neo-membranes of a pachymeningitis, or even into the cord itself. The results of the former accident may be transitory, as the effused blood is reabsorbed; but the consequences of any extensive hemorrhage into the cord are irremediable. Thus, caries of the spine may determine in the same patient all three of the cord lesions under consideration in this article,—meningitis, myelitis, and spinal hemorrhage.

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[1] Sayre claims to determine diaphragmatic breathing artificially by means of the plaster jacket, and ascribes to this part of the beneficial influence of his method.

[2] Mme. Conta, Du Mal de Pott au-desssous de la Moelle chez les Enfants, Thèse de Paris, 1887.

It is necessary to make a diagnosis between the three lesions whenever motor and sensory symptoms in the limbs complicate the evident manifestations of Pott's disease. It is also often necessary to decide, in presence of such symptoms, whether or not Pott's disease be present as their ultimate cause, although its distinguishing features are not yet manifest, but latent.

The special symptoms of meningitis are pain in the back, spontaneous and exaggerated by pressure, and still more by movement of the spinal column; pains radiating to the trunk and extremities, with diffused hyperæsthesia of the skin and muscles; stiffness and rigidity of the spinal column and of the limbs, the latter often intermittent; exaggerated reflexes, and muscular spasms. The spinal pia mater is richly furnished with nerves, which enter from the posterior roots and follow the blood-vessels.[1] Inflammatory irritation of this membrane causes therefore severe localized pain. The muscular spasms depend on reflex irritation of the motor roots, from the irritation of the sensitive fibres of the pia. It is the tetanic spasm of the muscles of the back that causes its rigidity. The generalized hyperæsthesia and exaggerated reflexes are due to erethism of the intra-medullary foci of origin of the posterior spinal roots. (Vulpian.)

Among these symptoms, localized pain and tenderness in the back also indicate caries uncomplicated by meningitis. But in such a case the pain is much less severe, and the tenderness, instead of extending all along the spine, is limited to the small area of the one or two diseased vertebræ. Finally, there may be entire absence of pain over a carious vertebra. Rigidity of the spine exists both in caries and in meningitis; but in the latter it is much more marked, tending towards opisthotonus; the patient is unable to sit up. In uncomplicated caries the patient, though holding the back stiffly, is not prevented from walking. By a special manœuvre the real rigidity of the spine is shown to be confined to a limited area. The child lying prone upon a couch, the physician with the left hand fixes the spinal column, while with the other he raises the legs and bends them backward at the pelvis. Normally the spine of a child is so flexible as to permit movement during this manœuvre, at the lumbar, lower dorsal, or upper dorsal region; but, if any vertebra be diseased, the movement is resisted in the corresponding region. But if meningitis be present, the foregoing test is too painful for execution. The rachialgic symptoms of meningitis consequent upon caries may be said to be the symptoms of the caries itself, intensified and extended.

Rigidity of the limbs is a symptom which in meningitis may be caused in three ways: by direct irritation of the motor roots, by reflex spasm of irritation of the posterior roots, and by increased excitation of the gray matter of the cord. The symptom, however, does not belong to meningitis alone, but occurs in the course of the myelitis which in children usually, and in spinal caries always, complicates the meningitis. The rigidity is then accompanied by increased reflexes, and both are due to the sclerosis of the pyramidal tracts which sets in below the seats of the principal lesion.

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[1] Kölliker, Handbuch der Gewebelehre, 1863, p. 340.

Such sclerosis cannot be ascribed to a meningo-myelitis until cerebral disease has been excluded, as the degeneration of the pyramidal tracts is much more frequently secondary to lesion of the brain than of the cord.

It is, finally, to be remembered that exaggeration of the reflexes and some degree of rigidity of the limbs may be due simply to hysteria. Such a manifestation of hysteria is, however, particularly rare in children.

The muscular rigidities and exaggerated reflexes of myelitis may be distinguished from those of meningitis by their late occurrence and consecutive paresis or paralysis. For in myelitis these symptoms are due to the secondary degeneration ; in meningitis, to the primary disease, and are among its initial phenomena. Meningitis alone does not cause paralysis, and the appearance of this symptom among those proper to irritation of the meninges marks the moment of invasion of the cord. Although compression myelitis is always immediately caused by an inflammatory exudation over the dura mater, it not infrequently happens that the meningeal process remains latent, and that the first complication of the Pott's disease which is observed is loss of motor power in the lower limbs, indicating disease of the cord itself. In the absence of root symptoms the paresis or paraplegia may be unattended by any perversion of sensation whatever. Indeed, not only the meningitis but the vertebral caries may have remained latent and the entire illness apparently begin in a loss of power to stand, while all other functions remained normal. Careful examination will then detect localized stiffness at some point of the spine, and the caries be discovered which is the root of the whole mischief.

It is characteristic of compression myelitis that the paralyzed limbs are at first stiff, and continue so as long as the lesion remains limited to the white columns of the cord. As the disease progresses towards the gray substance, and the ganglionic cells of the anterior cornua are destroyed, the stiffness yields to the flaccidity proper to anterior polio-myelitis. Loss of sensibility similarly indicates invasion of the gray substance in its posterior segment. Both the flaccidity and the anæsthesia decisively distinguish myelitis from meningitis, and also indicate a severe degree of the medullary disease. In the typical cases of compression myelitis the symptoms due to disease of the meninges, the nerve-roots, and the cord are always combined in the sequence which has been described. Primary affection of the cord does not exist ; but its symptoms may appear first, while those of the pachymeningitis remain latent.

Hemorrhage into the membranes or substance of the cord, when complicating compression meningo-myelitis, is distinguished by the sudden occurrence of motor and sensory paralysis, or by the suddenly-marked exacerbation of paralytic symptoms which had previously existed. The hemorrhage may be capillary, and result in such rapid disintegration of the cord as may appear, on examination, due to an acute myelitis. Hemorrhage much more frequently occurs into the neo-membranes of the spinal dura, the process being completely analogous to that of cerebral hemorrhagic pachymeningitis observed in the adult.

On the whole, hemorrhagic accidents seem to be rare in compression meningo-myelitis.[1]

In childhood vertebral caries is so much the most frequent cause of compression of the cord that the diagnosis of a compression meningo-myelitis carries with it that of Pott's disease, in the immense majority of cases. This diagnosis may, however, be erroneous, as is shown by a case of Dr. Gee's:[2]

Case IX.—A boy four and a half years old was admitted to hospital in February, after six weeks of undefined languor and malaise. At this date the right arm was paralyzed, the right upper eyelid drooped, the right pupil was rather smaller than the left. The muscles of the paralyzed arm no longer responded to electricity. There was no loss of cutaneous sensibility. The superficial reflexes were well marked, but the patellar reflex could not be obtained. In March the legs became paraplegic and flaccid. In April there was moderate fever, and shortly after the breathing became wholly diaphragmatic. Loss of faradic contractility in all the muscles of the legs, except the gastrocnemii and hamstring muscles. In May the cutaneous sensibility diminished; there was incontinence of fæces. An elastic swelling appeared at the right side of the upper dorsal and lower cervical vertebræ. It was noted that the child perspired excessively when asleep. A vertebral caries was suspected, with an abscess involving the inferior sympathetic ganglion, and accompanied by a subacute descending polio-myelitis.

The child died in June. At the autopsy was found a sarcomatous tumor springing from the lower cervical and first three dorsal vertebræ, and connected with the three upper ribs. The spinal cord was reduced in size opposite the last cervical and first dorsal vertebræ, and softened throughout. The gray substance was pale and ill defined. In the cervical enlargement the large motor cells were found swollen and often deprived of their nuclei and processes.

Thus the tumor, beginning outside the spinal canal, had spread in two directions,—towards the right inferior cervical ganglion, which had apparently been destroyed, and internally towards the cord, which had become affected with a subacute myelitis, chiefly central. This central softening contrasts markedly with the peripheric induration which is the characteristic result of vertebral caries, which proceeds gradually inward, and which is invariably attended by descending sclerosis of the lateral columns.[3] In commenting on the results of the autopsy, Dr. Gee remarks that the flaccidity of the legs observed during life, and contrasting with the rigidity which would have been caused by a lateral sclerosis, should have been interpreted as indicating some other cause of compression of the cord than caries. Besides this important negative circumstance, the positive symptoms of a lesion of the inferior cervical ganglion, partial ptosis of the right eye,[1] and contraction of its pupil, should also contradict the diagnosis of Pott's disease, and indicate some cause of compression lying entirely outside of the spinal canal. This could hardly be anything else than a tumor.

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[1] Gowers says that hemorrhage has not been proved. Yet he himself cites a case where a child, affected with an angular curvature, fell in walking across the room, and on being lifted up was found paraplegic. Recovery ultimately took place.

[2] Myelitis due to vertebral sarcoma in a boy four and a half years old, Gee and Abercrombie, St. Bartholomew's Hospital Reports, 1882, vol. xviii.

[3] Michaud, loc. cit.

The prognosis of compression myelitis is not altogether unfavorable. Among the twenty cases detailed by Coudroy, recovery occurred spontaneously in eleven, and twice the recovery dates from the appearance of a cold abscess,—that is, from the external migration of pus. The cases of Gowers, Soltmann, Glynn, and Faure, already quoted, resulted in recovery.[2] Althaus,[3] Charcot,[4] and Ricord[5] attribute recovery to treatment by the actual cautery. But from the well-established fact that cure takes place spontaneously in more than half the cases, it is difficult to decide how far this treatment really influences the curative process. The prognosis remains favorable so long as the morbid process remains limited to the surface and anterior region of the cord. This limitation is to be inferred so long as the paraplegic limbs remain rigid instead of becoming flaccid, the reflexes exaggerated instead of being diminished or abolished, the sensation intact, the sphincters unparalyzed, and no bed-sores threaten. Still, even flaccid paralysis is often recovered from.[6]

Restoration of motor power depends on four circumstances:

1. An accumulation of caseous pus may find an external outlet, forming a cold abscess in the back or groin. The dura mater is then relieved of the irritation which had excited and kept up the pachymeningitis; the meningeal inflammation subsides, and the progress of the secondary interstitial myelitis is arrested.

2. Nerve-tubes in the sclerosed patches may regenerate, especially those which have not lost all their myeline sheaths.[7] Atrophied ganglionic cells in the anterior cornua apparently never regenerate, but, as their destruction is only partial, enough remain intact to maintain the function of the limb.

3. In the absence of an accumulation of pus, the pachymeningitis may spontaneously subside, coincidently with amelioration of the osseous inflammation. This latter may be arrested either before the formation of a spinal curvature or after the vertebra has broken down, and carious material has been eliminated from the bone-tissue.

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[1] Attributed to paralysis of unstriped muscular fibres in the upper eyelid.

[2] See also Leudet, "Sur la Curabilité des Accidents paralytiques consécutives au Mal vertébral de Pott," Soc. de Biol., 1862; Kahler, Rückenmarks-Compression bei tuberculöser Caries der unteren Halswirbel, Prager Med. Wochenschr., 1883; Grasset, Maladies du Système Nerveux, 1886, p. 492; Billroth, Allg. Wien. Med. Zeit., 1884, p. 563.

[3] British Medical Journal, May, 1886.

[4] Loc. cit.

[5] Thèse de Paris.

[6] Leyden in 1874 pronounced a very unfavorable prognosis.

[7] Bramwell, Diseases of the Spinal Cord, 1882, p. 166.

4. Fixation and extension apparatus applied to the spine tends to facilitate the cure of paraplegia by limiting the peritubercular irritation of bone-tissue, by relieving the diseased parts of superincumbent weight, and by raising portions of the collapsed vertebræ which may be pressing against the cord and irritating the dura. The latter effect of the extension apparatus, including the plastic jacket, is probably the least important, and yet it is not to be disregarded or denied. The influence is undeniable where paraplegia has occurred suddenly and at the same moment an angular curvature has appeared for the first time on the spine. A paraplegia of two or three weeks' duration has been seen to disappear immediately when the child had been suspended in Sayre's apparatus and encased in the plaster jacket. The degree to which the spinal vertebræ can be extended by hanging is slight, but this slight extension may suffice to remove the bone from contact with the dura. The influence of the extension and fixation apparatus in preventing paraplegia is even more marked. It would be most useful to establish a comparative statistic in regard to the occurrence of paraplegia in the cases treated with and in those treated without fixation apparatus from the beginning. But I am not aware that any extensive comparison in regard to this point has as yet been made.

OTHER FORMS OF MENINGITIS.

Vertebral caries is the most frequent cause of spinal pachymeningitis in childhood, and of the myelitis secondary to it. But several other conditions are liable to induce inflammation of the spinal membranes. Tubercle, traumatisms, insolation, exposure to cold and fatigue, may all occasionally be followed by meningitis. Kahler asserts that childhood constitutes a predisposition to the disease.[1] The following is a fairly typical case of traumatic meningitis :

Case X.—Boy four years old, kicked in the loins. Feverish for the next few days ; on the fourth day, restless and moaning, indisposed to move his limbs. On the fifth day the breathing became labored ; there was great tenderness over the dorsal region. In the afternoon of the same day the arms became powerless, the breathing shorter, and the lips twitched. Death in convulsions on the sixth day. At the post-mortem "strong evidence of spinal meningitis continuing downwards at the first lumbar vertebra."[2]

This *naïve* and incomplete history fails to explain the paralysis of the arms, and does not mention whether paralysis of the legs existed. It is evident, however, that an ascending central myelitis had rapidly complicated the meningitis primarily developed at the seat of the blow.

Case XI.—Girl of twelve. Three months before admission to hospital, while jumping, fell on lower part of the spine, immediately upon sacrum. There was sharp pain at once, and in two days the girl could not sit up without pain. On admission, the coccyx was tender, the whole spine curved, while distinct fulness could be detected over the sacral region. Pain extended down both legs; there were sensations in them of pins and needles, and a constant tendency to draw them up. The sensibility of the right leg was diminished. There seems to have been no paralysis. The patient was treated by rest in bed, small blisters over the back, and minute doses of medicine,—two grains of iodide of potassium and five drops of tincture of belladonna three times a day. After six months of such treatment recovery was complete.[1]

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[1] Meningitis Spinalis, H. Kahler, Leipsic, 1861,—a most erudite monograph. The writer claims (p. 2) that the first autopsies on the spinal cord were made in the fifteenth century.

[2] Robinson, Lancet, March 1, 1862.

Case XII.—Boy of twelve. Subject to epileptic attacks for two years. Illness began in one of these. Admitted to hospital with temperature of 39° C. Pulse 110. Severe pains in head and back, especially in lumbar region, increased by movement or pressure on sacro-lumbar muscles, when opisthotonus was produced. The pains radiated to trunk and limbs, and were so severe in the neck that the patient could not turn his head. Diffuse hyperæsthesia over entire body. Reflexes and electrical reactions persist. Sphincters intact. No paralysis. Cerebro-spinal meningitis diagnosed, due to congestion of epileptic attack. Treated by leeches, bromide of potassium, salicylate of sodium for fever, calomel for constipation. Recovery.[2]

Case XIII.—Boy of five years. Struck with snow-ball in the neck. Next day, pains in head, neck, and abdomen. In two days, unable to turn head, all movements painful. Pupils dilated. Difficult but intelligible speech. Spine extremely sensitive. Temperature 40.5° C. Treated by ice-bladder, warm baths, iodide of potassium. Cure.[3]

Case XIV.—Boy of fifteen. Blow on back with fist. In a little while severe pain, at first relieved by leeches, then returning with fever. Abscess formed, presenting on right side of sacrum, and there opened. Meningeal symptoms, however, persisted and increased; pain in back and all parts of the body became intensified; head retracted. There was no paraplegia, but, towards the end, incontinence of urine and fæces. Death after twenty-two days. Subperitoneal abscess at anterior part of sacrum, extending behind psoas muscle to left side of last lumbar vertebra, passing into spinal canal through lumbo-sacral foramen. External and internal surface of dura mater covered with greenish pus, as also the cauda equina. Membranes inflamed over entire extent of cord, the latter being firm and healthy. The lumbar and bronchial glands had a trace of tuberculous deposit.[4]

The situation of the abscess below the cord explains the absence of paraplegia; the presence of tubercle perhaps explains the slight resistance of the organism to the traumatism, or even suggests that tubercular deposit had been present in the cord or membranes, or overlooked:

Case XV.—Boy, five years. Fell on neck from roof of a house. In two days unable to turn head, on account of pain. Entire spine sensitive. Speech difficult. Then all movements painful. Temperature 40.5° C. Pulse 108-132. Treated by bromides, leeches, ice, and salicylate of sodium, with little success. Lukewarm baths and cold affusions. Finally mercurial ointment and iodide of potassium. Illness from March 22 to April 11, then complete recovery.[5]

Kahler describes a meningitis due to rheumatism, and relates the following case as an example:

Case XVI.—Girl of eleven, compelled to hard work amidst much cold and damp. Illness began with incontinence of urine and fæces; general weakness; persistent choreiform

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[1] Day, Lancet, May 5, 1883.

[2] Meningitis Spinalis, Rendu, Gaz. des Hôp., 1884.

[3] Förster, Jahrb. für Kinderheilkunde, Bd. xv., Hft. 3 u. 4.

[4] Wilks, Guy's Hospital Reports, 3d Series, vol. ii. p. 158.

[5] Förster, loc. cit.

trembling of the extremities; œdema with swelling of face, hands, and feet. After three months' illness, some incontinence of urine and fæces persisted, joined, however, with constipation. Dragging and boring pains in lumbo-dorsal region of back and along spine occurred often, radiating to limbs. In latter much tingling and formication. Œdema had disappeared; trembling persisted, so that child could not long stand upright, but only walked with support. Head affected with trembling. No tenderness on pressure over cervical or lumbar spine. Diagnosis made of chronic rheumatic spinal meningitis, with plastic exudation in cavity of arachnoid or in the pia, whose pressure upon the sacral and lumbar segments of the cord caused the incipient sphincter paralysis. The choreiform phenomena were held to be independent. Treatment by scarified cups, permanent vesication, camphor, senna, and diuretics effected a cure in four months.[1]

The characteristics of acute spinal meningitis, as illustrated by the foregoing cases, have already appeared less distinctly in the description given of the chronic meningitis of Pott's disease,—pain in the back, usually severe, increased by movement and pressure, often most intense at the neck, radiating to the trunk and limbs, accompanied by cutaneous and muscular hyperæsthesia, by fever, and sometimes by muscular spasm. Difficulty of speech seems liable to occur when the lesion begins in the cervical region; convulsions are imminent in young children. Exclusion of myelitis is made by the negative symptoms,—namely, the absence of motor or sensory or sphincter paralysis, and the absence of derangement of reflexes or of electrical reactions. The first indication that the inflammation is extending to the cord is an exaggeration of the reflexes, but so long as this symptom is isolated the myelitis is superficial. Tetaniform symptoms may be so prominent that the diagnosis from tetanus is rendered difficult. New-born children, liable to septic tetanus from the umbilical wound, are also liable to septic forms of meningitis. Gerhardt states that Billard has found purulent spinal lepto-meningitis in twenty cases out of thirty new-born children who had died in convulsions. In only six of these was the brain also implicated. Such a cause of convulsion would be associated with fever. An apyretic irritation of the spinal membranes at the same age is liable to occur by extension into the arachnoid cavity of blood from a hemorrhage at the base of the brain during parturition. This hemorrhage does not often excite inflammation of the meninges. (Erb.) Kahler[2] calls meningitis the hyperæmia of the spinal meninges, which may be produced during the convulsions of tetanus or during the convulsions of teething. In the subacute form of tubercular meningitis, tetanus, or even cataleptiform rigidities of the limbs, may also be most prominent symptoms. In a case seen by the writer in a boy of five, when the four limbs were raised vertically to the body, they would remain in this position indefinitely, in the waxen rigidity of the cataleptic trance. The child at the time was entirely unconscious.

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[1] Kahler, op. cit., p. 29. See also Ball, Du Rhumatisme viscéral, Thèse d'Agrég., 1866, p. 94. The cases quoted from other writers by Ball seem all adult. They can be referred to here only to justify the occasional etiology of undoubted spinal meningitis in attacks of rheumatism.

[2] Op. cit., p. 26.

NON-CARIOUS MENINGITIS FROM TUBERCLE.

Tubercle is a more frequent cause of spinal meningitis than is traumatism. "It is easy," observes Liouville, "to convince one's self that tubercular meningitis extends throughout the entire cerebro-spinal system."[1] The lesion in the cord, as in the brain, is principally located in the pia mater,—is a lepto-meningitis. Tubercular granulations extend along the vessels on the posterior surface of the cord, where the vascular supply is most abundant. The dura is injected on both surfaces, and sometimes covered with fragments from the visceral face of the arachnoid. The vascular net-work of the arachnoid contains tubercular granulations, and the membrane itself is changed into a thickened, irregular, opaque membrane, covered with little prominences. The subarachnoid cellular tissue is infiltrated with puriform turbid serosity; an abundance of citrine fluid accumulates in the cavity of the arachnoid, or, in its absence, all the membranes are agglutinated by adhesive inflammation. Tubercular granulations penetrate the fissures as they follow the Sylvian fissures of the brain. They may even follow the vessels into the cord-substance, and there agglutinate into a tubercular tumor, occasioning a special form of myelitis. Other granulations run along the nerve-roots.

These concomitant spinal lesions are the proximate cause of many of the symptoms observed in the course of cerebral meningitis, as the stiffness of the trunk, the tetanic attacks, the contraction and rigidity of the limbs, the jerking and trembling of their muscles; to a considerable extent, even the distribution of the motor and sensory paralysis.[2]

But tubercular disease may begin in the meninges of the spine, or a generalized tuberculosis localize its primary eruption there, before invading the cerebrum. The symptoms are then more insidious than in traumatic meningitis, developing more slowly, with less violence, and, although in reality far more serious, seem for a long time to indicate much less dangerous disease. After exclusion, if possible, of compression meningo-myelitis, nothing is of more importance in the prognosis of a spinal meningitis than to decide upon its simple or tubercular nature. The absence of traumatism, the insidious onset, low fever, irregularity in the march, long duration, together with signs of constitutional depression,—loss of weight, failing appetite, irritability of disposition, the secretion of abundance of very clear urine,—in a word, all the signs usually interpreted as pointing to a tubercular cause of local inflammation, must here be carefully considered. If the spinal symptoms have been preceded by a subacute pneumonia, whose resolution is still imperfect, if caseous glands can be detected around the bronchi or elsewhere, the probabilities of a tubercular origin of a spinal meningitis are of course greatly increased.

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[1] Archives de Physiologie, 1870.

[2] Liouville, loc. cit. According to Kahler (loc. cit., p. 43), tubercle of the spinal meninges is always secondary to cerebral or to general tuberculosis. But Liouville admits the possibility of primary spinal tuberculosis.

When the disease begins in the brain, it extends down the spinal canal by contiguity; hence the first symptoms appear in the neck. The difficulty of speech noted in some of the cases cannot be a spinal symptom: it implies the participation, primarily or secondarily, of the medulla and hypoglossal nerve. While tubercular spinal meningitis is almost invariably fatal, traumatic meningitis is far from being so,—indeed, tends towards recovery under appropriate treatment, unless the cord become rapidly and deeply involved.

INFECTIOUS MENINGITIS.

Diphtheritic paralysis, so long regarded as "essential," then as due entirely to infectious inflammation of nerve-roots (Buhl, Déjérine), has more recently been found to be associated with spinal meningitis. There are patches of localized meningitis around the nerve-roots affected with perineuritis,[1] but also diffused lesions of the meninges, with fibrinous exudation in the spinal canal, and hemorrhages in the cavity of the arachnoid.[2] There is in addition a slight tephro-myelitis;[3] the ganglionic nerve-cells are altered by the diphtheritic poison. This, according to Landouzy, is the primitive lesion; then follows lesion of the roots, and, finally, parenchymatous neuritis.[4]

After diphtheria, typhoid fever is the infectious disease most liable to be followed by paralysis. Yet, on the whole, such sequence is rare. Landouzy quotes from Colin a statistic of eight hundred cases of acute diseases, out of which only two were followed by paralysis.

It is highly probable, however, that the spinal meninges become irritated and congested under many circumstances in which severe pain in the back exists, but no paralysis appears. The violent rachialgia at the beginning of small-pox and even diphtheria is probably due to the circulation of the specific poison of the disease through the spinal pia mater, richly supplied with nerves. In heart- and lung-diseases, acute and chronic, the impediment offered to the entrance of venous blood into the thorax is liable to result, for the spinal cavity as for the cranial, in a great accumulation of venous blood in the meninges. On this account, Kahler enumerated heart- and lung-diseases among the causes of spinal meningitis; but inaccurately. For such secondary congestion is not an inflammation, but resembles the arrest of circulation in the sinuses of the brain, liable to follow cholera infantum.

CHRONIC SPINAL MENINGITIS.

Chronic spinal meningitis in children, unless we accept the rheumatic-choreiform affection described by Kahler as such, seems to exist only as a complication of vertebral caries or of intra-spinal tumor. The cord is always affected when the pia is involved; the lesion is that of a compression meningo-myelitis.

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[1] Pierret, Comptes-Rendus Soc. de Biol., 1876.

[2] Oertel, Deutsches Archiv für Klin. Med., 1871, viii.

[3] Déjérine, Archives de Physiologie, 1878.

[4] Landouzy, Des Paralysies dans les Maladies aiguës, Thèse de Concours, 1880.

Nevertheless, Seeligmüller relates as a case of chronic spinal meningitis the history of a girl twelve years old, which some observers might rather interpret as hysteria:

Case XVII.—Girl of twelve, blonde, anæmic, delicate, *irritable and egotistic,—so much so that her entire illness seems to have originated in a fit of jealousy against a sister* who, in her place, had been taken on a sleigh-ride. The illness began with vomiting, moderate fever, coated tongue, constipation, pains in epigastrium, head, back, and extremities. *Excessive vomiting lasted fourteen days*, then diminished, then returned after an indiscretion of diet and *lasted with few interruptions for nine weeks.* Coincidently occurred *clonic contractions* in the arm-muscles, then in the thighs, then in the back. By the ninth week these convulsions, which were painful to the patient, were sufficiently frequent *to resemble chorea.*

Paralytic symptoms began in the third week, in the neck and trunk, so that the patient could not hold up her head nor sit up. In the sixth week the lower extremities were paralyzed and also stiff; there was a less intense degree of paralysis of the upper extremities. There was hyperæsthesia of the spinal column (*spinal tenderness*), *none of the skin or muscles; complete analgesia to the prick of a pin*,(locality not stated). There was obstinate constipation, slight incontinence of urine.

Patient was seen by Seeligmüller in the fourth month: was then completely helpless, carried dangling like a doll on the arm of an attendant. The head could not be held up; the lower extremities were completely flaccid. Not the least voluntary movement except of the toes; the feet were in moderate equinus. *There were, however, no emaciation, no contractions, hardly any loss of electrical reaction.* The patellar and plantar reflexes, on the contrary, were lost.

The treatment instituted was electrical, hydrotherapeutic, and roborant, and lasted ten weeks, with only slight improvement. A powerful supporting apparatus was then procured, and the *same day that this came home the child began to use it*, though up to this time she had not made the least exertion. In six weeks she had improved immensely, and was entirely cured by December.[1]

In citing this curious history I have emphasized by italics the record of such circumstances as seem to me to show that the symptoms were not due to meningitis nor to anterior polio-myelitis, whose possibility the author discusses, but rather to a succession of hysterical affections.

IDIOPATHIC MYELITIS.

Observations of idiopathic myelitis in children are few. (Gerhardt.) It most frequently originates in accidents, but it is not the blows or other mechanical violence that excite meningitis, but rather exposure to damp, cold, and fatigue. Thus:

Case XVIII.—Boy ten years old, very poor, had suffered greatly from exposure to cold. Went to bed apparently well, but was seized in the night with convulsions, which succeeded each other for fifteen hours, and terminated in death. At the autopsy the brain was found congested over its entire surface. The spinal cord looked "as if it had been dipped in blood." It was softened and diffluent for a space of two inches in the lower dorsal region. "The most rapidly produced case of softening on record."[1]

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[1] Lähmung nach Spinal Meningitis, Archiv für Kinderheilkunde, 1880, Bd. ii., 2, S. 133.

Case XIX.—Boy of ten years. After severe exposure to cold and wet, accompanied by fatigue, he was seized with chill and convulsions, followed by giddiness and vomiting. Remained in bed ten days, and then found himself unable to stand or walk without assistance. There was incontinence of urine, and occasionally of fæces. On admission to hospital, six weeks from beginning of illness, there was complete sensory and motor paralysis of lower extremities, and these were wasted and shrunken. There was tenderness on firm pressure over upper lumbar spine. The boy was treated by a blister to the spine, and the internal administration of iron, cantharides, and quassia. Treatment was begun January 22; on the 29th the incontinence of urine had ceased; on February 5 sensation was partly restored, the motor functions improved so that the boy could walk a few yards. Recovery was complete in April. No electrical treatment was used. Strychnine always had ill effects.[2]

In some cases an acute transverse myelitis has been found after a fall:

Case XX.—Child two and a half years old, twelve days after a fall, had left-sided hemiplegia. Seven weeks later both lower extremities were paralyzed and anæsthetic, both upper extremities paretic. The reflexes were abolished. The head was retracted; the temperature 37.7° C.; the rectal sphincter was paralyzed. The retraction of the head and paresis of the arms disappeared later in the disease. The child died from broncho-pneumonia. The gray substance of the cord was found swollen. Red softened foci in anterior horns of lumbar cord. Around these foci, perivascular spaces filled with granular leucocytes. On left side, ganglia-cells almost disappeared; on right side, degenerated. Diffused infiltration of leucocytes. Sclerosis the entire length of the antero-lateral columns.[3]

Case XXI.—Girl, nine and a half years, fell out of a window forty feet high. Picked up insensible, and remained so for two days. Then her four limbs were found to be paralyzed and anæsthetic. The sphincters were paralyzed. Sensation returned in a week; in four weeks the arms could be moved a little, and the urine was retained. Then the right leg began to grow stiff, though its rigidity would sometimes relax spontaneously. It was not wholly removed by chloroform.

On admission to hospital, with the right leg extended and stiff, the patient had regained feeble voluntary power over left. Faradization excited reflex flexions at all joints. There was well-marked ankle-clonus, but no exaggeration of the reflexes to tickling the soles of the feet. Sensation was natural in all the limbs. At the arms the paralyzed muscles were much more wasted, and the wasting was conspicuous in the small muscles of the hands. Great rigidity of the flexors of the fingers in both hands. The right pectoral muscle and the muscles of the back were much wasted. The spine was curved to the right, and the patient could not sit up without support. The respiration was abdominal, the chest scarcely moving.

Two months later the condition of the thorax and upper extremities was the same, but the girl could stand alone and walk a little.

In this case the myelitis, after an initial generalization and the setting in of descending secondary degeneration, seems to have concentrated itself on the anterior cornua of the cervico-dorsal region. There developed in consequence a condition resembling a very acute progressive muscular atrophy.[4]

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[1] MacSwiney, Dublin Medical Monthly, 1867, p. 243.

[2] Abbotts Smith, Lancet, August 17, 1861.

[3] Turner, Jahrbuch für Kinderheilkunde, 1879, Bd. xiii.

[4] Gee, St. Bartholomew's Hospital Reports, 1880, vol. lxvi.

A similar case is related by Gull; but in this the localization was effected at the beginning:

Case XXII.—Boy of fifteen, received a blow of the fist between the shoulders. A week later his head began to droop, then the muscles of the arms to waste, so that they hung useless. The intercostals ceased to act, and the breathing became diaphragmatic. The erector spinæ muscles and the lower two-thirds of the trapezii also wasted. The ribs were flattened from paralysis of the intercostals. Fourteen months later the patient could walk, but could not sit up without support. The wasted muscles, as in idiopathic progressive muscular atrophy, contracted to faradism in proportion to their mass.[1]

The following case of focal myelitis was developed without obvious antecedent cause, and associated with much more extensive meningitis:

Case XXIII.—Boy of eleven. Admitted to hospital in February, having suffered for a week from pains in the small of the back, gradually extending around the body on both sides as high as the umbilicus. These pains were spontaneously worse at night, but walking caused severe pain. Pressure over the spinous processes was painful. There was neither sensory nor motor paralysis. The sphincters were intact. Inveterate fever was present. Two days after admission, the temperature rose to 103.2° F., the fever being attended by profuse perspiration. Slight hyperæsthesia in legs; incontinence of urine, constipation, priapism. Early in March the boy became completely paraplegic in the lower extremities, and anæsthetic to the seventh intercostal space and the fifth dorsal vertebra. The sphincters were paralyzed. Burning pain in back, increased by pressure. Temperature in groin 102.1°, in axilla 100.4° F. Bed-sores on sacrum and trochanters. Death six weeks later. Inflammatory lymph was spread over the lower dorsal portion of the cord. The membranes were very much injected, and partly adherent to the bones. Opposite the fifth dorsal vertebra the cord was in white softening for an extent of half an inch.[2]

The following case lies just beyond the limits of childhood, in a girl of eighteen:

Case XXIV.—Sudden attack of nausea and vomiting, with sharp pain in the cervical spine. In two minutes loss of power in arms, in a few minutes more total paralysis of them. In fifteen minutes violent tremor of lower extremities, followed by paresis, and finally total paralysis. The abdominal muscles were flaccid, the breathing almost wholly diaphragmatic and dyspnœic. Sensation and reflexes remained normal. In four days there was a superficial bed-sore on the buttock. On the eighteenth day violent retching, severe dyspnœa, and cyanosis for half an hour. Several similar attacks occurred during the next three days, and finally death from asphyxia on the twenty-second day of the illness. An acute myelitis was found in the cervical region from the origin of the third pair of nerves to the dorsal region, where it ceased abruptly. The blood-vessels were greatly enlarged. The "nerve-elements in the anterior and central parts of the gray matter replaced by numbers of granular corpuscles."[3]

From the extreme suddenness of the attack, it is to be inferred that the myelitis originated in hemorrhage of at least capillary dimensions, although no observation of hemorrhage is made at the scantily-described autopsy.

Sometimes the focal myelitis is of limited extent, and its symptoms soon become merged in those of the descending sclerosis, as in the next case:

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[1] Gull, Guy's Hospital Reports, 3d Series, 1858, vol. xiv. p. 195.

[2] Oxley, British Medical Journal, October, 1870.

[3] Shann, Lancet, December, 1881, p. 1048.

Case XXV.—Girl of twelve. Without any evident cause, her legs began to give way; she suffered from severe pains in the knees and legs, and these would draw up spontaneously. In a fortnight she was completely paraplegic, with complete loss of sensation below the waist. Pain in back on moving, and radiating pains in limbs. Two weeks later, paralysis of sphincters, which lasted three months. Bed-sores occurred at the same time. In a month, however, sensation returned, and in three months the patient was able to move her limbs, but could not stand. The legs were very stiff, the feet pointed in rigid plantar extension, the sural muscles permanently contracted. The legs were crossed, from contraction of the adductors. Excessive patellar reflexes, but the contraction of the sural muscles prevented the development of ankle-clonus. The case was a type of Erb's spastic paraplegia, except in the fact that it was secondary to a focal meningo-myelitis. The patient had a very marked lateral curvature of the spine, which Dr. Buzzard, who relates the case, thought might have some etiological relation to the medullary disease, and therefore proposed to treat by suspension and the plaster jacket.[1]

Another case reported by the same author (Case XXVI.) belongs to a special form of myelitis,—special at least in its etiology, which was syphilitic. In this case, also, the secondary spastic paraplegia was the most marked feature. The patient recovered completely under the influence of twenty-grain doses of iodide of potassium given three times a day. The author attributes this recovery to the removal not of an overgrowth of connective tissue in the lateral columns, but of hyperæmia and liquid effusion in the same locality.[2]

SYPHILITIC MYELITIS.

It is a very important fact that inherited as well as acquired syphilis is capable of exciting myelitis. Thus:

Case XXVII.—Boy, fifteen, had had previous symptoms of inherited syphilis. Weakness in legs for two years. A month before admission, pain in lumbar region, increased on pressure, then shooting pains in lower limbs, muscular twitchings; in a month, complete paraplegia. Admitted to hospital with paralysis of both motion and sensation; paralyzed muscles rigid, and reacting excessively to electricity. Superficial and deep reflexes both exaggerated. Pain and tenderness in lumbar region persistent, accompanied by a sense of constriction at the abdomen. Paralysis of the bladder, incipient bed-sore, no fever. Sensibility began to return in a month, and patient, under antisyphilitic treatment, entirely recovered in four months. The attack was attributed to a syphilitic thrombosis, followed by circumscribed, but not irreparable, softening of the cord.[3]

The lesions, though not the symptoms, of a syphilitic myelitis are described by Kahler in the case of a child five months old, who died in the course of a congenital syphilis:

Case XXVIII.—The cord presented nothing abnormal microscopically. Below the decussation of the pyramids was a patch of gray discoloration in the left lateral column from six to seven millimetres broad. In this were sieve-like perforations, which under the microscope were seen to be perivascular spaces. The ground-substance of the patch consisted of a thick net-work of the finest fibrillæ, containing only a few cells, no nerve-fibres, an excess of blood-vessels, whose walls were thickened and lumen narrowed.[1]

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[1] Buzzard, Lancet, July 23, 1881, p. 123.

[2] "Anything which interferes with the transmission of motor impulses from the cerebrum down the lateral columns exaggerates the tendon reflexes, because the normal balance between the reflex function of the cord and the controlling influence of the brain is thereby disturbed."—Buzzard, loc. cit.

[3] Dixon Mann, Lancet, July 21, 1884.

Savard (Étude sur les Myélites syphilitiques, Thèse de Paris, 1882) quotes two cases of paraplegia in congenital syphilis, related by Leubuscher and Henoch; also a case, described by Potain, of a six-months-old fœtus with a syphilitic liver whose cord was completely sclerosed. The author himself has observed no case under the age of eighteen.

TUBERCULAR MYELITIS.

Tubercle, which has been shown to be not infrequent in the spinal meninges, and the cause of the inflammation, may also be located in the cord itself. It is then not disseminated, but concentrated in the form of tumor.

Case XXIX.—Boy three and a half years old. After an illness of only three days, both lower extremities became paretic, the reflexes slightly exaggerated, and the sphincter of the bladder relaxed. Two months later the child was found considerably emaciated, with an eschar on the right gluteal region and left analgesia below the eighth dorsal vertebra. Sensibility to touch was preserved. Extreme hyperæsthesia over the same territory, complete paraplegia, increased reflexes; no pain over spinal column. Marked apathy. Death six days after admission. Cheesy tubercle at lower end of dorsal cord in the gray substance. Surrounding white substance swollen and gelatinous. Slight tubercular meningitis at base of brain.[2]

Myelitis due to a tubercular neoplasm is essentially a compression lesion. But as the compression is exercised within the cord, instead of first irritating the membranes, the consequences are from the beginning a necrobiotic process, and not an irritative proliferation of connective tissue. The symptoms, therefore, from the beginning indicate depression of function, and are not preceded by initial symptoms of irritation.

ACUTE INFECTIOUS MYELITIS.

When the poison of acute infectious diseases invades the spinal canal, the meninges and nerve-roots are more profoundly affected than the cord itself. (See *supra.*) As has been already mentioned, however, the ganglionic cells of the anterior cornua also show traces of the action of the poison. Landouzy (*loc. cit.*) insists further on the hortensia tint of the nerve-centres observed in autopsies made after acute infectious diseases. This is not due either to congestion or to hemorrhage, and the author attributes it to a solution of the hæmoglobin, which transudes through the walls of the blood-vessels and tinges the surrounding tissues. This solution is attributed to the high bodily temperature existing during the disease; and the question arises whether the ganglionic cells are also directly affected by the heat, and whether the fibrillary net-work and cylinder axes of nerve-tubes may become thereby coagulated. Such changes would be the initial lesions of the myelitis.

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[1] Kahler, Myelitis in Hereditary Syphilis, Jahrbuch für Kinderkrankheiten, 1879, Bd. xiv. S. 392.

[2] Eisenschutz, Jahrbuch für Kinderkrankheiten, Bd. iii., N. F., 1870.

MacSwiney reports the following case after a "tonsillitis" that may very well have been diphtheritic, though the author does not himself seem to think so:

Case XXX.—Boy of six. Paralysis first manifested by a tendency to trip in running. Then the head was held depressed to left shoulder, and two months later there was well-marked wry-neck. Then the legs became weak, also the arms, but not the hands, while the muscles of the trunk were enfeebled. The child walked in a tottering, straggling way. At the end of three months there was complete motor paralysis of the four limbs. Muscles responded perfectly to electricity; reflexes undecided. Sensation normal. After eight days of complete paralysis, motility began to return to arms, then to legs, and in a fortnight from the beginning of convalescence the boy left the hospital perfectly well.[1]

Möbius diagnoses as multiple neuritis rather than myelitis a somewhat analogous case occurring after pertussis:

Case XXXI.—Child of three. The paralysis began in the legs, ascended, and finally affected all the muscles of the body, even the diaphragm, so that the respiration was carried on by the intercostals, and imperfectly, as shown by the presence of cyanosis. Tendon reflexes absent; superficial reflexes preserved. Sphincters intact. Improvement began in a month, and recovery was complete in two months.[2]

Tetany, the spasmodic affection of infants which has generally been considered functional, has been found associated with myelitis:

Case XXXII.—Girl of eighteen months, had cramps of the four extremities, diagnosed as arthrogryposis, during an attack of bilateral catarrhal pneumonia with diarrhœa. The cramps occurred every day, persisting several hours. Death from exhaustion. The cervical cord was found to be softened, with a flattening of its anterior and posterior horns.[3]

The spinal paralysis of the new-born child, which, as already mentioned, is sometimes due to a hæmatorrhachis, or a meningitis excited by that, has also been attributed to a species of traumatic myelitis due to traction on the legs at birth.

SPINAL HEMORRHAGE.

The most frequent spinal lesion, however, in new-born children is the third of those we have here under consideration,—namely, hemorrhage. This resembles the cerebral hemorrhage of parturition, in occurring oftener in the meninges than in the nerve-tissue itself. It may be produced without any obvious traumatism to the spine;[4] and, if the child survive, paralysis of greater or less duration is imminent.[5] Litzmann relates the following case:

Case XXXIII.—Breech presentation. Some uterine inertia, also narrowed pelvis. Head, however, did not seem compressed. Some hours after birth child's voice feeble, clonic twitching of muscles, and in twelve hours legs paralyzed and insensible. Reflexes not obtained till the tenth day. Partial paralysis of bladder. In five or six weeks return of sensibility. In left foot some permanent plantar flexion. At five months still completely paraplegic; sensibility diminished. Reflexes absent in peroneal and tibial anticus muscles. Electrical reactions lost in both crural territories, while the thigh flexors and calf muscles respond to faradism.

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[1] MacSwiney, Dublin Monthly, 1884, vol. lxxvii. p. 464.

[2] Möbius, Centralblatt für Nervenheilkunde, 1886.

[3] Geza Dulacska, Centralblatt für Klin. Med., 1882, Bd. iii. S. 528; Litzmann, Archiv für Gyn., 1881; and Medical Times and Gazette, February 26, 1881.

[4] Weber, Beiträge zur Pathol. Anat. der Neugeborenen, Kiel, 1859 (quoted by Litzmann).

[5] Little, Transactions of the Obstetrical Society of London, 1862.

Electrical treatment begun in eighth month, three séances a week. Galvanization of cord, also labile excitation of nerves and muscles of paralyzed limbs. Great improvement of paralysis.[1] Diagnosis of hemorrhage in spinal canal, with gradual reabsorption and shrinkage of the clot.

The tendency to spontaneous disappearance of the lesion complicates the estimate of the therapeutic value of galvanism in a case like the foregoing. On the other hand, the process of shrinkage, if the clot be closely applied to the cord, is liable, from the irritation of pressure, to excite a cortical myelitis, a sclerosis with descending degeneration. No symptoms of the latter—*i.e.*, no spastic paraplegia—are recorded in this case.

The spinal hemorrhages produced during labor are almost always associated with an effusion of blood into the cavity of the arachnoid at the base of the brain, and the spinal effusion is an extension from this. It therefore occupies the corresponding arachnoid cavity of the cord within the dura; ordinary traumatic hemorrhage, on the other hand, usually occupies the spinal canal, external to the cord and its membranes, and this therefore is the seat of the lesion in older children. In such case, although the onset of the symptoms be sudden, they at first consist only in pains, generally intense in the back, but radiating more or less to the trunk or limbs. Myelitic symptoms proper—numbness and other paræsthesias and paralysis—develop more gradually, and as the hemorrhagic clot begins to compress the cord. Thus, in a case by Foot:

Case XXXIV.—Boy, eight years old, fell down a flight of stone steps. There was obscure malaise for forty-eight hours, then incomplete motor paralysis of the four limbs, dysphagia, loss of intercostal breathing. The head seemed loose on the neck, falling forward. The child was quite unable to stand, or even to raise himself in bed. Severe pain in back and neck. No alteration of sensibility, reflexes, or sphincters. Fed on enemata exclusively for six days. No respiratory movements except of diaphragm and alæ nasi. Both sterno-mastoid muscles paralyzed. The bronchial tubes became loaded with mucus; the face puffed and violet, from the imperfect shallow respirations, though these were forty-one a minute. Violent headache and delirium, due to venosity of the blood, and relieved by post-auricular leeching. Temperature ranged from 99 6° to 102.8° F. At the end of a week the patient began to recover power of swallowing, and the sterno-mastoids and trapezii to regain their tone. The leaden color of the face disappeared. In ten days he was able to raise his hand to his mouth; on the fourteenth, to raise his head from the pillow; on the twentieth, to drink easily, and to stand with support. In twenty-six days left the hospital, able to breathe naturally and use his limbs, though still somewhat unsteady in walking.[2]

Dr. Foot attributes the foregoing symptoms entirely to a temporary compression of the cord, between the third and fifth vertebræ, from forcible flexion of the neck during the fall, and consequent functional disturbance. He does not think myelitis could have occurred, as recovery was too rapid. The case beautifully illustrates paralysis of the spinal accessory nerves,[1] the phrenic—originating higher up than the seat of the lesion—being intact. The intercostal nerves were also paralyzed. The paralysis of the pharynx and the glottis coincidently with that of the sterno-mastoids indicates the path which is taken by the spinal accessory filaments in the pneumogastric nerves. But it is remarkable that the comments on this case fail even to discuss the origin of the symptoms in a traumatic hemorrhage. Yet to this they were in all probability due. It is in the highest degree improbable that in the wide cervical canal the cord could have been compressed unless through fracture and displacement of the vertebræ, which evidently had not occurred. In the following case the cervical hemorrhage was demonstrated:

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[1] Jahrbuch für Kinderkrankheiten, 1881, Bd. xvi.

[2] Dublin Monthly, 1881, vol. lxxi.

Case XXXV.—Healthy girl of fourteen. On the 2d of May noticed weakness in fingers, which persisted on the 3d. On the 4th was unable to move her arms except at the wrists; could not pick up a pin. On the 5th the action of the intercostal muscles became impaired. A moist crepitant râle was heard all over the chest. On the 6th great loss of power in all the voluntary muscles of respiration, and in the muscles of the arms, back, and chest supplied by cervical nerves. The diaphragm was also becoming fixed, and the cheeks slightly livid. The temperature fell. Death occurred thirty hours later, without pain or loss of sensation or of consciousness.

The brain was found congested and soft; there was a softened spot in the cerebellum. The whole cervical portion of the spinal cord was embedded in an oblong clot of dark venous blood lying outside the membranes; all the cervical nerves passed through this clot. The effusion apparently took place very gradually and coagulated slowly, compressing the nerves so as to cause death by paralysis of respiration. The condition of the cord is not described.[2]

In the following case a hemorrhage, probably similar in nature, was recovered from, because occurring in a far less dangerous locality:

Case XXXVI.—A six-year-old girl fell on her buttocks from a chair. For nine days there were no symptoms of injury. Then severe pains in both legs, followed in a few hours by paraplegia. The next day convulsions, which, subsiding, left the paralyzed limbs hyperæsthetic. Motor power began to return in a month, and recovery was complete in four months.

The diagnosis was made of a hemorrhage within the dura mater, from blood-vessels dilated in consequence of the fall, the effused blood compressing the cauda equina.[3] But, as already stated (*supra*), lesions of the cauda equina determine paralysis of isolated muscles, not complete paraplegia. It is probable that the hemorrhagic clot lay above the cauda equina and upon the lumbar cord itself.

The characteristic circumstance distinguishing primary hemorrhage, within or without the cord, from even the most acute myelitis, is the suddenness with which the symptoms of complete paralysis appear.[1]

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[1] Under which title it is described.

[2] Hughlings Jackson, Lancet, July, 1869.

[3] Jahrbuch für Kinderheilkunde, 1876, Bd. ix. N. F.

Thus, in a case by Goldtammer:

Case XXXVII.—Girl, fifteen years, not yet menstruated, of good health previously, felt suddenly, while quietly seated, an extremely violent pain between the two shoulders. The pain extended rapidly to the right arm, then the left, then encircled the base of the thorax. The girl fell from her chair, and noticed at the same time a paralysis of her right leg; the left was paralyzed half an hour later. In two hours there was complete paraplegia of the lower extremities, and absolute anæsthesia on both sides to nipples in front and fourth dorsal vertebra behind. Pains rapidly disappeared. Reflexes preserved, and electrical reactions; bladder paralyzed. After four months signs of descending degeneration, atrophy of thighs and legs, cystitis, eschars; death in a year. Hemorrhagic focus in cord at level of second dorsal vertebra. Cord healthy above and below, except descending degeneration of lateral columns, and ascending sclerosis of columns of Goll, as far as the calamus scriptorius.[2]

When the symptoms are preceded by prodromata, it is to be inferred that the hemorrhage is itself the result of a meningitis or myelitis, as in the following case:

Case XXXVIII.—Delicate girl of ten years. Subject during undefined period to attacks similar to that which preceded by four days her death. There was general prostration, with alternations of heat and cold, stiffness in the neck, pain over the cervical spine, with inability to move the left arm on account of the pain. Neither vomiting nor convulsions. Apparent amelioration, then sudden death during the act of defecation. In the lower part of the cervical cord the tissue was softened, and extensively infiltrated with blood. There was a coagulum of effused blood the size of a bean. The precise situation of the coagulum is not stated, but presumably it was in the middle cervical region, as death is attributed to sudden "compression" (irritation?) of the phrenic nerve. More probably the death was caused by the shock communicated to the respiratory centre.[3]

The meningeal hemorrhage which is the immediate consequence of traumatism seems often to avert hemorrhage from the cord, so much more rare is hæmatomyelia than hæmatorrhachis. It has been even asserted that hemorrhage into the cord is never really a primary process, but that it is always preceded by a myelitis, in which the blood-vessels become dilated and the tissue of the cord softened.[4] Capillary hemorrhages may occur in the course of a myelitis, acute or chronic, without occasioning new symptoms. They are sometimes even found after having remained latent during life and developed during an attack of some general disease. Thus, Pick discovered abundant minute hemorrhages in the cord of a ten-months-old baby (Case XXXIX.) who had died of cholera infantum. The blood-vessels were stuffed with corpuscles. The hemorrhages were most numerous in the gray substance, and especially in the posterior cornua. The author suggests that such minute hemorrhages may very possibly be the real cause of many so-called reflex paralyses.[1]

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[1] Fox relates an interesting case in an adult woman, who, while carrying a heavy weight, felt a sudden pain across the loins, and immediately lost all motor power in the left leg. Medical Times and Gazette, August, 1876.

[2] Revue des Sciences Médicales, viii., quoted by Grasset, op. cit., p. 540.

[3] Page, Lancet, March 20, 1880.

[4] Hayem, Thèse d'Agrégation, 1872. Leyden relates a case in a woman, who, two days after confinement, was seized with pains and motor paresis of the lower extremities. In two days more paraplegia was complete, and anæsthesia reached to the ribs; the sphincters were paralyzed, the reflexes abolished. Death on the fifth day. The dorsal cord was found softened through an extent of five centimetres. A hemorrhagic clot occupied the posterior columns and the peripheric part of the antero-lateral columns, and here and there reached the gray substance. The hemorrhage was evidently consecutive to a septic myelitis. Revue des Sciences Médicales, April, 1889, p. 490.

But intraspinal hemorrhage, other than capillary or from intracranial hemorrhage, is rare,[2] and its causes always obscure. External and internal pachymeningitis, for the cord, as for the brain, is a causal condition, the vessels in the inflammatory neoplasm easily rupturing. Traumatisms, or the exaggerated congestions consequent upon certain diseases, tetanus, hydrophobia, epilepsy, strychnine convulsions, or certain infectious diseases, especially yellow fever, with its characteristic tendency to hemorrhages, may all be counted in the etiology of the accident.[3] Hemorrhage into the cord is theoretically possible from syphilitic arterial thrombosis. Practically it seems always to have been consecutive to myelitis. It has been seen in a child seven months old.[4]

The prognosis of intraspinal hemorrhage is seen to depend—

1st. On its general situation. Extremely dangerous in the cervical region, the danger diminishes in proportion as the cauda equina is approached.

2d. On its occurrence within the membranes,—hæmatorrhachis,—or within the cord itself,—hæmatomyelia. The latter is much the more dangerous, and, if the immediate accidents are survived, is unavoidably followed by some degree of myelitis. Meningeal hemorrhage may occasion only a superficial meningitis, not causing paralysis; or, on the other hand, during the retraction of the clot the cord may be compressed, and the symptoms and lesions of a compression myelitis be induced. If the nerve-roots are involved in the clot, irritative "root symptoms" will precede the paralysis.

3d. The third element in the prognosis of spinal hemorrhage is, naturally, its extent. The sanguineous effusion may destroy so large a part of the central gray substance of the cord as to destroy its trophic centres, and hence cause rapid decubitus and sloughing on the trunk.

4th. The condition of previous disease of the cord or meninges does not seriously influence the prognosis apart from other circumstances; for, as has already been noted, such disease exists in the majority of cases.

5th. Extremely localized hemorrhages do not produce paraplegia, but some special form of myelitis or neuritis. Thus, if limited to one anterior horn, the hemorrhage occasions, or accompanies, an anterior polio-myelitis, with paralysis of a single limb or group of muscles. The hemorrhage, however, is then rarely accidental, but an element of a special systematic disease.

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[1] Pick, Jahrbuch für Kinderkrankheiten, 1883, Bd. xix.

[2] "Spontaneous meningeal hemorrhage is unknown in early childhood." Gowers, op. cit., p. 209.

[3] Grasset, op. cit., p. 578. We have cited a case (Case XII.) where epilepsy was followed by meningitis.

[4] Clifford Allbutt, Lancet, 1870, vol. ii. p. 84.

DIAGNOSIS.

The several steps in the diagnosis of myelitis, spinal meningitis, and spinal hemorrhage are the same for the child as for the adult. They have already been indicated in the description of the compression myelitis of Pott's disease.[1] Disease within the spinal canal is indicated by the occurrence of bilateral paralysis, motor and sensory, or exclusively motor, or predominantly motor, associated with paræsthesias and hyperæsthesias. It is the bilateral character of these symptoms, and the absence of lesion of the cerebral nerves or of the intelligence, that exclude a cerebral origin for the paralysis.

Pain in the back, or tenderness on pressure over the spine, pains radiating from it, girdle-sensations around the waist, are also important, though less pathognomonic, symptoms. Rigidity of the spine and limbs, muscular spasms and contractures, retraction of the head, or curvature of the spine, are important indications of spinal disease. Exaggeration and abolition of the reflexes, loss of electrical reaction in paralyzed muscles, together with their atrophy, paralysis or tonic spasm of the sphincters, alkaline urine, cystitis, decubitus, are all phenomena to be expected. Respiratory disturbance without demonstrable lesion of the thoracic viscera or kidneys points to spinal-cord disease, and the latter may become complicated with bronchitis, pulmonary congestion, or œdema, which are, nevertheless, evidently secondary. According to the seat of the disease in the cord will there be interference with intercostal or diaphragmatic breathing, or paralysis of the spinal accessory, or shock to the medullary centres, or Cheyne-Stokes respiration. Alterations of the pulse, pupillary phenomena, profuse sweating from vaso-motor disturbance, all occur in the course of spinal-cord disease, and, taken with more decisive symptoms, would tend to confirm the diagnosis. Vomiting, convulsions, and fever, of course, do not of themselves point to an intraspinal affection; but when associated with special localizations of pain they help to indicate that the cause of the latter is an inflammatory process within the spinal canal.

In young children any acute spinal affection is apt, at the outset, to be accompanied by cerebral symptoms, not only the vomiting and convulsions already mentioned, but also headache, delirium, and retraction of the head.[2] If, however, these be due only to sympathetic hyperæmia of the cerebral meninges, they will subside, while the symptoms characteristic of spinal lesion will persist and become predominant. Epidemic cerebro-spinal fever is distinguished by the eruption (present in more than half the cases), the aural symptoms, enlarged spleen, persistence of cerebral symptoms, often the special violence of the vomiting, the absence of traumatism, often the presence of a demonstrable endemic or epidemic influence.

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[1] See Lange, Jahrb. für Kinderkrankh., 1879, Bd. xiii. S. 94.

[2] Retraction of the head may be called either a spinal or a cerebral symptom.

After excluding cerebral disease, rheumatic fever, tetanus, and multiple neuritis remain as the only febrile non-spinal affections which could simulate the initial phenomena of acute meningo-myelitis. Of the first, it is only the exceptional form localized in the muscles of the back which is liable to occasion confusion. The back may then be not only painful, but rigid almost to opisthotonus. The pains, however, do not radiate to trunk or limbs, and from the latter are also absent all the other symptoms habitually present in meningitis. Rheumatism could not be confounded with pure myelitis. In tetanus the tonic spasms begin at the jaws, while these are affected last, if at all, in meningitis. The spasms are intermittent, and during the remissions the patient may be free from pain and muscular rigidities. Confusion, indeed, is possible only between incipient stages of tetanus and meningitis; well-marked forms of either have each a physiognomy perfectly characteristic.

Multiple neuritis is more difficult to distinguish from myelitis. Certain forms of disseminated paralysis, as that following diphtheria, have been shown to be sometimes of peripheric and sometimes of central origin. "In neuritis the sphincters escape; there are no girdle-sensations, bed-sores, or cystitis; the advance of the paralysis is usually from the legs to the forearms, the trunk and thighs escaping, and, as a rule, only the distal portions of the extremities are paralyzed. There is usually some ataxia; the tenderness on pressure is over the affected nerves and muscles, while it is absent from the spine."[1]

The second step in the diagnosis, after the previous analysis shall have localized disease within the spinal canal, consists in the special differentiation of the spinal disease. Tubercular meningitis, which we have considered among the idiopathic and traumatic forms of spinal disease, is usually cerebro-spinal. It may be impossible during several weeks to distinguish it from the subacute form of cerebro-spinal fever, or an attack of the latter may become the starting-point of tubercular disease of the meninges.

Multiple sclerosis has been observed in children, and is said to have a certain predilection for the age of three or four years.[2] The insidious commencement and chronic march of this disease suffice alone to distinguish it from acute transverse myelitis. The trembling upon intentional movements is as pathognomonic of diffused sclerosis in children as in adults, and is quite absent in common meningo-myelitis, even with its secondary sclerotic degenerations.[3]

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[1] Multiple Neuritis, M. Allen Starr, New York Medical Record, January and February, 1887.

[2] Marie, Revue de Médecine, Juillet, 1883, Sclérose en Plaques chez les Enfants.

[3] It is more liable to be mistaken for chorea.

Among the systematic diseases of the cord, the anterior polio-myelitis so frequent in childhood is particularly to be considered in the diagnosis. At the very outset, and perhaps for several days, the diagnosis might be impossible. For the "infantile" paralysis may be ushered in by convulsions and vomiting, and for several days be attended by fever, pain in the back and limbs, and diffused or paraplegic paralysis. The symptoms, however, are usually much less violent; especially is it noticeable that the pain is (usually) slight or altogether absent.[1] There are no muscular rigidities or spasms; the reflexes are unaffected until they begin to diminish,—thus are never exaggerated. In the great majority of cases the initial paralysis recedes after a few days, and remains limited to certain groups of muscles, which alone atrophy and lose their electrical reactions. The latter phenomena are produced with special rapidity.

During the period of suspicion the diagnosis should incline towards anterior polio-myelitis, on account of its much greater frequency, even after a traumatism.

Among the other systematic diseases of the cord, true locomotor ataxia is almost unknown in childhood. Friedreich's disease is characterized by its insidious march, its hereditary character, and the precedence of ataxic over paralytic symptoms. Sclerosis of the lateral columns, occasioning the spastic paraplegia or hemiplegia which is by no means rare in childhood, is attended by a history of cerebral disease or of focal myelitis, or by symptoms pointing to the persistence of one or the other.

If the question of diagnosis has been narrowed down to a question between common myelitis, transverse and focal or diffuse and ascending, meningitis, and hemorrhage, the third step remains to be taken in deciding between these three.

Inflammations of the meninges and cord are usually associated, and the term meningo-myelitis, though intended to be limited to lepto-meningitis, is for children the most appropriate designation for all forms. The disease may predominate in either the cord or the meninges, but is hardly ever limited exclusively to one or the other through its entire course. The predominance of pain, muscular spasm, and rigidity in the back and limbs points to meningitis; the predominance of motor paralysis, and still more the occurrence of sensory paralysis, indicate myelitis. (See Compression Meningo-Myelitis.)

The diagnosis of hemorrhage depends, as has been shown, mainly on the suddenness with which the symptoms occur. Hemorrhage into the meninges is attended with much pain and little paralysis at the beginning, but this is liable to increase from compression of the cord by the retracting blood-clot. With hemorrhage into the cord the paraplegia is sudden and complete from the beginning. Sudden exacerbations of symptoms in the course of a myelitis often indicate an intramedullary hemorrhage.

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[1] When pain is severe, it is to be inferred that a localized meningitis complicates the systematic lesion, or that the medullary lesion has extended to the posterior root zone.

PATHOLOGICAL ANATOMY.

The pathological anatomy of meningitis, myelitis, and intraspinal hemorrhage is, like the elements of diagnosis, the same in the child as in the adult. Only a brief description need, therefore, be given.

Tubercular disease, relatively rare in the adult, is much more frequent in children. Tubercular meningitis has already been described. Tubercle of the cord exists as a solitary tumor, giving rise to the symptoms of tumor of the cord, which are so difficult to distinguish from chronic myelitis, and which, indeed, are immediately due to a myelitis excited by the tubercle.

Compression myelitis, the other most frequent—almost characteristic—form of transverse cord-disease in childhood, has also been described.

In chronic myelitis—which in children appears hardly ever to occur except as a result of compression, or, in a subacute form, as a sequel to acute myelitis—the lesions closely resemble those of compression, being especially interstitial, and consisting in an overgrowth of connective tissue, at first containing cells, but finally losing these and becoming a more or less dense reticulum which replaces nerve-elements. The lesion extends from the periphery to the centre, the meninges always being simultaneously affected, but it is characteristic that the induration affects the whole thickness of the cord. In chronic myelitis the lesion is more partial; it may also be more diffused.

In acute transverse myelitis the nerve-elements are primarily affected, the cord is softened instead of being indurated, and may even become diffluent like cream, although this last change is said to take place only after death.

The initial step of the lesion is a swelling of the nerve-elements, both of the ganglia-cells and of the cylinder-axes of the tubes.[1] The processes of the cells break off, the nucleus becomes indistinct, the protoplasma granular, finally the swollen and shapeless body begins to shrink, atrophies, and at last disappears. Correlatively, the myeline in the tubes segments, then wastes; tubes sometimes persist for a long time whose cylinder-axis is surrounded by a narrow rim of myeline. These are the tubes which are most liable to regenerate, and that without having traversed all phases of degeneration. In the focus of softening, besides the swollen or wasted tubes and nerve-cells, appear isolated cylinder-axes, swollen and fragmented, myeline drops, the spider-cells of Deiters,—*i.e.*, neuroglia-cells whose processes have become distinct through inflammatory swelling,—and the granular corpuscles, which represent the ultimate modification of the neuroglia-cells. The tissue is also infiltrated with leucocytes, blood-corpuscles, and small angular and fusiform cells, so that in patches all traces of nerve-elements have disappeared, and under a low power of the microscope the patch has a granular appearance. The blood-vessels are dilated and stuffed with blood-corpuscles, and many of them greatly thickened by cellular infiltration of the perivascular sheath. The lumen is sometimes encroached upon, and the vessel obliterated, a change which facilitates necrosis. In syphilitic myelitis the characteristic thickening of the intima of the blood-vessels may be expected.

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[1] This is beautifully shown in some of Bramwell's illustrations from nature, *loc. cit.*

The secondary ascending and descending degenerations of the cord are the same for ordinary softening as for compression myelitis. But, as the acute form of the disease in children is more frequent than the chronic, time is often lacking for the production of these secondary lesions.

Meningitis is rarely focal or limited to a single segment of the cord, except in the pachymeningitis of vertebral caries. Even when it has been caused by a blow falling in one place, the inflammation, though most intense at the seat of direct injury, usually extends over the greater part of the spinal canal. It is supposed that the cerebro-spinal fluid becomes a vehicle for readily transmitting irritamenta from any given focus of inflammation. In acute cases there is intense hyperæmia of the meninges and the external and internal surface of the dura, usually associated with similar hyperæmia of the pia, turbid flocculent serum in the cavity of the arachnoid, opacities and thickening of the arachnoid from cellular infiltration, and adhesions of the pia, on the one side to the arachnoid, on the other to the nerve-tissue. The arachnoid cavity sometimes contains pus, and there is sometimes also a purulent infiltration of the meshes of the pia.[1]

Chronic meningitis is indicated by the diffused or localized thickening, opacity, and adhesions of the membranes. The pachymeningitis secondary to caries is characterized by an abundant development of inflammatory granulation-tissue, ultimately pseudo-membranes.

Meningeal hemorrhage is unequivocally demonstrated by the presence of effused blood in the spinal canal or the cavity of the arachnoid. If some time has elapsed since the occurrence of the accident, the fluid portion of the blood will have been absorbed; only the blood-clot or masses of hæmatin crystals or fibrinous exudation will remain. If the fibrin become organized into a pseudo-membrane, it will be difficult from inspection of the lesion alone to distinguish between a pachymeningitis and a hemorrhage. But taken together with the clinical history the lesions may establish the correct diagnosis.

Hemorrhage into the cord is more difficult to distinguish from myelitis. When a softened portion of the cord is red, capillary hemorrhages at least have occurred, and are indicated by the presence in the softened focus of hæmatin and blood-corpuscles. This is the red softening, or "hemorrhagic myelitis."

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[1] In epidemic cerebro-spinal fever the pia-infiltration is gelatinous, excessively abundant, and produced with great rapidity.

PROGNOSIS AND NATURE OF THE DISEASE.

The prognosis of compression meningo-myelitis has been already discussed. This is the form which is at once by far the most frequent in childhood and also the most favorable. In the idiopathic and traumatic varieties of disease the danger is usually proportioned to the violence of the initial symptoms. But an apparently mild case may be really insidious, and then far more dangerous. This is true for tubercular meningitis, and, if untreated, for syphilitic myelitis.

Analysis of the anatomical lesions of common myelitis shows that this is essentially an acute necrosis of nerve-tissue. It can be experimentally imitated by ligating the abdominal aorta and so cutting off the blood-supply from the cord for a certain number of hours.[1] It is evident, therefore, that whatever interferes with the nutrition of the nerve-tissue of the cord is capable of producing myelitis. Pressure seems to act not by directly cutting off the blood-supply, but by exciting the growth of the interstitial tissue, in which, through pressure, the nerve-elements become stifled. Falls and other traumatisms, which cause great commotion of the spine and violent vibrations of the nerve-tissue, seem able suddenly and profoundly to impair the capacity of its nerve-elements to appropriate nutrition. Infectious diseases act in an obvious manner directly to poison nerve-elements. Cold, exposure, and fatigue seem primarily to affect the meninges; and the nutrition of the cord is impaired when its blood-supply is perturbed through paralytic dilatation of the nutritive blood-vessels of the pia mater.

It is possible that in children the cord may be directly exhausted by excessive fatigue, as in adults by sexual excesses. The lumbar portion of the cord is the latest part of the cerebro-spinal axis to develop, and during immaturity its functions should not be excessively exerted.

In the prognosis of spinal-cord diseases is involved not only the question of danger to life, but also that of recovery from paralysis and deformity. This has been already discussed in speaking of compression myelitis from caries, where the prognosis is, on the whole, good. It has been mentioned in this connection that the nerve-tubes of the spinal column, like those of the spinal nerves, are capable of regeneration. They are less likely to be so in common transverse myelitis, probably for the reason that in this the nerve-elements themselves, and not the neuroglia, are primarily, and thus more extensively, affected. Where all hope of regenerating nerve-tubes themselves is lost, it may still be possible to stimulate the functions of the motor path which lie in adjacent tracts and are relatively uninjured. But there are as yet no statistics which may inform us in regard to this possibility.

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[1] These experiments have recently been repeated by Herter. See Philadelphia Medical News, March, 1889, A Study of Experimental Myelitis.

TREATMENT.

The treatment of meningo-myelitis varies with the cause. In that due to vertebral caries treatment of the bone-disease is of primary importance. It is curious to see the stress laid by many neurologists upon cauterization of the back, after the method of Percival Pott, while no attempt is made to immobilize the diseased spinal column. It is perhaps equally curious to notice the records of cures effected by this method, associated only with rest in the recumbent position and tonics. It is also remarkable that cauterization should have an effect upon compression myelitis which its partisans, as Charcot, assert is not to be expected for common transverse myelitis. It is to be noted, however, that both the medullary lesion itself and the bone-disease upon which it depends are affections of connective tissue,—here neuroglia, there osseous. It is to be anticipated, therefore, that its inflammation would be more easily controlled by counter-irritation, which tends to divert nutritive currents to the connective tissue of the skin. The focal myelitis which begins in the parenchymatous elements of the nerve-tissue, and which consists essentially in their necrosis, will not yield to similar treatment, because it is an essentially different disease. If cauterization has cured the paraplegia of Pott's disease without the aid of immobilizing apparatus, similar cures are on record due to the use of the apparatus without the burning. In American practice the immobilizing brace would always be applied for the treatment of the caries, and is expected not only to facilitate the cure of the bone-disease, but also to arrest the occurrence of paraplegia.

If, nevertheless, this appear in a child already wearing a brace or plaster jacket, the weight of testimony is in favor of cauterizing the back at the level of the bone-disease, with the hope of affecting by counter-irritation the pachymeningitis.

From this point the treatment of the paraplegia is identical with the treatment of the Pott's disease,—exposure to air and light in a wheel-chair, cod-liver oil, the hypophosphites, and abundance of food, especially milk. This is valuable, among other reasons, on account of the large admixture of carbohydrates it contains in association with the nitrogenous constituents.

In acute or subacute meningo-myelitis ice should be applied to the spine promptly, and nearly continuously, unless the patient complain of chilliness or discomfort from the use of it. The ice does not contra-indicate another remedy,—namely, extremely hot baths. Intense heat has been found very useful in tetanus, and a case of cure is on record where the patient was plunged for several days up to his neck in a smoking manure-heap. Halter[1] has recently revived this recommendation for tetanus. Whatever may be proved to allay the functional irritation of the spinal cord when due to disease of the medulla may be expected to have efficacy in the irritation resulting from disease of the cord-membranes. The temperature and the duration of the bath must be regulated by the sensations of the patient, and by their influence upon his restlessness and pain.[1]

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[1] Berliner Klinische Wochenschrift, 1888.

At the same stage of the disease at which ice to the spine and heat to the periphery are required, it is often desirable to apply leeches along the spinal column. They will relieve pain, and are to be repeated if this return after ten or twelve hours.

Ergot is given, in the hope of directly contracting the dilated blood-vessels. Large doses are required,—two or three drachms daily of the fluid extract, or five or six grains of the solid extract in capsules, although this is a less powerful method. The above is the dosage for adults, and it may be graded for children in proportion to their age,—for infants five drops of the fluid extract every hour or every two hours. The detestable taste of the drug is likely to nauseate them less than older patients, and the dilated condition of the blood-vessels of the nerve-centres seems to establish a tolerance. If necessary, the fluid extract can be given by the rectum, at least part of the time.

Tincture of belladonna, on the recommendation of Brown-Séquard, has also been largely used for the same purpose as ergot,—namely, to contract the blood-vessels of the inflamed tissues. But there is much less proof of its efficacy in this respect. An overdose, as shown in rabbits poisoned by belladonna, produces excessive venous congestion of the spinal membranes. It is best given in small doses frequently repeated: thus, for children five drops of the tincture every hour.

To quiet the excessive pains of meningitis, opium is often required, and seems to be free from danger. Opium is the basis of treatment in cerebro-spinal fever.[2] Its effect in slackening the oxidations of tissue, and especially of nervous tissue,[3] may possibly tend to increase the resistance of such tissue to the disintegrating influences of acute inflammation. Thus, while the palliative influence of opium is principally required for meningitis, it is permitted from the analogies with the epidemic disease to hope for some curative influence of opium upon inflammation of the cord itself. For this reason, opium is to be preferred theoretically to chloral; yet chloral may often be used symptomatically, to quiet restlessness or procure sleep.

Iodide of potassium has been given in the second and third stages of meningitis, with the intention of facilitating the reabsorption of inflammatory exudation. It has also, in large doses,—from sixty to one hundred and fifty grains a day for adults,—been strikingly beneficial in syphilitic disease of the cord. Its use is then to be combined with that of mercurial inunctions. The empirical use of the iodide in many forms of cerebro-spinal disease of undefined causation may perhaps have often been successful on account of the really syphilitic nature of the case.[1] But iodide of potassium has also been used with apparent success in cerebro-spinal fever,[2] and, as in the case of opium, its influence must be symptomatic. Such success justifies its employment in common meningitis and myelitis. The experiments of Benedikt upon frogs with iodide of potassium would indicate that this drug has a special action upon the cervical region of the spinal cord, with the respiratory (and cardiac) centres contained in it. These, in a sufficient dose, it first irritates and then paralyzes.[3]

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[1] Halter thinks that heat is useful in tetanus because tending to destroy its pathogenic microbe. In non-specific meningitis the object of the bath is rather to determine blood to the surface of the body, and to act upon the vast surface of cutaneous nerves. Still, it is to be remembered that the real origin of many cases of meningitis is entirely obscure, that the meninges are always liable to be affected in infectious diseases, and many cases of apparently spontaneous meningitis may yet be shown to depend on bacterial infection. Finally, diseases which begin with a chill frequently do well under treatment by heat.

[2] Stillé, Epidemic Cerebro-Spinal Meningitis, 1867, p. 158. Large doses are advocated as alone efficacious. They are needed early in the attack. As the opium can only act symptomatically in this disease, analogy would seem to justify its employment in other forms of meningo-myelitis.

[3] Nothnagel and Rossbach, Handbuch der Arzneimittel.

From such experiments no definite or precise view could at present be framed of the therapeutic action of iodide of potassium; but they at least indicate an elective tendency of the drug towards the spinal centres.

If there be reason to believe that a spinal meningitis is due to rheumatism, as shown by its alternation with arthritis in the course of the same illness, or else in the same patient, specific treatment by salicylates must be instituted. This form of meningitis is usually much more favorable than the others, and it is important, therefore, to examine scrupulously for its indications, as the history of the patient, high fever, strongly acid urine, profuse acid sweats, freedom from cerebral symptoms.[4]

If meningitis assume a chronic form, yet there be reason to hope, from its localization and from the improvement in the general health of the patient, that it is not tubercular, great reliance is to be placed on counter-irritation. Narrow strips of blistering plaster may be applied over the spine, so as to maintain a permanent vesication; or this may be applied at intervals of a week, and during the healing of the blistered surface hydro-therapeutic treatment may be employed. The cold pack, prolonged so as to induce sweating, and the alternate hot and cold douches, are of real value in dissipating the hyperæmia of chronic inflammation.

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[1] Behin, Schmidt's Jahrb., 1865, Bd. cxxvii. S. 16, relates three adult cases, the last that of a woman with paraplegia and sphincter-paralysis coming on after confinement. She had been treated for two months without success, when the iodide was first given, in doses of from one to five grammes a day. Improvement in ten days, beginning ability to walk in four weeks, and recovery in eight weeks. The sphincter-paralysis in this case excludes the diagnosis of neuritis.

[2] Wunderlich, Arch. für Heilk., 1864, Bd. v., and Schmidt's Jahrb., 1865, Bd. cxxvi. S. 38. Three cases out of six were treated by iodide in large doses, and recovered; the other three died.

[3] Ueber die Wirkung des Iod und Iodkalium auf das Nerven-System, Benedikt, Schmidt's Jahrbücher, 1861, Bd. cxv. S. 284.

[4] Rheumatic meningitis of the cerebrum is more frequent than that of the cord, and its clinical identity is better established; but it does not seem to complicate the spinal rheumatic meningitis.

In chronic inflammation the iodides and mercurials, especially the bichloride of mercury in cinchona,[1] have long been held to possess a specific influence in promoting the disintegration and reabsorption of plastic exudations. The experiments of Hoegges,[2] showing the rapid combination of iodine with the albuminous molecule, lead the author to infer that by this means the lowly-organized tissue of inflammatory exudation may become starved out, and hence reabsorption be effected. In syphilitic infection this action may possibly be also exerted upon the microbe of the constitutional disease.[3]

Local massage is, with the views of the present day, a theoretically appropriate remedy for inflammatory exudation situated much less profoundly than many for which it is now being tried. No cases of its use in children are, so far, known to the writer ; but it is most rationally indicated, even in cases where there is little or no paralysis, but where the symptoms of irritation and spastic contractures persist.

The employment of electricity in meningo-myelitis is one of the most disputed questions of therapeutics. The passage of the constant current over the spine—*i.e.*, according to the experiments of Legros and Onimus, through the cord—is alleged to facilitate the retrogression of a myelitic process, and to favor the reabsorption of meningeal exudation.[4] It is advised to pass the current for a short time only,—say, five minutes,—but the direction of the current is of little consequence.[5]

There is much more agreement on the value of electricity directly applied to paralyzed nerves and muscles than on that of the application of the current to the spine. Galvanism does seem to have the power, to a certain extent, to retard and limit the degeneration and to facilitate the regeneration of nerves. The clinical history of patients treated by galvanism is undoubtedly full of illusions, from the tendency to spontaneous recovery in curable cases, and from the probable influence on the cord of the treatment directly applied to it. Still, galvanism to the paralyzed limbs should not be omitted from the treatment, so soon as the acute symptoms have completely subsided. Great care is required to avoid fatiguing the paralyzed nerves or muscles by the galvanic stimulus. The patient must be carefully watched, and if increased irritation or weakness follow the treatment it must be interrupted, or be lessened in frequency or severity or duration. The faradic current must not be tried until after toleration for the galvanic current has been well established. It is useless so long as the reaction of degeneration persists; and it is always useless over the cord, to which it does not penetrate.

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[1] One-sixtieth grain of the salt three times a day for children under ten.

[2] Arch. f. Exp. Pharm., 1878, Bd. x.

[3] The reports of Seguin on the beneficial influence of iodide of potassium (one hundred and fifty to one hundred and eighty grains) upon rapidly-advancing syphilitic myelitis all refer to adults.

[4] Erb, Ziemssen's Handbuch.

[5] The marked difference in the action of the poles, which are external to the cord, does not imply any corresponding difference in action at the intrapolar space occupied by the diseased tissues.

Strychnine, introduced by the famous recommendation of Magendie, seems to be rarely well tolerated by children, even in flaccid paralysis.

When this form has lasted some weeks and seems to have become stationary, it may be desirable to procure supporting apparatus, by which the child may be enabled to move about in a sitting position. A brace should remove the weight of the body from the spine to the rigid uprights supported on the pelvis. With this brace on, the child should be placed in a wheel-chair, that he may propel himself by his arms. In many cases the paralysis which persists after an attack of meningo-myelitis is functional, and quite disproportioned to the organic residues of the previous inflammation. Such functional paralysis requires the stimulus of voluntary exertion, while at the same time the patient is protected from any unnecessary exertion in sustaining weight.[1] It is needless to add that in those cases which have become chronic the most roborant regimen and treatment are essential.

From what precedes, it is evident that no radical difference exists in the treatment of meningitis and that of myelitis. The treatment for the first constitutes the treatment for the incipient stage of the second; and the treatment of myelitis represents the therapeutics of the advanced stage of meningitis, in which, indeed, as a rule, some degree of myelitis also exists and is the proximate cause of the symptoms. The special indications for the treatment of the compression myelitis of caries have been mentioned. In addition to these, and if such special treatment prove insufficient, the case is to be treated on general principles, as has just been detailed.

The treatment of intraspinal hemorrhage is negative and expectant. Evidently nothing can be done to cure a hemorrhage which has taken place. The indications for treatment are to diminish the hyperæmia in which the hemorrhage has originated, or which is collateral to a vascular thrombosis that has been the indirect cause. This indication practically means, in the majority of non-traumatic cases, treatment of the entire antecedent disease to which the hemorrhage is secondary. Immediately after the occurrence of symptoms of hemorrhage the patient should be placed in bed, in the prone position (as far as possible), with an ice-bag attached the whole length of the spine, and absolute rest enforced. Ergot is then given, as in acute myelitis. The treatment of subsequent symptoms is that of the meningitis or myelitis consecutive to the hemorrhage, or in the midst of which the latter may have occurred.

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[1] In the hysterical paralyses which are so easily confounded with those dependent on chronic meningitis (see *supra*, Case XVI., from Seeligmüller), this supporting method is absolutely essential. An adult woman under the writer's care, confined to bed and a recumbent position for three years, was enabled to walk in a few weeks by means of a Taylor brace and Shaffer chin-piece.

POLIOMYELITIS ANTERIOR.

By WHARTON SINKLER, M.D.

Synonymes.—Infantile paralysis; Essential paralysis of children; Acute atrophic paralysis; Infantile spinal paralysis; Atrophic paralysis; Regressive paralysis; Tephromyelitis.

Under these various titles has the disease of which we are about to treat been described. It has been very commonly called infantile paralysis, and, as a rule, this term is understood to mean the form of paralysis under consideration. The name is undesirable, however, because it does not designate what the character of the disease is, for we may have various forms and varieties of paralysis in children, all of which would properly come under the generic term "infantile paralysis." Neither does the name "infantile spinal paralysis," proposed by Dr. Mary P. Jacobi, describe it more fully, as spinal paralysis in children may arise from pressure myelitis in Pott's disease, from tumors, from spinal meningitis, or from other causes. Acute anterior poliomyelitis describes the pathology of the disease, and therefore the name seems preferable.

Definition.—Poliomyelitis anterior is an affection in which loss of power in the voluntary muscles occurs suddenly or in the course of a few hours or days. It is unaccompanied by sensory changes. After a few days the paralysis leaves some of the parts originally involved, but the others undergo wasting and remain powerless. The paralyzed muscles become atrophied and relaxed, and the shortening of their opponents causes permanent deformities in the affected limbs. The disease is not peculiar to children, although a large proportion of the cases occur under five years of age, and in the infantile form there are certain distinct features which separate it somewhat from the adult type.

History.—The first writer who described this disease was Jacob von Heine, who wrote a monograph upon the subject in 1840, detailing the atrophies and deformities which are characteristic of the affection. Rilliet and Barthez also treat of it in their work on Diseases of Children, published in 1853. Duchenne, in his treatise on electricity, discusses the disease at length, and asserts his belief that its origin is spinal, although no post-mortem examination had at the time been made to confirm his view. This author applied the name "acute fatty atrophic paralysis," indicating thereby the muscular changes which take place. It was not until 1863 that distinct alterations in the spinal cord were observed first by Cornil, and in 1865 Prévost and Vulpian located the essential anatomical lesion in the anterior horns of gray matter in the cord. Lockhart Clarke confirmed these observations in 1868. Charcot and Joffroy in 1870 reported a case in which with great care and thoroughness they pointed out the degeneration in the anterior gray horns. Since then numerous autopsies have been reported in other observations in Germany, France, England, and this country, and in all of them there has been striking uniformity in the lesions found.

Varieties.—There are two forms of the disease,—the acute and the subacute or chronic. The former is by far the most frequently met with in children.

Etiology.—As before remarked, the disease may occur at any period of life, but is much oftener found during the first three years than at any other time. According to Gowers,[1] of all the cases under ten years three-fifths occur in the first two years of life and four-fifths in the first three years. It is infrequent during the first year, but it may occur at that time, and it occasionally comes on soon after birth.

There is little doubt that a considerable number of cases are congenital.[2] These cases seldom come under the observation of the physician, for they are usually taken to an orthopædic surgeon for treatment of the club-foot which is the prominent feature. Dr. T. G. Morton[3] believes that most cases of congenital club-foot are the result of an intra-uterine paralysis, for in all when examined there is palsy of some of the muscles in the limbs affected.

The following case is a good illustration of congenital club-foot from an intra-uterine spinal paralysis:

Edward P. H., aged three and three-fourths years. Seen March 17, 1890. When his mother was four or five months advanced in pregnancy, she saw a man "who walked with his feet extended in the air," and she was much affected thereby. The child was born with club-foot. On examination he was found to have pure valgus of the right foot. The plantar arch is absent, giving rise to flat-foot.

The right calf is seven and a half inches, the left eight inches; the right thigh is nine and a half inches; the left nine inches. The right leg is three-eighths of an inch shorter than the left leg, and the right foot three-eighths of an inch shorter than its fellow. Electrical examination shows that the muscles all respond to the faradic current except the posterior tibial.

One of Duchenne's cases was attacked as early as twelve days after birth.

Bramwell[4] records a case in which an attack of acute anterior poliomyelitis appears to have developed when the child was three weeks old. The patient, however, was not seen until she was twenty-eight years of age, and the evidence of the age at which the attack began depended on her having remembered that she heard an aunt say that the paralysis occurred at the age of three weeks.

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[1] Diseases of the Nervous System, p. 254.

[2] Gowers (op. cit., p. 254) does not believe this, and says that there is no valid evidence of the intra-uterine development of the paralysis.

[3] Philadelphia Medical News, July 12, 1890.

[4] Studies in Clinical Medicine, vol. i. No. 1, p. 11.

Of three hundred and fifty cases which I have classified from my own note-books and those of the Philadelphia Infirmary for Nervous Diseases, the age of the patient at the onset of the attack is noted in three hundred and thirty-five cases. Of these, fifty-six cases occurred under one year: one patient was but six weeks of age when attacked, two were three months old, four were four months, two were five months, eleven were six months, and thirty-two were between six months and one year. Between one and two years of age one hundred and thirty-four of the patients were attacked, between two and three years there were seventy-seven cases, between three and four years thirty-eight cases, between four and five years twelve cases, and between five and eleven years but eighteen cases. Two hundred and forty-seven of the three hundred and thirty-five cases occurred under three years, and the average age of the attack in two hundred and forty-four cases was two years, one month, and two and one-fifth days.

Table showing the Age at the Time of the Attack in Two Hundred and Forty-Four Cases.

MONTHS.	YEARS.										
	0	1	2	3	4	5	6	7	8	9	10
0	2	19	27	19	8	0	2	4	0	2	1
1	0	11	2	1	0	0	0	0	0	0	0
2	0	6	1	1	1	0	0	0	0	0	0
3	2	9	8	0	0	0	0	0	0	0	0
4	4	5	1	1	0	0	0	0	0	0	0
5	2	2	0	2	0	0	0	0	0	0	0
6	11	23	9	4	0	1	1	2	0	0	0
7	3	1	0	0	0	0	0	0	0	0	0
8	4	6	1	0	0	0	0	0	0	0	0
9	6	1	4	0	0	0	0	0	0	0	0
10	6	5	1	0	0	1	0	0	0	1	0
11	4	4	1	1	0	0	0	0	0	0	0
Totals.	44	92	55	29	9	2	3	6	0	3	1

Average, 2 years, 1 month, 2.21 days.

It is stated by some writers that boys are more frequently attacked than girls, but from my own cases it does not appear that the difference is conspicuous. Of three hundred and forty-five of the cases to which I refer above, one hundred and eighty-four were boys, and one hundred and sixty-one were girls. In adults, however, the disease is probably more frequent in males than in females.

The hereditary influence is not great: occasionally two cases are met with in the same family, but this is rare. In the cases which I have examined there were three instances in which a brother and a sister suffered from the disease, and in one case a cousin also had anterior poliomyelitis.

In one of the instances in which a brother and a sister were both attacked, we are reminded of the epidemic reported by Cordier, of which I shall give an account later. The cases were as follows:

Ralph S., aged three and a half years, and his sister, two years older, were healthy until July, 1886. The girl was taken sick on July 2, with fever and pain all over. The temperature was 102° F. After three or four days she got up, but was lame and walked stiffly and with a limp for a few days, after which she recovered fully. The boy was taken down two or three days later, with fever and general soreness. He cried with the pain in his limbs. He lay on a lounge for three or four days, and when he got up he could not walk or stand. Both legs were equally affected at first, but in a few days the right leg began to improve and soon recovered entirely. When I examined him, ten months after the attack, he was able to walk well with the aid of an apparatus on the left leg. He could make every movement with his leg, but it was weak and greatly atrophied. The right thigh measured ten and a half inches, while the left was but eight and a half inches. The muscles of both legs responded to the slowly-interrupted faradic current, but in the left it required a stronger current.

In many of the cases there was a history of nervous disease in the family; in two instances a sister had chorea. Duchenne reports an instance of twin brothers who were simultaneously attacked with poliomyelitis anterior after an attack of measles, and Seeligmüller reported three brothers in the same family who were affected with infantile paralysis.

The season of the year is a most conspicuous factor in the causation of this disease in children,—a fact to which I called attention some years ago.[1]

Of the three hundred and fifty cases above quoted, in two hundred and seventy the season in which the attack took place was recorded: of these there were two hundred and thirteen, or 78.8 per cent., attacked in the hot months of the year,—that is, from May to September, inclusive:

Spring,	27 cases,—viz.,	March	9 cases.
		April	4 "
		May	10 "
		Month not stated . .	4 "
Summer,	174 " "	June	27 "
		July	52 "
		August	65 "
		Month not stated . .	30 "
Autumn,	59 " "	September	29 "
		October	25 "
		November	4 "
		Month not stated . .	1 case.
Winter,	10 " "	December	3 cases.
		January	4 "
		February	3 "
		Total	270 cases.

The accompanying diagram exhibits the relation between the temperature and the onset of the attack of poliomyelitis in children. I have been enabled, through the courtesy of the Chief Signal Officer at Washington, to give a table of the mean maximum temperature, the relative humidity, and the actual barometric pressures at Philadelphia covering the period of time since accurate records have been kept at the Signal Office. There will also

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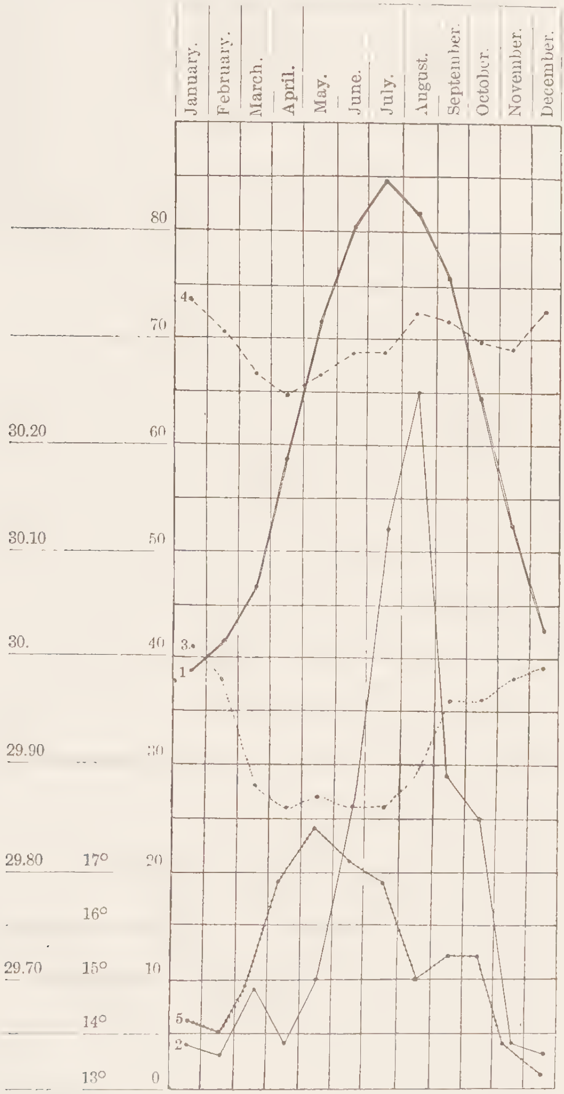
[1] American Journal of the Medical Sciences, April, 1875.



1. Mean maximum temperature for sixteen years, 1874 to 1890; 2, diagram showing month of onset of two hundred and thirty-five cases of poliomyelitis, 1872 to 1890; 3, actual average barometric pressure for seventeen years, 1873 to 1890; 4, average relative humidity for nineteen years, 1871 to 1890; 5, mean daily range of thermometer for ten years, 1876 to 1885, inclusive.

be found a curve giving the mean thermometer—that is, the average daily range of temperature—for ten years. This has been taken from a diagram in a paper by Dr. Morris J. Lewis on "The Seasonal Relations of Chorea and Rheumatism."[1] My friend Dr. H. W. Cattell has kindly assisted me in preparing these tables, as well as the other statistical work in this paper.

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[1] Phila. Med. News, Nov. 13, 1886.

On examining the diagram, it will be seen at a glance that the cases of infantile palsy increase in number as the temperature rises. The highest point reached by the thermometer was in July, in which month the maximum temperature was 84.6° F. The maximum number of cases occurs in August, when the temperature was but little lower than in July. The greater number of cases in August may be explained in two ways,—first, because the intense heat of July has prostrated the children to such an extent that they more readily succumb to the spells of heat which follow; and, secondly, in August the relative humidity is greater than in July, the figures being 72.1 and 68.6 respectively. By the mean relative humidity is meant the *mean percentage*, and not the *mean actual amount*, of moisture which can be held in solution at the mean temperature of each month,—the amount representing complete saturation being indicated by 100. The degree of humidity does not show any influence on the number of cases of infantile paralysis unless associated with heat. For example, the relative humidity is highest in the month of January, when the number of cases is at a minimum. Nor does the range of temperature appear to have any bearing on the disease. The greatest daily range was in May, in which month the number of cases was comparatively small.

Gowers states that of his cases two-thirds were attacked between June and September. The same fact has been noted in poliomyelitis in adults. Barlow[1] has also made the observation that a large proportion of cases of this disease occur in the summer.

The influence of heat is strikingly shown in a case reported by Dyce Duckworth:[2]

A child two and a half years of age, after exposure to great heat on a steamboat-landing, became paralyzed in all four limbs, but the paralysis was subsequently confined to the lower extremities. With the onset of the paralysis the patient became delirious and suffered from anæsthesia and temporary paralysis of the sphincters. The paralyzed muscles wasted rapidly and lost faradic contractility. Treatment was begun by faradization one month from the beginning of the attack, and recovery was complete three months later.

One of my cases was attacked after long exposure to the sun:

Maggie O'D. was well until nine years of age. In August, 1873, she was one day exposed very much to the sun. That night she was taken sick. She had no delirium, and but slight headache. On the fifth day it was found that there was general loss of power. There was no loss of sensation, and no trouble with the bladder or bowels. In four weeks she began to move the arms, and in six weeks she began to use the left leg. When seen at the age of eleven, the right leg was atrophied, the thigh being two inches smaller than the left, and the calf one and three-eighths inches smaller than its fellow. The knee was contracted, and there was slight pes equinus.

Exposure to cold and chilling of the body undoubtedly have a marked influence upon the production of these cases. In several of my patients it was stated that the child had been sitting on a stone step prior to the attack, or that it had become chilled after overheating. Gowers has met with two cases in which the disease followed sitting on damp grass.

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[1] Regressive Paralysis, William H. Barlow, M.D., Manchester, 1878.

[2] Lancet, 1877.

An interesting example of the influence of hot weather in the causation of this disease is an epidemic of infantile paralysis which is reported by Cordier.[1] This author saw thirteen cases of the disease during the months of June and July, 1885, in one locality of a district containing from fourteen hundred to fifteen hundred inhabitants. The age of the patients was from one to thirty months, boys and girls being attacked in about equal numbers and with equal severity. Almost all of them were in good health previous to their attack by this disease, to which four succumbed on the third day. Those who survived showed definite and characteristic lesions of the affection. In almost every case the health of the child was good before the onset, which was without premonitory symptoms.

The fever was variable in intensity, the extent of it being apparently governed by the gravity of the disease or the extent of the lesions in the cord. Convulsions occurred in about one-half of the cases; profuse sweating was also observed, and was present in all except the four fatal cases. The paralysis was present in several cases after the second or third day, either in the lower extremities alone or in all four extremities at the same time.

In some cases the neck-muscles were so paralyzed as not to be able to sustain the weight of the head; in other cases the extent of the paralysis was so great that the children were unable to nurse or to cry. In these severe cases the lesions were not limited to the cord, but extended to the bulb, the gray nuclei of the ventricles being probably involved in fatal cases. In the four fatal cases death came at the end of the third day, the patients being the youngest of the series. In those which recovered, paralysis did not disappear with equal rapidity in all: in some there were improvement and gradual disappearance of the paralysis, which finally remained fixed in a single muscle or group of muscles, while in other cases atrophy and deformities remained.

The author's opinion in regard to these cases is that they constituted an epidemic identical with epidemics of diseases whose infectious character is well recognized. In regard to the infective agent in this disease, it is believed that it did not enter the organism with the food or drink, nor by inoculation, but with the air that was inspired. The propagation of the disease was traced from one patient to another according to the dates of invasion and the relations which were sustained between the different families that suffered. The contagious character of the disease was believed by Cordier to have been demonstrated by the fact that one child was attacked thirty-six hours after a visit to another who was already suffering from the disease. In two other cases a boy and his sister were exposed, and the first evidences of the disease were apparent only eight or ten hours afterwards.

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[1] Lyon Médical, January and February, 1888.

While these cases are very interesting, the facts are not strong enough to warrant us in accepting the author's theory as to the infectious character of the affection: moreover, he does not report any post-mortem appearances.

The symptoms in this epidemic differ considerably in character from those of the usual form of anterior poliomyelitis. The frequency of convulsions and the fatality are unusual in this disease. These facts do not hinder us from accepting the opinion that these were cases of poliomyelitis, for it is well known that in all epidemics the ordinary types of disease change, being more severe and more fatal.

Not unfrequently over-exercise in a young child has preceded an attack of poliomyelitis. In several cases which have come under my notice there has been a history of a long walk, after which the child seemed much fatigued and within twenty-four hours was attacked with this disease. The following case was attacked after over-exertion:

Miss J. S., aged seventeen, an only child, has always had good health up to the present trouble. During the whole summer she was at the sea-shore, and took a great deal of exercise in walking, bathing, and dancing. In the latter part of August she went up to the mountains, and soon after getting there, one evening after a day of unusual fatigue, she had some fever which continued during the night. The next morning she got up, but found she was scarcely able to walk; would have fallen had she not had assistance, and was helped back into bed. Her legs felt a little numb. The loss of power was not complete at first, but in two or three days there was paralysis of both lower extremities. Then the arms began to get weak,—first the left, then the right; and at this time there was considerable pain in the right arm. The right arm was never completely paralyzed. For one day the loss of power in this arm was considerable, but it soon recovered. When I saw her, September 20, a little over three weeks after the onset of the attack, there was complete loss of power in both legs from the hips down. Sensation was unaffected; knee-jerk was absent in both legs; there was no plantar reflex and no abdominal reflex, but the epigastric reflex was present. The right arm was unaffected, as was the left arm except the shoulder-muscles, which were paralyzed and she was unable to lift the arm. Already considerable atrophy had taken place, and the muscles did not respond to a strong faradic current and responded but feebly to the galvanic current.

In a great many cases which are brought to a physician, the friends are inclined to trace the trouble to a fall; but in most instances the interval between the fall and the onset of the attack is so great that there can be no relation between the injury and the disease. Occasionally the attack does follow a fall so closely that there are grounds for believing that the injury was its cause. In two of my cases the paralysis came on the day after a fall, and in three cases two days later. In one case the child had a fall in which the clavicle was broken, and the attack of poliomyelitis came on three weeks afterwards; in another case the child fell out of bed, and within a few days afterwards the paralytic symptoms were observed.

From the fact that the disease occurs most frequently during the teething-period, it seems likely that there is some influence produced by dentition. While we do not believe that myelitis can be caused by dentition, still while teething the nervous system is particularly susceptible to impressions of all kinds, and it is well known how frequently reflex convulsions and other disturbances occur at this time. The period of second dentition is not specially liable to the onset of this affection.

Long-continued ill health seems to have little influence on the production of the disease. Most of the children are in excellent health at the time of its onset, but a number may have been suffering from summer complaint. Occasionally it follows convalescence from some acute disease,—for instance, one of the exanthems.

Of the three hundred and fifty cases above referred to, in twenty-eight the attack occurred during convalescence from some acute disease.

Among the diseases noted are measles, seven cases; rheumatism, one case; scarlet fever, four cases; whooping-cough, two; cholera infantum, twelve; and pneumonia, one case.

Climate exerts little or no influence on the production of the disease, and as many cases occur in the country as in the city in proportion to the population. The colored race is not as exempt from this disease as it is from chorea.

Symptoms of the Acute Form.—As a rule, the patient, who has been in good health, is seized with fever and vomiting and sometimes diarrhœa. He is restless and fretful, and cries when moved or handled as if there were backache and hyperæsthesia of the surface. After a few hours, quite often on the morning following the beginning of the attack, the child is found to be paralyzed in one or more limbs. The fever and general disturbance last for three or four days. A few days after the subsidence of the fever and general soreness, one or more of the paralyzed limbs begin to improve. If both legs and arms have been paralyzed at the onset, the arms are the first to recover. By the end of the second week one or both arms can be used fairly well. Then improvement begins in the legs. Some of the muscles regain power, while others remain stationary. After the fourth week the progress is slow, and it can generally be determined now which muscles are doomed to be permanently palsied. There is no loss of sensation, and the bladder is unaffected. Soon the paralyzed limbs begin to atrophy, the muscles shrink, and the skin and subcutaneous cellular tissue become adherent. The surface looks blue and mottled, and the temperature is lowered. No bed-sores occur, however. If an electrical examination is made, it is found that after a few days there is no response to the faradic current, but the muscles still contract to the galvanic current. The muscles do not become contracted, as is seen in cerebral paralysis, but deformities of the feet occur from shortening of the paralyzed muscles, owing to the position in which the foot hangs, or from overaction of the opponent muscles.

The extent of the paralysis varies, but it is seldom that all the muscles of both legs remain completely powerless. In some cases only certain groups of muscles in one or both limbs remain paralyzed, and the patient then soon begins to walk with support. The following case illustrates the features of a typical case of the acute form of this disease:

Joseph R. L., aged four years, was seen by me November 18, 1874. He is an only child. The father died of kidney-disease, but the mother is living and healthy. He is a robust, healthy-looking boy, with light hair, blue eyes, and sound teeth. Was walking when fifteen months old, and was in good health, except that he was teething. One day in July he was seized with vomiting and fever, which lasted three or four days; at the end of that time he was found to be completely paralyzed in both legs. He had no convulsions, and there was no loss of power in the bladder or rectum. During the time that the fever lasted he seemed to be unconscious, but screamed when lifted or handled. In two weeks the right leg regained entire strength, but the left did not improve, and for a month he was weak and feeble.

Present State.—There is no difference in the length of the legs, but the left foot is one-quarter inch shorter than the right. The right leg is strong and well developed, but the left is small, flabby, and cold. There is absolute loss of power in all the muscles from the hip down, except in the flexors of the leg, in which there is slight movement. He can walk without assistance, but the leg is simply swung around, and when he bears any weight upon it there is great recurvation of the knee. The left thigh is eight and a half inches in circumference, the right ten inches; the left calf is six and three-quarter inches in circumference, the right eight inches. The temperature of the left sole is 69° F., of the right 73°, of the left calf 74°, of the right 84°. These temperatures were taken in a room where the thermometer stood at 70° F. The muscles of the left leg do not respond to a strong faradic current, but a fifty-cell galvanic current produces good contraction.

Symptoms of the Subacute or Chronic Form.—In this variety the child has little fever or constitutional disturbance, and the onset of the paralysis is gradual. The loss of power shows itself as a weakness in one leg, the child limping for a few days and getting worse by degrees. In a week or longer the paresis extends to the other leg, and in the course of a few days more both legs are more or less completely paralyzed. After a stationary period of a few weeks regression takes place, as it does in the acute form, some of the muscles recovering power completely, while others remain palsied.

Gowers[1] considers the subacute form very rare in children, and says that in it there is no regression, but a steady progression, of the paralysis. He is certainly mistaken in this, for cases are met with in which there is a history of gradual onset of the paralysis followed by return of power.

For example, a boy whom I saw at the Infirmary for Nervous Diseases was well and active until the age of two years. In July, 1874, it was noticed that he was lame in his right leg, then he became weak in both legs, and later both arms and the trunk became so feeble that he could not sit up. This condition came about in four weeks. During this time he had a little diarrhœa, but no fever was observed. At the end of eight or nine weeks he had completely lost power in the legs, but not wholly in the arms. About five months later he began to regain power in the legs. When seen in March, 1875, eight months after the attack, the left leg had regained all the movements, but it could not yet support the weight of the body. The right leg was powerless, but the foot could be feebly flexed and extended.

Erb[2] reports a case somewhat of this kind:

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[1] Diseases of the Nervous System, p. 273.

[2] Brain, 1883.

A girl of six began to have paralysis of the right leg; it came on gradually in July, and in two weeks it had extended to the left foot. In August there was complete motor paralysis, without any disturbance of sensibility. After a course of electrical treatment, power of motion began in the peronei muscles in November, and by January the child was able to walk and the electrical reactions were normal.

Symptoms in Detail.—There are seldom any prodromes. Sometimes a child shows a disinclination to walk or to stand for several days before an attack, or he may have been merely "droopy" and inactive for a few days. A patient of Seeligmüller's suffered from intermittent muscular contractions for six months before the onset of the paralysis.

The disease may be divided into four stages:

1. The initial stage, in which the paralysis is increasing. This may last from a few hours, in the acute form, to some weeks, in the subacute variety. Occasionally there is no initial stage, the paralysis coming on abruptly.

2. A stationary period, which may last from one day to a month or even longer.

3. The stage of regression, which is the characteristic feature of the disease. During this time the paralysis leaves certain parts, but other parts remain powerless. This stage lasts from one to six months.

4. The chronic stage, in which the muscles atrophy and undergo fatty degeneration, contractures take place, and the limb ceases to grow, and becomes cold and mottled in appearance. Slight and gradual improvement takes place in this time and may go on for many months, but usually at an almost imperceptible rate.

There are several modes of invasion. First, the attack may be absolutely sudden, coming on without warning or previous sickness. In one of my patients the child was at play and there was a sudden loss of power in the legs.

The following cases show how abrupt the onset may be:

Lewis S., aged two years. The youngest of six children, the others being healthy. Well until September, 1875, at thirteen months, with the exception of an attack of "summer complaint" just previous. One day while playing on the floor his older brother noticed him fall over, and on examination found that there was complete loss of muscular power in the left leg and partial loss in the left arm. He began to use the arm in a week, but it was a month before he could sit alone or draw up the leg. On the day of the attack he had fever and cried when handled. There was no retraction of the head, and no bladder-trouble. The fever disappeared in a day or two, but the soreness to the touch lasted for a couple of weeks. When examined almost a year later the leg had improved but little. The chief loss of power was in the flexors of the foot.

William McC., aged two and a quarter years. About the middle of August, 1877, on a very hot day, he had been playing in a damp yard. That evening he vomited and had fever. Two days later, while running about, his mother noticed sudden loss of power. The paralysis was general and complete. For two weeks he was unable to sit up. Then he began to use his arms, and soon after to sit up and to use his left leg. On examination five weeks after the attack the right leg was still paralyzed; he could move the toes, but could move no other muscle. There was no response to the faradic current, but twenty cells galvanic current gave good response. Two years later there was power to swing the leg from the thigh. There was still total loss to the faradic current, and the reaction of degeneration was present to the galvanic current. Twenty cells produced contractions AnClC> KClC.

Walter M., aged seven. When four years old, on April 11, after walking in the sun from nine A.M. until two P.M., he came home walking badly and complaining of pain in the back. That night he had some fever and delirium, but no convulsions. Next day he continued unwell. Had epistaxis and pain in the left brow. He had some pain and soreness in the extremities. One day later, while sitting on a trunk, at nine A.M., he suddenly fell off, and was found paralyzed in all his limbs. He was not unconscious. His neck was stiff, and tenderness on movement lasted two weeks. In one week the arms began to improve, and in three weeks he could use the left leg. When seen three years later there was loss of power in the extensors of the right foot and in the flexors of the left.

In a considerable number of cases the child is found to be paralyzed in the morning after a quiet night's sleep. In the majority of cases, however, sickness of some kind precedes the attack for a few hours or days.

In two hundred and fifty-four of my cases in which the child's condition was noted, in one hundred and seventy-eight the attack was preceded by indisposition of some kind; in forty no sickness was observed immediately before the attack; in thirty-six it is not stated whether there was sickness or not. In one hundred and thirty-two of the cases in which sickness was observed preceding the attack the most conspicuous symptoms were as follows:

Fever	57
Fever and chills	5
Fever and diarrhœa	8
Fever and vomiting	21
Fever with vomiting and diarrhœa	10
Vomiting alone	3
Diarrhœa alone	12
Vomiting and diarrhœa	7
Muscular hyperæsthesia	9
Total	132

In two cases several lumbricoids were passed after the administration of a vermifuge at the time of the attack.

The length of time between the discovery of the paralysis and the beginning of the general constitutional disturbance varied considerably in the two hundred and fifty-four cases in which it was noted:

Immediately after	42
One day after	40
Two days after	15
Three days after	16
Four days after	3
Five days after	2
Six days after	2
"Several" days after	2
One week after	11
Two weeks after	13
Three weeks after	8
Four weeks after	4

Five weeks after	1
Several weeks after	6
Two to seven months after	8
Uncertain,—that is, often it was not noted until the child began to walk	32
Before any symptoms	3
Not stated	46
Total	254

As to the character of the fever preceding these attacks, it varies in intensity. Sometimes it is very slight; occasionally it rises several degrees above normal. In a patient of Barlow's the temperature was 104°. There is no relation between the intensity of the fever and the degree of the subsequent paralysis, as in some of the most complete cases of paralysis the primary fever has been very slight. Convulsions occurred during the initial fever in but a small proportion of my cases. Of three hundred and fifty cases, in one hundred and ninety-four there were no convulsions; in thirty convulsions occurred; and in one hundred and twenty-six it is not stated in the notes whether convulsions occurred or not.

In Seeligmüller's cases, convulsions occurred in eleven out of sixty-seven cases. Duchenne reports thirteen out of seventy; Heine, nine out of eighty-six.

The convulsions are usually slight, but occasionally they are severe and general. Delirium may follow the convulsion for a time, but this is unusual except in older children.

As already remarked, the time which elapses after the onset of the general symptoms before the paralysis has been observed varies. Frequently the severity of the symptoms has been so great that the patient has been unable to make any effort to move, and the paralysis may have existed for some time before the parents or friends have observed it. As a rule, when the patient becomes convalescent and attempts to get up it is found that one or more limbs are paralyzed. In the most extreme form of poliomyelitis the patient is unable to move hand or foot, or even the head, and very frequently in this condition the paralysis is overlooked because the inability to move has been attributed to general weakness.

In fifty-seven of my three hundred and fifty cases there was paralysis of all four limbs at the onset, and in twenty-two of these there was paralysis of the trunk and neck muscles as well.

Some writers speak as if at the very onset all the extremities were always palsied and rapid regression took place, but I am sure this is not the case. The paralysis may be absolutely monoplegic from the onset, and indeed even a single group of muscles only may be paralyzed.

In a patient whom I had the opportunity of seeing the day after the attack there was paralysis of the right arm alone. The case is as follows:

Jeremiah D., aged one year. Was seen August 9, 1876. The day previous it was found that he could not use his right arm. The child had been sick for some days with fever, and was nervous, but did not cry when he was moved. He had eight incisors, and the gums over the molars were not swollen or tender. The bowels had been undisturbed, but he vomited two or three days before the paralysis came on.

On examination he was found to be a healthy, well-nourished child. There was no spinal tenderness or general hyperæsthesia. He could move the fingers of the right hand and flex the wrist, but could not extend it, nor could he move the rest of the arm.

The extensors of the wrist responded to two inches secondary induced current, but it took three inches to cause the biceps to contract, and the deltoid and triceps did not respond at all to the faradic current. These muscles could be made to contract by thirty cells galvanic, the current being reversed.

The patient was under treatment and observation for a year. On September 7, 1877, the following note was made: "Slight power of movement exists in the triceps, but none in the biceps. The scapular muscles are wasted."

This was one of the rare cases where the muscles of the forearm escape when the upper arm is paralyzed.

The most frequent form is where both legs only are paralyzed at the onset. In the cases which I have tabulated the following is the distribution of the paralysis at the onset, and also at the time when the patients applied for treatment:

	Parts Affected at the Onset.	Parts Affected when Examined.
Both legs	107	92
Right leg	63	99
Left leg	62	82
Left arm and left leg (hemiplegic form)	14	6
Right arm and right leg (")	12	9
Left arm and right leg	..	1
Right arm and left leg	1	1
Both legs and right arm	6	6
Both legs and left arm	4	2
Both legs and one arm	..	1
Both arms	1	3
Right arm	5	4
Left arm	8	7
Arms and legs	35	16
Arms, legs, and trunk	22	9
Not stated	10	12
Total	350	350

As yet we have not had sufficient competent observation during the initial stage of the disease to know the exact character of the paralysis at the onset. But few cases have been recognized by the attending physician at the beginning; in fact, in the large majority of cases the physician has not been called in until after the paralysis was observed by some of the family. In one or two instances which have come under my own observation, in which there was a physician in attendance during the initial symptoms, the paralysis was not observed until after the febrile symptoms had subsided.

The original extent of the paralysis does not by any means correspond to the extent of paralysis observed later, because within a few days from the onset recovery takes place in certain of the affected limbs,—regression, as it is called.

Certain of the muscles seem always to escape paralysis: paralysis of the facial muscles is rare, and the muscles of the eyeballs, ears, and larynx always escape, as do the diaphragm and the intercostal muscles. This fact explains the absence of any interference with respiration. In the adult form of this disease facial paralysis is sometimes observed; I have met with it in one instance myself, and in this case it was double. Seguin reports a similar case.

The regression of the original paralysis is remarkable and very interesting. In a few instances complete recovery seems to take place in all the affected parts. For example, in the so-called temporary paralysis of Kennedy and Frey[1] there is complete motor paralysis of all the muscles of the trunk and extremities, followed in a few days by recovery and no trace of the paralysis remaining. This form of the disease is very rare, but Kennedy reported several examples coming under his own observation.

Barlow reports the case of a boy who at the age of five months was affected with universal paralysis but recovered entirely except in the extensor longus digitorum of the foot.

Although these exceptional cases do occur, it is far more common to find that the regression of the paralysis is only partial.

Laborde[2] has recorded a case in which there were no fewer than three separate attacks in the same child. There was complete regression after the first two attacks, but after the third the paralysis was permanent.

I have seen a case like Laborde's. The patient, a girl of eight, had an attack of paralysis affecting both the arms and legs, in July, 1888. She waked one morning with both legs paralyzed. The arms were unaffected at first, but after four or five weeks they became gradually palsied, so that finally she could not use them at all. In two or three months she began to use the arms, and by November of the same year she was able to walk about. She continued to improve, and during the following year and a half her mother says that she was as well as ever, except that the right leg remained weak and she swung it in walking. In June, 1890, there was an attack of general muscular weakness, but this lasted only a day or two. Two weeks later, that is, about July 1, she began to lose power again. The paralysis came on by degrees, beginning in the legs and extending to the arms. By August 1 she was paralyzed in both arms and legs, and the trunk muscles were so weak that she could not sit alone. At times she could not support the head.

She was now admitted to the Infirmary for Nervous Diseases, and there was found wasting of the arm and leg muscles. Inability to sit alone, and to move hands or feet; the knee-jerk was absent; muscles did not respond to the faradic current, and the galvanic current showed the reaction of degeneration. The sphincters were unaffected, and sensation was good.

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[1] Dublin Quarterly, 1850; Berlin. Klin. Wochenschr., 1874.

[2] De la Paralysie de l'Enfance, Paris, 1864, p. 36.

It is noticeable how much more frequent the paralysis is in the lower than in the upper extremities. By reference to the table on page 696 it will be seen that of the three hundred and fifty cases the paralysis was confined to the arms alone in but fourteen instances, while it frequently attacked either one leg alone or both legs.

The hemiplegic form, although rare, does occur, as is shown in the table, where there are twenty-six instances of this variety of the disease. Of these twenty-six cases of the hemiplegic form fourteen occurred on the left side and twelve on the right side. Even a crossed variety of hemiplegia may occur. There is one example of it in my table, in the table of Duchenne there are two, and Seeligmüller has reported one instance of this variety of paralysis.

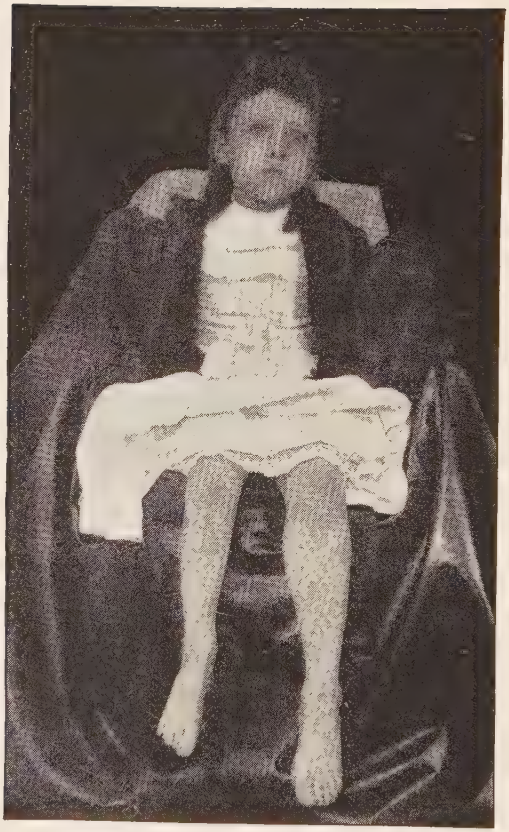
The one-sided paralysis of poliomyelitis is readily distinguished from a cerebral hemiplegia by the fact that facial paralysis is rarely associated with it, by the absence of unconsciousness, and by the electrical reactions, and also by the fact that there are no secondary contractions in the upper extremities in poliomyelitis, while in cerebral hemiplegia they almost invariably occur.

In one of my cases the original paralysis was confined to both arms, and did not affect the trunk or the lower extremities: this case was like one reported by Seeligmüller. In his case the paralysis occurred at the age of seventeen months, and attacked the arms only, but never retreated from them. At four years of age, when the patient was seen, the arms were much atrophied and faradic contractility in the muscles was lost.

The paralysis may be confined to a single group of muscles from the very outstart, and when only one group is affected this is most likely to be the flexors of the foot; sometimes the deltoid alone is paralyzed, and occasionally, after the regression of the paralysis from the upper extremity, the muscles of the hand alone are found to be paralyzed.

The maximum extent of the paralysis is usually reached in from one to four days: occasionally it takes a much longer time for its development to occur, and when it does not reach its height until after a week it may be regarded as a subacute variety of the affection. The following case illustrates an example of the disease in which all the extremities were affected at the onset:

Alice S., aged three years, had good health until the middle of June, 1889; was attacked one night with diarrhœa, vomiting, and fever. The next day there was considerable muscular twitching, but there were no convulsions. She seemed weak and prostrated, and took but little notice of what was going on about her. The fever continued several days, during which time she was sore to the touch, even complaining of the weight of the bedclothes, and cried when she was moved. In a day or two it was found that she did not attempt to move the arm or leg or lift the head. In two weeks she began to use her arms and sit up. In three or four weeks she began to use the left leg. At the end of thirteen weeks I examined her and found that there was complete use of both arms. She could stand on the left leg and use it freely. She could flex slightly the toes of the



Case of Poliomyelitis Anterior, showing Atrophy of Right Leg.

right foot, but could not flex or extend the foot or the leg. She could move slightly the flexors and extensors of the thigh; the knee-jerk was normal in the left side, but absent in the right side.

The paralysis usually remains stationary from one to six weeks, and then regression begins. It may, however, begin to decrease after two or three days; and, again, recovery may not begin for three or four months. As a rule, however, if regression does not begin in two months it is not likely to occur. The first improvement usually takes place in the parts last affected, and, as a rule, extends until all the parts have recovered except those which are to be permanently paralyzed. Within two or three weeks distinct wasting of the muscles begins: the muscles are flabby and without tone from the first, but very soon the atrophy begins and rapidly increases until the shape of the limb is greatly changed. In fat children the appearance of the limb is not so much altered, but in all cases there is great difference in the size of the limbs. In a child three years of age there may be as much as an inch of difference in the circumferences of the calves of the legs.

In the paralyzed limb there is not only atrophy of the muscles, but also arrest of growth in the bone. In the course of a few months the limb becomes notably shorter than its fellow. In the case of the lower extremities this is very marked; and it is noticeable not only in the length of the leg, but also in the length of the foot. After a very few months the foot is found to be much shorter than the unaffected one,—a difference of from one-half to three-quarters of an inch being noted in many cases.

Other changes occur in the nutrition of the affected part. The skin becomes adherent to the connective tissue underlying it, and when one attempts to pinch up the skin it cannot be separated from the tissue as in a healthy skin, but the whole mass is brought up together.

The temperature of the limb is very much lowered, there frequently being two or three degrees' difference between the sound and the paralyzed limb. In one case which I examined, in which there was paralysis of the left leg, the temperature of the left calf was 74°, while that of the right was 84°,—a difference of ten degrees. The shortening of the limb and foot cannot be considered as due to atrophy of the bone, since in adults no such change occurs; it is merely a retardation of the growth. Seeligmüller has observed an actual elongation of the limb in some cases, which he ascribed to the fact that the growing epiphyses suffered traction instead of the normal compression.

The joints that depend for their support on the tendons that pass over them become relaxed, and the limb may appear elongated from this cause: for example, in paralysis of the shoulder muscles the head of the humerus drops out of the glenoid cavity to a considerable extent.

The circulation in the affected limbs is greatly disturbed, the capillary circulation being sluggish, giving rise to a dusky purplish hue of the skin. Very frequently the surface has a mottled appearance, even in warm weather. There are never, however, any bed-sores, ulcerations, or extreme trophic changes in the skin.

During the acute stage of the primary symptoms the child usually complains of pain and soreness in the extremities and trunk; when moved or lifted he frequently cries out with pain. There is, however, no tenderness on pressure over the spine, and no hyperæsthesia. With the febrile symptoms the pain and soreness usually pass away. There is seldom, if ever, any loss of sensation during the initial symptoms. It is possible, though, that this may occur and escape detection because it has not been specially looked for; but there are no cases recorded of any loss of sensation in the affected parts. Occasionally there is tingling or formication in the limbs.

There is very seldom any loss of control of the sphincters; there is no incontinence of urine, and no loss of control of the bowels. Occasionally there is paralysis of the bladder, and this is met with in unusually severe cases, in which spinal pain and some evidences of inflammation of the meninges are present. In a patient whom I saw in consultation there was loss of bladder-control for a few days after the febrile symptoms had subsided. In six of the cases which I have tabulated there was retention of urine lasting a few hours. The longest time the retention lasted was twenty-four hours. Of the cases in which the bladder was affected four were girls and two were boys. In all of them there was marked pain on movement or on being handled. In one case, a boy of four years, the attack of paralysis came on after he had been bathing in a creek in the end of July. There was stiffness in the back, and the patient complained of pain. It is probable that in most, if not all, of these cases there was some meningitis or a mild form of transverse myelitis. Gowers[1] thinks that when the bladder is affected the trouble is likely to last a long time. He reports a case of a child two and a half years of age, who woke up one morning with headache, fever, and weakness of the legs, followed by complete paralysis. In a few days the urine escaped involuntarily. The arms began to recover in six weeks, and were well in six months; the legs remained permanently paralyzed. The incontinence of urine lasted for a year.

The paralysis generally develops rapidly. It begins in one limb and spreads to the others quite speedily, or it apparently attacks all the extremities at once.

When single muscles are affected, the deltoid suffers alone more frequently than any other muscle of the arm. It may be paralyzed with other muscles; for instance, the deltoid, supra- and infra-spinatus, biceps, and triceps are all affected, producing the so-called upper-arm type of palsy of Erb. The extensors and supinators of the hand are often paralyzed, and in some cases the muscles of the hand alone are affected, as in a patient under my care, in whom the intrinsic hand muscles are wasted and the deltoid is paralyzed, while the other muscles of the arm are unaffected. In this case there is also paralysis of the legs. The serratus magnus is occasionally affected, and so are the trapezii and the other scapular muscles. There is seldom, if ever, complete paralysis of the intercostals or other trunk muscles, but they are frequently greatly weakened, so that the patient is scarely able to sit up; and this weakness produces lateral curvature or antero-posterior curvature of the spine if the patient is allowed to be in the erect posture to any extent.

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[1] Op. cit., p. 257.

The individual muscles most frequently found paralyzed are those of the leg. The tibialis anticus is paralyzed alone oftener than any other muscle. Next in order of frequency comes the group of muscles of the anterior part of the leg forming the flexors of the foot and the extensors of the toes. Then follow the extensors of the leg. The facial muscles are rarely paralyzed. There is one case of facial paralysis in my tables, and Gowers and Barlow each report one.

The following schedule of symptoms by Jacobi[1] is useful in showing the diagnostic features of the different palsies:

Upper Extremity.—Deltoid.—Absence of deformity, which is averted by weight of arm. Inability to raise arm. Sometimes subluxation. Frequent association with paralysis of biceps, brachialis anticus, and supinator longus.

Lower Extremity.—Ilio-Psoas.—Rare except with total paralysis. Associated with paralysis sartorius. Loss of flexion of thigh. Limb extended (if glutæi intact).

Glutæi.—Thigh adducted. Outward rotation lost. Lordosis on standing. Frequent association with paralysis of extensors of back.

Quadriceps Extensor.—Flexion and adduction of leg (if hamstrings intact). Loss of extension of leg. Frequent association with paralysis of tibialis anticus.

Tibialis Anticus.—Often concealed if extensor communis intact. If both paralyzed, then fall of point of foot in equinus. Dragging point of foot on ground in walking. Big toe in dorsal flexion (if extensor pollicis intact). The tendons prominent. Hollow sole of foot (if peroneus longus intact).

Extensor Communis.—Nearly always associated with that of tibialis anticus. Toes in forced flexion.

Peroneus Longus.—Sole of foot flattened. Point turned inward. Internal border elevated.

Sural Muscles.—Heel depressed. Foot in dorsal flexion (calcaneus). Sole hollowed if peroneus longus intact, flattened if paralyzed. Point turned outward (calcaneo-valgus).

Extensors of Back.—Lordosis on standing. Projection backward of shoulders. Plumb-line falls behind sacrum (unilateral). Trunk curved to side. Trunk cannot be moved towards paralyzed side.

Abdominal Muscles.—Lordosis without projection backward of shoulders.

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[1] A System of Practical Medicine, Lea Brothers & Co., 1886, vol. v. p. 1123.

Occasionally sequelæ occur during the chronic stage, but they are rare. As a rule, the general health of these patients is excellent, and they suffer but little except from the difficulties of locomotion and from the deformities.

In a few instances, however, some other degenerations of the cord have occurred after the subjects of poliomyelitis have reached adult life. Gowers states that he has twice seen the symptoms of lateral sclerosis without atrophy slowly develop,—in one case at seventeen, in the other at twenty-eight years of age.

Progressive muscular atrophy has been observed to begin in the paralyzed limb in these cases, and I have seen an instance of a general poliomyelitis in the adult followed by progressive muscular atrophy.

The deformities which occur result from contraction of the paralyzed muscle, from over-action of a sound muscle when its opponent is paralyzed, or from the use of the limb in walking. These contractions occur at varying periods of the paralysis. The earliest deformities that have been observed occurred two months afterwards. Seeligmüller has reported a case of pes equinus and pes calcaneus which developed in four weeks. The time at which the deformities occur, however, is influenced largely by the time at which the children begin to walk. Most of the cases of congenital club-foot are probably the result of an intra-uterine paralysis, and the deformities which occur here are obviously not the result of any weight upon the limbs, but the result of position.

The most frequent varieties of club-foot are equino-varus, knock-knees, and valgus. There may be deformities of the upper extremity as well as in the foot, but they are rare. Occasionally club-hand is met with, or flexion of the wrist. These have been reported, but I have never met with them. The deformities of the lower extremities occur in at least seventy-five per cent. of the cases.

Besides the varieties of club-foot above mentioned, there are deformities of the knee,—recurvation, and permanent flexion. In the hip we may have dislocation, permanent flexion, and permanent adduction; the two latter are, however, rare. The deformities of the knee are always the result of the use of the limb in walking, occurring from the weight of the body on the relaxed ligament of the joint. The rarest of all the forms of club-foot in poliomyelitis is calcaneus. This is the result of paralysis of the calf muscles, the flexors of the foot remaining intact. When this condition exists, the patient walks on the heel, and a very striking deformity is produced.

Deformities of the trunk, as before remarked, occur quite frequently, and are the result either of weakness of the truncal muscles or of difference in the length of the limbs. Where one leg is paralyzed, the patient in walking will throw his weight on that side, so that the hip falls and a lateral curvature is acquired. Where there is paralysis of the glutæi, lordosis occurs, the shoulders being thrown too far backward, and the hips forward.

In the notes of one hundred and seventy-two of the cases in my tables reference is made to the presence of deformities. The deformities which existed are as follows :

Talipes equinus	28
" calcaneus	5
" valgus	19
" varus	11
" valgus, both feet	3
" varus, both feet	1
" equinus, both feet	8
" equino-varus	7
" equino-varus, both feet	2
" equino-valgus	3
" equino-valgus, both feet	1
" calcaneo-valgus	3
" valgus (L.) and equinus (R.)	2
" equino-varus (R.) and equino-valgus (L.)	2
" equino-varus and calcaneo-valgus	1
Genu valgum	1
Contraction at knee	1
Contraction of plantar fascia	1
Deformities present, but character not stated	9
No deformity present	64
Total	172

Contraction of the plantar fascia occurs in a large proportion of cases in connection with equinus and valgus. It is one of the most troublesome conditions to deal with, and often causes much pain to the patient in walking.

The most remarkable symptoms of poliomyelitis are the rapid wasting and atrophy of the paralyzed parts and the early electrical changes. The atrophy takes place almost immediately, and, as Jacobi (*loc. cit.*) remarks, is even more rapid than that following the section of a nerve. All the parts of the limb undergo atrophy, even the blood-vessels and nerves. At times the wasting is concealed somewhat if the child is very fat, but even then the shape and color of the paralyzed members are very different from those of the sound limbs.

The lowering of the temperature is due to loss of muscular contractions, which attract blood to the part, to diminished or lost nerve influence, and to shrinkage of the blood-vessels. Not only are the smaller vessels atrophied, but the main trunks are much smaller. The entire iliac artery and even the lower part of the aorta have been found distinctly reduced in calibre.[1]

The electrical changes are met with early in the disease. Faradic contractility in the affected muscles is diminished in from three to five days after the onset of the paralysis. In the case of Jeremiah D., page 695, on the day after the attack there was marked loss in the contractility of the palsied muscles to the faradic current. At the end of a week or ten days there is complete loss in the muscles in which the paralysis will be permanent. We can say almost with certainty that those muscles which do not respond to the faradic current at the end of two or three weeks will be permanently paralyzed. This rule is not absolute, however. The muscles in which regression takes place recover the faradic contractility with restoration of power, although the latter may precede the return of electrical contractility.

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[1] Charcot and Joffroy, quoted by Jacobi, loc. cit.

When the muscle has completely lost excitability to the faradic current it still will respond to the galvanic current, and the reaction may be different from that which is found in healthy muscles. There may be contraction at the opening as well as at the closing of the circuit. When the paralysis has lasted for some weeks the so-called reaction of degeneration is found; that is, the contraction which occurs at the anodal closure equals or is greater than that of the kathodal closure. We have first in the affected muscles "quantitative" changes,—that is, changes in the degree of irritability; then, later, are seen "qualitative" changes, by which are meant changes in the order of the response. For example, the normal reaction is—1, KClC; $2, \left\{\begin{matrix}\text{AnClC}\\ \text{AnOC}\end{matrix}\right.$; 3, KOC; meaning that the order of strength of contraction is—first, kathodal closing contraction; next, anodal closing contraction and anodal opening contraction; and lastly, kathodal opening contraction. When the paralyzed muscle has undergone degeneration, the reaction is—1, KClC = AnClC; 2, AnOC; 3, KOC; and later, as follows: 1, AnClC; 2, KClC; 3, KOC; 4, AnOC. When the muscle begins to improve, there is first a return to the normal reaction to the galvanic current, and later a response to the faradic current.

The reflexes are lost early in the disease. The knee-jerk disappears within the first day or two, and the skin reflexes are lost as early. Of course the extent to which the reflexes are abolished depends on the portion of the cord affected. If the lower part of the cord only is involved, the plantar and cremasteric reflexes are absent. When the ganglion-cells in the anterior horns through the whole cord have degenerated, the skin and tendon reflexes will be abolished. In case of recovery the knee-jerk may return. In those cases where there is degeneration of the lateral columns as a secondary effect there will be exaggerated knee-jerk and skin reflexes.

Pathology.—For many years there was great diversity of opinion as to the nature of the pathology of this affection, some writers holding that it was a peripheral disease involving the muscles primarily, while others thought that there were organic changes in the centres. Of late years improved methods of examination and advanced knowledge have established without question the nature and location of the lesion. For some time it was generally admitted that the disease was spinal; but it remained for Charcot and Joffroy to demonstrate the constancy of the lesion in the great ganglion-cells of the anterior cornua.

Within a few years (in 1885) Dr. D. Drummond[1] has had the opportunity of making an autopsy in a very recent case. A child of five years died after a few hours' illness. In the cervical portion of the cord there was undue redness of the anterior gray matter. The vessels from the surface to the cornua were distended with blood. Microscopically there was seen distention of the capillaries and extravasations in the gray matter, with swelling of the neuroglia and of the ganglion-cells, which were granular, with indistinct processes.

Damaschino[2] made an examination of the cord of a child of two and a half years, who died twenty-six days after the attack. The left leg and the right arm had been palsied. Foci of red softening were found in the anterior cornua in the left lumbar and right cervical regions. There were also present distention of the blood-vessels, enlargement of the muscular net-work, granular corpuscles in lymphatic sheaths, marked atrophy of the cells and of myeline sheaths of fibres in anterior roots; axis-cylinders had disappeared. Lesions were marked throughout the cord.

Another recent case was reported by Dr. Charlewood Turner in 1879.[3] A child of two and a half years had an attack of paralysis of both legs, coming on suddenly about two weeks after a fall on the back, which did not seem to affect her at the time. A few days after the loss of power in the legs the upper extremities became powerless. The child was admitted to the London Hospital two weeks after the beginning of the attack. At this time there was complete loss of power in all the extremities, with loss of sensation in the lower limbs, and the stools were passed involuntarily. The child had an attack of measles and died six weeks after the onset of the paralysis. At the autopsy changes were found in the anterior horns and antero-lateral columns through the whole length of the cord. About the centre of the lumbar enlargement was found a small patch of reddened gelatinous-looking matter in the left anterior cornua. In the neighborhood of this hemorrhagic focus the nervous tissues were completely disintegrated, so that no nerve-structure could be distinguished in the anterior horn and in the outer part of the base of the posterior cornua.

In this and some other recent cases acute changes have been found in the anterior horns more advanced than in Dr. Drummond's case referred to above. They may be quite general, but slight in degree, with greater intensity at different points, usually in the cervical or in the lumbar enlargement, or in both. At these spots there is softening; sometimes there is hemorrhagic infiltration, and sometimes an actual cavity, as in Charlewood Turner's case. The microscope shows extravasated blood often massed along the vessels and scattered throughout the gray matter, together with cells such as are found in myelitis. There are found granule-corpuscles and other products of degeneration of the nerve-elements. The motor cells have almost disappeared. When the changes are slight the motor cells may be structurally intact, but swollen and granular. The changes are confined to the anterior horns or may extend in slight degree into the antero-lateral column. The posterior columns are unaffected, but it is possible that a hemorrhagic infiltration may extend into the posterior horns, as seen in Turner's case. In this case the lesion at the base of the posterior cornua explains the loss of sensation in the lower extremities which had existed during life. Slighter and diffused changes may be found beyond the softened areas. These changes consist in single granular cells scattered throughout the gray matter, increase of nuclei, dilatation of the blood-vessels, and degeneration or disappearance of the multipolar ganglion-cells. The antero-lateral columns have been found reduced in size and the seat of a slight sclerosis. The trabeculæ are then thickened and the nerve-fibres are atrophied. The anterior roots are diminished in size, and show evidences of degeneration under the microscope.

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[1] Quoted by Gowers, op. cit., p. 264.

[2] L'Union Médicale, 1883, quoted by Jacobi, op. cit.

[3] Ross, Diseases of the Nervous System, vol. ii. p. 121.

In cases which have been examined post mortem several years after the onset of the paralysis the morbid changes are generally the same. The anterior horns are atrophied, and the antero-lateral columns appear to the naked eye gray, translucent, and atrophied. The posterior cornua and vesicular column of Clarke are almost, if not entirely, normal. Microscopically lesions are to be found in the anterior horns at the cervical and lumbar enlargements, and in addition to these lesions more or less diffused changes are met with in the gray matter and white columns. The anterior cornua are shrunken in size, and within the diseased foci which they contain is found fibrillated connective tissue rich in nuclei. The blood-vessels are enlarged and their walls are thickened. Granule-cells are not found, but generally a large number of corpora amylacea and pigment-granules are met with. The large ganglion-cells and nerve-fibres are more or less completely destroyed in the diseased foci, and those which remain are in all stages of degeneration and atrophy. Outside of the diseased foci healthy multipolar ganglion-cells may be found.

More or less sclerosis of the antero-lateral columns is met with, the neuroglia is thickened, and generally there is some atrophy of the nerve-fibres. The sclerosed area is sometimes confined to the immediate neighborhood of the anterior horns, sometimes diffused throughout the antero-lateral columns, the pyramidal tract in the latter case being particularly liable to suffer. As a result of these changes the diseased half of the cord is smaller than the other, and this is often apparent to the naked eye. The alteration in the shape of the cord is most marked when the damage to the gray matter is greatest. The anterior nerve-roots at the most diseased points are small and gray, and the degeneration of the nerve-fibres may be traced down the nerve-trunks. Often a few fibres appear healthy, although all the rest have perished.

It is now generally agreed that the primary lesion is an inflammatory condition in the anterior horns of the gray matter, and that this may be preceded in rare cases by a slight hemorrhage. The inflammation spreads over the greater portion of the cord, but is most intense and violent in the cervical and lumbar enlargements, where foci of softening and destruction of the ganglion-cells occur. When the inflammation subsides, a gradual improvement takes place in those areas where the destruction of the gray matter has been incomplete; but when the nervous matter has been entirely destroyed there is a gradual development of cicatricial connective tissue in its place.

The peripheral nerves have been found to be atrophied.

The muscular tissue undergoes degeneration at an early period. At first the muscular fibres are narrowed and in a state of granular degeneration, with increase of the nuclei of the sheath and of the interstitial tissue. Granules and pigment masses are found between the sarcolemma sheaths. In advanced cases there is complete disappearance of the muscular fibres, and their place is taken by fibrous tracts, which are developed partly from the sarcolemma sheaths and partly from the interstitial connective tissue. Here and there will be found healthy muscular fibres. Sometimes fat accumulates in the interstitial tissue, so that the muscle does not materially lessen in bulk. In some of the lighter cases in which incomplete recovery takes place, some of the fibres are restored to their normal appearance, but they are smaller and the interstitial connective tissue is greater in amount than usual. Barlow (*loc. cit.*) says that it is now generally conceded that a muscle may be much wasted in bulk without showing any marked sign of degeneration microscopically.

The tendons are atrophied and stretched. The growth of the bones is always retarded. The medullary portion is increased relatively, and the fatty contents are more abundant; the external hard lamella of the bone is thin and friable. The bones are not, however, especially liable to fracture. The ligaments of the joints are stretched and relaxed, and the articular extremities of the bones are atrophied, the cartilages thin, and sometimes eroded. The arteries are diminished in calibre.

For the above description of the pathology of this disease I am largely indebted to Ross, Jacobi, and Gowers.

Diagnosis.—This is not difficult when we consider the essential features of the disease. Certain of the symptoms are so striking and peculiar that when present the diagnosis can readily be made. But all cases are not typical, and in the early stages before the full development of the paralysis it is not easy to make a diagnosis. The fever which precedes the attack may be mistaken for the pyrexia of cold, indigestion, or some other cause, and when the paralysis does appear it may be, and often is, mistaken for weakness from the prostration following the fever. If we bear in mind that the prostration following an attack of illness never amounts to complete loss of power to move, the mistake will not be made.

The chief characteristic features of poliomyelitis anterior are—

1. An abrupt onset, generally accompanied by fever, vomiting, or diarrhœa, or the paralysis is observed in the morning, after more or less fever has existed for twelve or twenty-four hours.

2. The paralysis usually is complete within a few hours. This is not always the rule, for the paralysis may be gradual and involve one limb after another.

3. Absence of disorders of sensation, rigidity, or contractures of the limbs. There may be hyperæsthesia or articular soreness on motion, but this is transient. Absence of vesical or rectal paralysis.

4. The regression. This almost invariably takes place. Occasionally, when but one limb or group of muscles is attacked in the onset, there is no regression. The loss of power recedes gradually from some of the muscles, and finally becomes localized in certain groups which are supplied by the same nerve.

5. The early loss of response to the induced electrical current in the affected muscles, and later the reaction of degeneration which is found. The return of response to the faradic current with restoration of power.

6. The arrest of growth in the paralyzed limb. The shortening of the bones of the foot. The deformities which result, giving rise to the various forms of club-foot. The peculiar condition of the skin in the affected part, with the absence of bed-sores or trophic lesions.

The diseases which are most likely to be confounded with poliomyelitis are cerebral palsies, general or transverse myelitis, progressive muscular atrophy, diphtheritic paralysis, and multiple neuritis.

The diagnosis from *cerebral palsies* is, as a rule, not difficult. A cerebral paralysis is almost always hemiplegic, while in poliomyelitis this form is rare, and especially so in the onset. The hemiplegic type may remain after the recession of the paralysis from the other limbs. Cerebral paralysis is generally preceded by convulsions, and there is stupor or delirium. Both of these conditions are rare in poliomyelitis. Facial paralysis is frequent in cerebral hemiplegia, and very unusual in the disease under consideration. In cerebral palsy the muscles respond readily to the induced current, and in poliomyelitis there is no response. The knee-jerk is exaggerated in one and abolished in the other. The skin reflexes may be lost in both.

The following table from Jacobi[1] is convenient in showing the differential points in the two diseases:

POLIOMYELITIS.	CEREBRAL PARALYSIS.
Paraplegic or monoplegic the rule. Hemiplegic as residuum from paraplegia and involving facial nerve (very exceptional).	Hemiplegic the rule. Monoplegic as residuum of hemiplegia, or as result of solitary tubercle (exceptional).
Intelligence free (rule).	Intelligence depressed (rule).
Intelligence depressed, when spinal paralysis has affected imbecile children.	Intelligence free (exception, especially with solitary tubercle).

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[1] Op. cit., p. 1152.

Poliomyelitis.	Cerebral Paralysis.
Disposition lively.	Disposition apathetic or cross.
Initial convulsion unique; general symptoms of a few hours' duration (rule). Convulsions repeated during two to three weeks before paralysis (?); fever a month (rare exception).	Convulsions repeated; pyrexia prolonged several days or weeks (rule).
Sensibility intact (rule).	Sensibility intact after initial period.
Reflexes cutaneous, and tendon lowered or lost (rule). Reflexes preserved when the single muscles in groups are paralyzed.	Reflexes intact.
Associated movements of hand absent (Seeligmüller).	Associated movements frequently observed in hand.
No rigid contractions of upper extremity.	Extensive and rigid contractions of upper extremity very frequent.
Atrophy of paralyzed muscles and arrested development of limb very marked.	Atrophy very slight.
Faradic contractility diminished or lost; degenerative muscular reaction.	Electrical reactions normal.

In *myelitis* there is more violent and persistent fever. There is hyperæsthesia, which is intense, and is followed by anæsthesia. There is considerable pain on movements of the trunk, and girdle-pain is generally present. The reflexes and knee-jerk may be absent at first, but in a few days they return and become excessive. The muscles may atrophy, but there is no loss of response to the faradic current. Bed-sores occur early. The onset of the paralysis is, as a rule, much less rapid than in poliomyelitis. Transverse myelitis in children is usually due to disease of the vertebræ, and this gives rise to deformity, which can be detected.

Progressive muscular atrophy is a rare disease in children, and should not be mistaken for poliomyelitis, even in the chronic form. The onset of the paralysis is gradual and corresponds to the increase of the atrophy. Faradic contractility remains as long as any muscular fibres are left. The knee-jerks often remain to the last. There is no arrest of growth in the bones.

Diphtheritic paralysis may sometimes be mistaken for poliomyelitis. Often there is so long an interval between the diphtheria and the paralysis that there is a difficulty in diagnosis. In diphtheritic paralysis there is a history of throat-lesion; the paralysis is less general, and usually affects the pharyngeal muscles as well as those of the limbs. The response to the faradic current is lowered, but not completely lost, and there is no recession of the paralysis from certain muscles or groups of muscles, but recovery takes place gradually and steadily.

Multiple neuritis seldom occurs in children, but when it does it resembles poliomyelitis in many respects. There is more marked hyperæsthesia, and there is tenderness over the nerve-trunks. Movements of the paralyzed limbs are painful. There are sensations of numbness and pricking in the parts affected. The paralysis comes on rather more slowly, and the loss of response to the faradic current is more gradual; moreover, regression does not occur. In paralysis from lesion of a peripheral nerve there is a close resemblance to poliomyelitis when the paralysis is limited. It is distinguished by observing the distribution of the injured nerve and the accompanying disturbances of sensibility and trophic skin-lesions.

Hip-joint disease may be mistaken for infantile paralysis. I have seen one case in which the parents thought the leg was paralyzed, when it was immovable from pain in an inflamed hip-joint. An examination shows that there is no paralysis and no interference with reflex actions; indeed, the knee-jerk may be exaggerated. In hip-disease it is common to find difficulty in extending the leg, but the presence of the knee-jerk shows that spinal paralysis does not exist.

Hemorrhage into the gray substance of the cord may give rise to symptoms which resemble those of poliomyelitis in its sudden onset, the paralysis followed by atrophy, the absence of reflex action, and the loss of electrical irritability. In hæmatomyelia, however, the paralysis is more sudden, the initial fever is absent, and there are disturbances of sensibility, paralysis of the sphincters, and bed-sores.

Birth-palsies are observed immediately after delivery; the face and arm are usually affected, and the wasting which occurs is localized to certain muscles. Recovery generally takes place early.

Spastic paraplegia is readily distinguished by the fact that the paralysis is gradual, there is but little atrophy, the limbs are in a rigid condition, the reflex actions are exaggerated, and there is no loss of faradic irritability.

Prognosis.—As regards life, the prospects of the patient are exceedingly good. In some cases the child may die from the intensity of the initial symptoms or from implication of the respiratory muscles at the onset of the attack. Marked cerebral symptoms are of serious import. After an attack a child may be left so much weaker that he is readily overcome by some other illness.

I have seen one case, not a child, but a youth of eighteen years, who succumbed to an attack of poliomyelitis twelve days from the onset, from paralysis of the respiratory muscles. The patient I saw in consultation with Dr. Radcliffe Cheston. He had gone to the sea-shore on an intensely hot day in July, and in the evening there was a sudden change in the temperature. The patient became exceedingly cold, and the next day he had weakness of the legs. In five days there was general paralysis, but it was not complete in all the affected parts. The legs were motionless, but the forearms, hands, and fingers could be moved feebly. The head could be extended, and turned from side to side, but not flexed. Swallowing was difficult, but was relieved for two days, to become again almost impossible. Sensation was good, except for some confusion to the compass-points. All the skin and tendon reflexes were lost, except the cremasteric. There was retention of urine for a time on the tenth day, but the bladder regained its tone. The muscles responded to a slowly-interrupted faradic current. On the eleventh day the respiratory muscles became weaker, and the patient died on the twelfth day. The mind was clear throughout.

The danger to life in the great majority of cases is slight, but the important question soon arises, How far will the patient recover, and which muscles are likely to remain paralyzed? This point cannot be decided until the paralysis has become stationary; and this, as a rule, does not take place until after a week or ten days. An electrical examination will now be of great service in helping us to determine which muscles are likely to recover and which will have definitive paralysis. Those muscles which do not respond to the faradic current are likely to remain permanently paralyzed. An electrical examination should be made every few days, and it may be found that the muscles which at first did not respond to the induced current will after a week or more give a feeble response. We may then expect partial or complete recovery to take place in these muscles. When there is no loss of faradic contractility a few days after the paralysis has become stationary, we can with reasonable certainty look for recovery in a few weeks or months. If the electrical contractility is gradually lost, wasting and paralysis will result for a long time. If after two or three months there is much wasting and the loss of power is complete, there will never be much gain in strength. If the patient is seen in the chronic stage, the prognosis will depend on the degree of atrophy which has taken place and the electrical condition of the muscles. If there is even a feeble response to the faradic current, we can safely predict considerable gain; but if the muscles fail to respond to this current and give the reaction of degeneration to the galvanic current, there is little or no hope that there will be any restoration of power. In any event it is extremely rare that perfect recovery takes place. Even when every muscle in the paralyzed limb has regained power of motion there remain a certain enfeeblement and a degree of wasting which prevent the member from being as perfect as its fellow. The loss of power is most conspicuous in the leg. In the arm a slight amount of weakness or atrophy is not noticeable. On the other hand, we should not give an unfavorable prognosis even if the paralysis has persisted for many months or even for a year or two. Faithful and intelligent treatment will often bring about gain in an atrophied and powerless limb. As regards walking, there are few cases which cannot be made to walk very well with the aid of suitable apparatus, and often the improvement after the apparatus has been applied will increase for some time. I am, however, far from agreeing with Dr. Gibney when he says, "It will be seen that surgery, after all, offers the only relief in infantile paralysis."[1] I have seen a number of cases in which conspicuous gain followed the use of massage, electricity, and other therapeutic means. Many others have had the same experience.

In one patient, whose case I have related elsewhere,[2] there was, eighteen months after the attack, no response to the faradic current in any of the muscles of the left leg except the anterior tibial group, and the child was unable to move any part of the limb, with the exception of flexing the foot and toes. In six weeks the patient had recovered the power of most of the leg movements. Another patient, who was in the Infirmary for Nervous Diseases, was totally paraplegic. There was loss of power even in the adductors of the thighs. This little patient regained the control of nearly all the leg muscles, and walked quite well with crutches.

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[1] The Limitation of Therapeutics in Infantile Paralysis, New York Medical Journal, April 3, 1886.

[2] American Journal of the Medical Sciences, April, 1875.

Treatment.—The treatment may be divided into that which is suitable for the initial stage and that for the chronic period. At the onset of the attack the symptoms should be examined closely, and everything that might tend to produce reflex irritation should receive attention. If the gums are swollen and tender, they should be lanced. If there is any reason to suspect the presence of worms in the intestinal canal, a vermifuge should be administered. I have seen two or three cases in which a dose of santonine and calomel expelled several lumbricoids and speedy amelioration of the paralysis followed. If the fever is high, means should be used to reduce it.

Leeches may be applied over the spine at the cervical and lumbar enlargements of the cord. Counter-irritation, in the form of mustard plasters or tincture of iodine, is advisable. The application of the spinal ice-bag is a means that is to be strongly recommended. Medicinally, ergot is the remedy which theoretically should be of value, and its use is often followed by improvement. It is best given to children in the form of the fluid extract by the mouth, or ergotine may be given in suppositories. Ergotine has been advised hypodermically in this disease, but it is excessively painful in this way, and should not be administered except in cases where the patient cannot swallow and the bowel is unretentive. Ergot should be given in large doses as long as the stomach will tolerate it. Hammond recommends the fluid extract in doses of ten drops three times a day for infants of six months, and half a drachm for children between one and two years. Belladonna has been advised, but its value is doubtful. Mercury and iodide of potassium have also been employed, but they are not adapted to the acute stage. If there is any evidence of meningitis as a complication, these remedies are indicated.

The treatment for the chronic stage consists in remedies to restore power to the paralyzed muscles and in the use of means to prevent and correct deformities. The most important of the former are electricity and massage. Electricity may be begun in one week from the onset of the paralysis, provided that there is no fever nor muscular hyperæsthesia. Until after the third or fourth week, only the mildest currents should be used, as stronger currents might cause increase of the disease in the cord. Erb recommends central galvanization at this stage. It is given by placing the positive pole (anode) over the spine at the seat of the lesion and the negative pole (kathode) at the epigastrium. It has also been advised to use galvanism to the cord. It is extremely doubtful, however, if either of these methods is of any value. In using electricity to the muscles it should first be determined which current to use. This will depend upon whether the muscles respond to the faradic current. If they do, then this current should be selected. A cardinal point in the administration of electricity is to use the least amount that will cause a muscular contraction. The slowly-interrupted current is preferable, because a greater amount of contraction can be obtained in this way with less pain. The application should be of short duration; each muscle may be made to contract three or four times. The treatment should be used daily. If the muscles fail to respond to the faradic current, galvanism should be employed. A great obstacle to the use of this form of electricity is the pain it causes. Children are much more intolerant of galvanism than of faradism. If the former is used, it is impracticable to use a current strong enough to produce muscular contractions. We must content ourselves with a current of moderate intensity. The best way to use it is to apply the anode, which should be large and well wetted, over the nerve-trunk, and stroke the kathode firmly over the surface of the entire limb. In children, and indeed in adults, it is best to begin the use of galvanism by simply applying the wet sponges to the limb affected, without any current, going through the manipulations, however, as if there really was a current. The next day one or two cells may be used, and after this the current should be gradually increased from day to day. Static electricity has sometimes been used advantageously when neither faradism nor galvanism has been of avail.

Massage is of the greatest value in infantile spinal paralysis. Not only does it keep up the nutrition of the muscles and reduce the wasting, but it assists in relaxing the shortened and contracted muscles and tendons. Massage should be given by a person skilled in the employment of it, and at the same time that the massage is used the contracted muscles should be thoroughly stretched. Care should be taken not to allow the application of massage to be kept up too long. It is best to have the application short, but used daily. The length of each application will depend, of course, on the extent of the paralysis. If only one leg is paralyzed, ten minutes is long enough. In some cases the tension of the contracted tendons is so great that massage and stretching will not overcome it, and then tenotomy is necessary; but in a much larger number of cases than is generally believed, faithful and thorough massage will do as much as tenotomy. Shortening will occur after the division of tendons if care is not used to keep them stretched. It is very necessary to overcome contractures as early as possible, for all efforts to restore muscular tone in a paralyzed muscle will be nugatory if it is kept stretched by the contraction of its opponent. Muscles which have no faradic irritability while stretched in this way will often regain it when the rigidity of their antagonists has been overcome.

The tendons about the foot most often require tenotomy, especially the tendo Achillis and the plantar fascia. After tenotomy an apparatus is needed, and manipulation should be kept up as long as there is a tendency for contraction of the tendon to recur. Dr. V. P. Gibney (*loc. cit.*) speaks of an operation for shortening an over-stretched tendon, as, for example, in calcaneus when the tendo Achillis is much lengthened. The operation was devised by Mr. Alfred Willett, of St. Bartholomew's Hospital, and consists in excising a portion of the tendon and suturing the ends with the surrounding tissues. In the use of massage it is often suggested by the friends of the patient that various applications be made; but it is best to manipulate with the bare hand. A little vaseline or oil may be used to facilitate the movements and to prevent irritation of the skin, but a good masseur will rub better with the uncovered hand and never cause irritation of the skin.

The paralyzed limbs should be kept warm. In winter the temperature of the part is greatly lowered. It is well to keep the limb constantly covered with silk or woollen material, or with chamois-skin. The application of dry heat to the paralyzed part is useful in connection with other means. It has been advised in the case of a paralyzed leg to put the limb through a hole in a newspaper and toast it before an open fire. On putting the child to bed at night it is a good plan to use brisk friction for a few minutes over the affected parts, and then envelop them in flannel.

Medicines are of little value in the chronic stage. Strychnine has been recommended both per os and hypodermically, but it has never been proved to be of use. If the general condition is poor, cod-liver oil or the hypophosphites are indicated; but the general health in infantile paralysis is usually excellent.

It is of prime importance to give the paralyzed parts as much use as possible. If the lower extremities are affected, an effort should be made to have the child walk as early as possible. To accomplish this, apparatus of some kind is needed. The lightest form of instrument should always be used. Theoretically, the use of elastic bands to supply the place of the paralyzed muscles should aid the patient in walking; practically, the chief use of the apparatus is to support the joints. Care should be taken to have the steel rods thoroughly padded at the joints, so as to avoid friction and the formation of callosities. A well-fitting apparatus should not make pressure at a joint. If all the leg and thigh muscles are powerless, the apparatus should have a lock or catch at the knee, so that in standing the joint shall be rigid, and in sitting the catch can be released, to allow flexion of the knee. It is remarkable how well a child who is paralyzed in both legs can walk with apparatus. To aid a child in walking with apparatus some form of crutch is needed. For several years the Darrach wheel-crutch has been used at the Philadelphia Infirmary for Nervous Diseases, and has proved most satisfactory. It is simply a light frame with wheels which turn readily in any direction; the top of the framework is padded where it fits under the arms, and there is a piece of covered metal for the hands to hold. The use of such apparatus enables the unaffected muscles to be exercised, and those enfeebled are brought into play. Another important use of apparatus is the prevention of deformities, and it is with this object in view that the early application of the instruments should be made. Of course care must be taken to keep the apparatus in repair and fitting properly. The child is constantly outgrowing them, and parents are often careless about attention to the repairs. As a rule, the instruments need not be worn at night; but if there is much tendency to contraction of the tendons a lighter form of apparatus should be applied when the patient goes to bed. There is no indication for the application of apparatus in paralysis of the upper extremities. An apparatus has been recommended in cases of wrist-drop from paralysis of the extensors, but it is not of any practical value in children. A sling or straps to support the arm when there is paralysis of the deltoid and shoulder muscles are of real utility, for they prevent the weight of the arm from dragging the head of the humerus out of the glenoid cavity, and the stretching of the deltoid is obviated.

Gowers warns us to be careful about exposure to cold in cases where the back muscles are weak. In such cases the respiratory muscles are feeble, and, "although normal breathing may not be impaired, the diminution in strength may render an acute bronchial catarrh rapidly fatal, and this even months after the onset of the paralysis."

In the treatment of these cases the utmost patience and perseverance are requisite on the part of the parents and the physician. If "eternal vigilance is the price of liberty," so are persistence and long-continued treatment and care the price of improvement in this disease. The parents of the patient must be made to realize that the treatment will have to be kept up for months or even for years. Constant attention will be required to prevent deformities, for the more the child walks the greater will be the tendency to deformities from contraction and relaxation of ligaments and tendons about the joints.

HEREDITARY ATAXIA AND LOCOMOTOR ATAXIA.

By CHARLES L. DANA, M.D.

FRIEDREICH'S ATAXIA.

Synonymes.—Hereditary ataxia, Friedreich's disease, Family ataxia, Generic ataxia.

Definition.—Friedreich's ataxia is a chronic degenerative disease, affecting the several long-fibre systems of the spinal cord, especially that of the posterior column, and later those of the lateral columns. It begins in the lumbar region and extends upward and downward, finally involving the medulla and especially the nuclei of the hypoglossal nerve. It develops in childhood, affecting persons with an imperfectly-developed spinal cord the result of a neurotic inheritance. Clinically the disease is characterized by ataxia beginning in the lower limbs and gradually involving the upper limbs and the organs of speech. Curvature of the spine, talipes, vertigo, and, finally, paralysis and contractures appear. The knee-jerk is, as a rule, absent. There is but little pain or anæsthesia, and optic atrophy and visceral troubles are usually absent.

History.—The disease was first described in 1861 by Friedreich, who reported six cases. Carre reported one case in 1865; Bradbury one case and Carpenter two cases in 1871; Kellogg two cases in 1875; Dreschfeld three cases in 1876; Friedreich three more cases in 1877; Kahler and Pick, Schmidt, Seeligmüller, Hollis, and Gowers reported eleven more cases; so that by the end of the year 1880 thirty-one cases had been reported. The first Italian cases were reported by Musso in 1884. American cases began to be recorded rapidly after Dr. W. E. Smith had reported his cases in 1885. Up to the present time one hundred and sixty-five cases have been put on record in greater or less detail.

There have been fourteen German observers and thirty-three cases, fifteen English and thirty-six cases, six Italian and twenty cases, eight French and sixteen cases, seventeen American and fifty-six cases. A few cases of Swiss and other nationalities have also been reported. American physicians have, as may be seen, been particularly active in studying the peculiar disorder under consideration.

Dr. Kellogg was among the first to observe the disease. Dr. W. Everett Smith not only added to its literature, but also contributed the results of a post-mortem study upon one case. In particular, Dr. J. P. Crozer Griffith has worked up the subject in great detail and with the most praiseworthy care. His monograph upon Friedreich's ataxia is the latest and by far the most complete analysis of the subject which has been published. An excellent monograph was also written by Dr. A. Brousse in 1882, and a valuable critical digest by Ormerod was published in *Brain* in 1884. Bury analyzed all previously published cases in 1886, and Vizioli did the same in the same year.

To all these writers I must express my indebtedness, but more particularly to Dr. Griffith. To his paper, published early in 1889 and containing an analysis of one hundred and forty-three cases, there can now be added a case published in this article by myself, a case of Dr. V. P. Gibney's,[1] three cases by Dr. E. E. Wells,[2] five cases by Tourette,[3] one of which had been previously reported by Blocq, three cases by J. M. Clarke,[4] one case by Dalché, of doubtful character,[5] two cases by Paul Blocq,[6] a case of Ladame's (*infra*), a second case of my own reported to the New York Neurological Society, January 11, 1890, and four cases reported by me for Dr. Rook, of Quincy, Illinois, at the same time: total, one hundred and sixty-five. Dr. P. Ladame has written a very good monograph on this subject in the *Revue Médicale de la Suisse Romande*, July, August, and November, 1889. Soca, in a Paris thesis, November, 1888, collects one hundred and sixty-five cases, but includes some which should not be classed as Friedreich's ataxia.

Etiology.—*Predisposing Causes: Heredity.*—The fundamental factor in predisposition is an inherited or connate lack of development of the spinal cord, more particularly of the columns of Goll and pyramidal tracts. This condition is inherited directly sometimes, but indirectly, as a rule; that is to say, the parents or other members of the family usually show simply a neurotic history, and it is in only a minority of cases that there is a history of ataxia in the direct line of ancestry.

The more frequent condition is this: the parents or grandparents have some neurosis, such as insanity, inebriety, or great nervous irritability; the ataxia occurring only in the children of one generation. Sometimes in a single family the uncles and nephews or cousins may be found to have the disease. Hence the name "family ataxia" used by some writers. There are a good many cases in which the parents were apparently perfectly sound and healthy: yet it is most probable that the sufferers from Friedreich's disease inherit a tendency to degenerative processes from some of their ancestors. This degenerative tendency may have been shown in those ancestors in a very slight degree. The parents rarely have locomotor ataxia, though this has been observed in a few cases. The children of locomotor ataxics do not have Friedreich's ataxia except in the very rarest instances, but this may be due in part to the fact that locomotor ataxia early establishes impotence.

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[1] New York Medical Record, October 1, 1887.

[2] Journal of the American Medical Association, 1888, p. 303.

[3] Iconographie de la Salpêtrière, vol. i.

[4] British Medical Journal, March 23, 1889.

[5] Le Progrès Médical, June 30, 1888.

[6] La France Médicale, April 28, 1888.

Syphilis in the parents is an element in some—perhaps in many—cases. Congenital syphilis lays a foundation for neuro-degenerative changes, just as does the acquired form. Habitual intemperance in parents undoubtedly is a factor sometimes; much more rarely consanguinity and tuberculosis act as predisposing causes of degeneration.

Race.—More cases have been observed in America than in any other country; while the fewest have been reported from France, despite the attention called to the subject by the French writers Brousse, Féré, Charcot, Blocq, and Tourette. I am inclined to think that America is rich in this form of degenerative disorder. Seventeen observers have reported fifty-six cases.

Age.—The disease develops at about the time of puberty, most cases occurring between the ages of six and fifteen years. It is not very rare, however, for symptoms to develop even in infancy, though some of the cases reported at this time were probably of a syphilitic character. In a given family the disease, as a rule, strikes the older members first, but the younger members are attacked at a relatively earlier age. Thus, the oldest child becomes ataxic at the age of sixteen, the youngest perhaps at the age of ten. The most typical time of development is a rather late one,—*i.e.*, after twelve years of age, as in Friedreich's original cases. The disease may come on after maturity: in such instances it is only the family history which will justify the diagnosis of Friedreich's ataxia. True locomotor ataxia may be present in the parents and Friedreich's ataxia in the children.

In American cases the age of development of the disease has been rather earlier than the average, with the exception of the cases reported by Wells.

Sex.—The male sex slightly predominates, its proportion being about sixty per cent. In America the female sex has, however, been more affected, the proportion being thirty-two to twenty-one.

Social Condition.—The patients, so far as reports give data, are the children of the laboring and agricultural classes. They have been found in the country oftener than in crowded cities.

Diathesis.—Nothing is known specifically regarding the temperament or diathesis of the families affected, except that nervousness and various neuroses were present.

Over-Productiveness.—Some weight has been attached to the fact that in many instances the family was a large one, and the excessive drain upon the mother was thought to be an element in causation. This is not the case, however. The ataxia appears in elder children, before any drain on maternal energies could have occurred; while very often the family is small. This is particularly the case with the French patients. In America one family of thirteen and one of nine children are reported. The remaining families were not large, so far as is known.

Exciting Causes.—Nursing at the mother's breast is thought to have tended to bring out the disease in one Italian family. But the most frequently reported excitants of the disease are the infectious fevers, particularly scarlet fever and diphtheria. Rheumatism, typhoid fever, variola, whooping-cough, and chorea are all said to have preceded the disease. In one of my own cases a severe blow on the head is credited by my patient with starting it up. In the other case it was preceded by whooping-cough. In most cases there is no known exciting cause.

Symptoms.—The patient first notices an uncertainty in the gait and some feebleness in the lower limbs. These symptoms gradually increase until they interfere seriously with progression and force him to leave off active work. With this there may be some slight pains or numbness in the lower limbs, and an examination will show, within a year or earlier, that the knee-jerk is gone. After five or six years the arms become affected with incoördination, and a little later bulbar symptoms, such as thick or scanning speech, and often nystagmus, appear. During this time the patient suffers little pain and has no trouble with the bladder or rectum. Vertigo and headache are often present. Dorsal flexion of the toes, talipes varus or some other form of club-foot, and lateral curvature of the spine are often observed. Oscillation of the head and choreiform or incoördinate movements of the extremities may develop. As the disease progresses, the legs become weaker, and finally paraplegia, with contractures and muscular wasting, sets in. The disease makes slow progress; often it remains almost at a stand-still for years, and the patients usually die of some intercurrent disease, such as phthisis or an infectious fever.

Among the rarely-observed symptoms are tremor, spasms, decreased electrical irritability, muscular atrophy, vaso-motor paresis, polyuria, glycosuria, anæsthesia, fibrillary tremor, choking attacks, ptyalism, strabismus, diplopia, blepharospasm, a slight degree of ptosis, sluggish pupils, tachycardia, profuse sweats, impotence, slight vesical incontinence, fragilitas ossium. Many of these symptoms are, however, exceptional and accidental.

The major and essential symptoms are (1) ataxia, beginning in the lower limbs and extending to the arms and tongue; (2) peculiar rolling, ataxic gait, ataxia gradually involving the arms; (3) disturbances of speech; (4) talipes and spinal curvatures; (5) gradual development of paraplegia; (6) loss of knee-jerk; (7) absence of cutaneous anæsthesia, of bladder-troubles, of eye-troubles except nystagmus, and of severe pains; (8) the development of the foregoing at about the time of puberty.

Analysis of Special Symptoms.—I have space only to go over a few of these.

Ataxia.—This is the fundamental and by far the most important symptom. By ataxia in its broadest sense we mean a loss of the power by which one recognizes the degree of (1) passive or (2) active movements of the limbs, (3) their position in space, and (4) the amount of resistance or weight applied to the muscles. Our cognizance of these different things depends upon afferent sensory nerves supplied to the joints, tendons, tendinous sheaths, and muscles. It depends in a very slight degree also upon the cutaneous sensory nerves.

(*a*) When there is loss of power to perceive the weight of objects and the tension of the muscles, the muscle-nerves are chiefly affected, and we have *muscular anæsthesia.* (*b*) When there is loss of power to appreciate the amount of tension in or contraction of muscle, and the relation of the limb-segments to each other, there is both a muscular and an articular anæsthesia, and we have a condition known as *static ataxia.* (*c*) When there is loss of power to determine the degree of active contraction of the muscles and the movements of the limbs, there is chiefly articular and tendinous anæsthesia and *locomotor ataxia.*

Now, in Friedreich's disease motor or locomotor ataxia is always present and very marked. Static ataxia, however, which calls more into play the muscular sense, is less marked. The patients, though walking with a most incoördinated gait, can often stand fairly well with the eyes closed. Finally, muscular anæsthesia, as tested by determining weights and the position of the limbs, is only moderately impaired, and sometimes is not involved at all.

In fine, then, the gait of patients suffering from Friedreich's disease is very incoördinate. The patient rolls and tumbles along like a drunken man. Yet he knows where his legs are, and he can tell differences in weights quite well.

A hitherto undescribed peculiarity of Friedreich's ataxia, at least as shown in my own patient, is that it involves even the trunk-muscles, so that there is considerable swaying of the body, even when seated.

This has been shown by a study of tracings of the head-movements when the patient is standing and when he is seated with closed eyes.

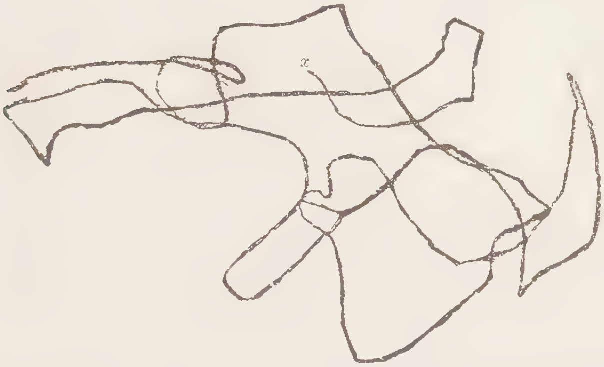
It is possible that this unusual degree of trunk-ataxia is due to the coincident involvement of the direct cerebellar tracts. I have observed it in a case of ataxic paraplegia in which the lateral columns were presumably involved.

Paralysis.—Paraplegia with contracture is a late symptom. Weakness of the lower limbs, with inability to walk far, is an early symptom. This inability is dependent largely upon the excessive incoördination and the consequent extra demand upon the nervous force in the act of locomotion.

The deep reflexes are generally lost early in the disease, and almost always in a year or two after it begins. The cutaneous reflexes are sometimes absent; but there is no diagnostic significance to be attached to them.

FIG. 1.

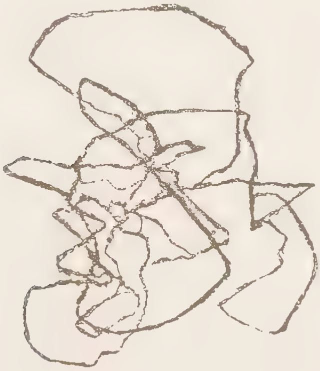
Anterior.



Ataxiagram-tracing in a case of Friedreich's disease. Patient standing, with eyes closed thirty seconds. x = beginning of tracing.

FIG. 2.

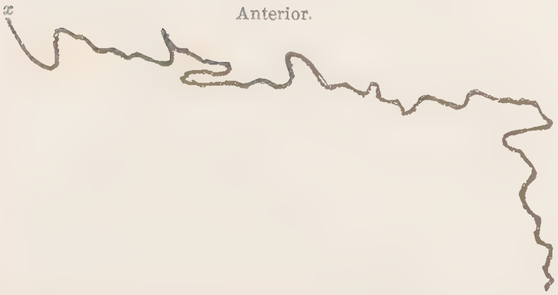
Anterior.



Same patient, sitting, with eyes closed.

FIG. 3.

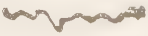
Anterior.



Ataxiagram of case of typical locomotor ataxia of seven years' standing; patient standing, eyes closed thirty seconds.

FIG. 4.

Anterior.



Same, sitting; eyes closed.

Sensory Symptoms.—Absence of pain and of any but light grades of anaesthesia is a characteristic of the disease. This absence of pain, however, is not absolute. There is almost always some complaint at various stages. The pains are not often severe and lightning-like, but are rather dull and myalgic.

The special senses are very rarely affected.

Speech usually becomes either syllabic or more often drawling, slow, and "thick." Vertigo and nystagmus conclude the list of most important symptoms.

Case of John Alexander Delahanty.—Age, eighteen years; single. U.S. Weaver. Admitted October 5, 1886. (See Photo. I.)

Family History.—Father died from alcoholism; mother died in confinement. Otherwise unknown or negative.

Previous History.—Patient was healthy as a child. Had rubeola, parotitis, pertussis, scarlatina, and diphtheria when about seven years of age. Has had intermittent fever several times. Denies syphilis and gonorrhœa. Has no alcoholic habits. Was in good health till about two years before admission.

Present Illness.—About five years ago, while at his work, he fell, striking on the back of his head. He was not rendered unconscious, and soon resumed his work. Previous to this he could walk and talk like other people. Soon he began to have dull and heavy sensations, was always tired and sleepy, and could be aroused from sleep only with difficulty. Then he had peculiar feelings in legs, pain about knees and ankles shooting upward. This was most severe in thighs. Had pains in lumbar region; constant and severe cephalalgia. Had formication in legs and back, especially about shoulders. This is less now. For about two years he had a band-like feeling of oppression over the stomach, and considerable nausea. This has now gone, but he vomits occasionally. When these symptoms became marked, his gait was so peculiar as to attract attention. He could not walk straight, but staggered like one intoxicated. This symptom persists. The floor and ground have always felt natural to him, but his ankles were unsteady, as if he were walking on skates. His speech has been peculiar. There is a sensation of the tongue being tied back, and he has difficulty in forming the desired word, this giving the speech a thickness. Formerly his words were natural. At the same time his face assumed a flushed appearance, that has persisted, at times being a dark red. Formerly he could walk readily; now the distance of a block is done with difficulty. He has no difficulty in urinating, but it is done frequently in excessive amount. At first he was habitually constipated, going four and five days without movement; now he is regular. Has much dizziness. He says that he has had diplopia and muscæ volitantes.

Physical Examination, May, 1889.—The patient is small of stature,—height, four feet ten inches. He is well nourished, not anæmic. Face is flushed, and hands are red and congested. His gait is rolling, staggering, like a drunken man's; he does not bring his feet down hard upon the heel, as in typical locomotor ataxia.

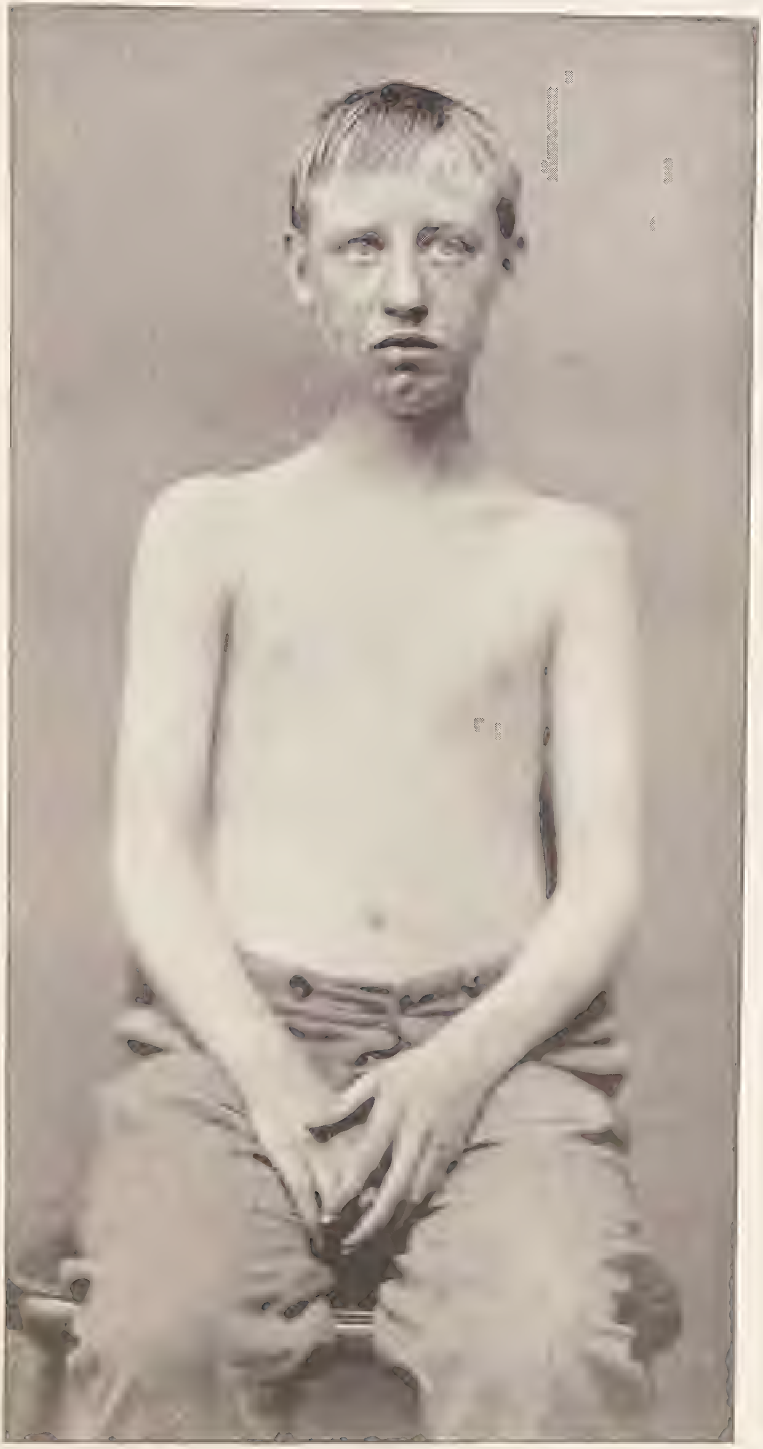
Heart normal, pulse slow and strong. Lungs normal. Liver-dulness normal. Digestive organs in fair condition; patient is inclined to constipation.

Urine of light specific gravity (1005 to 1010), and he passes one hundred and eighty to one hundred and ninety ounces daily. It contains no albumen or sugar, but the patient suffers greatly from thirst.

Mentally the boy is intelligent, but emotional and impressionable. His speech is thick, he speaks as though his mouth were full, but there is no "scanning" or syllabic utterance. He sleeps well; has sometimes headaches, and often attacks of vertigo. Muscular power is fairly good in the arms and legs. There is no atrophy. He has no choreic or tremulous movements, except that his head oscillates to and fro when he stands. He has no spasms or contractions.

There is a very slight tendency to flat-foot, and a slight anterior curvature of the lumbar spine, but no lateral curvature. Weight, one hundred and thirty pounds. The skin reflexes are diminished, the olecranon and patella reflexes normal; no clonus.

PHOTO I.



Case of Friedreich's Ataxia.

There was, on first examination, a slight amount of cutaneous anæsthesia of the lower limbs; later it disappeared, and he has had, if anything, cutaneous hyperæsthesia. His ataxia is very marked in the lower limbs, less in the upper. It is essentially a motor ataxia, being shown in locomotion and in using the arms and hands. In walking he staggers and rolls from side to side, and often falls if he is not careful. He can, however, stand fairly well with eyes closed. The degree of movement of the head is shown in the diagrams (Figs. 1–4). He has also trunk ataxia, as shown above. He knows the position of his limbs, and appreciates differences in weight.

His vision is good. My friend Prof. William O. Moore was kind enough to examine his eyes (February, 1889), and found nothing abnormal in the fundus. The late Dr. Loring, who examined him in 1887, said that he had then some nystagmus. I have never seen it. He has no disorder of the eye-muscles. His other special senses are normal.

Since he came under observation in 1886, the patient's gait has grown worse, but his general condition has improved. He has gained in weight, and his polyuria has nearly gone. He has often complained of pains in the head and back, and of vertigo, but has had no tightening or girdle pains. He has control of the bladder and rectum.

He has twice fallen, and broken first his fibula, and next his radius and ulna.

The features of special interest about this case are its origin from a blow on the head and the cerebellar ataxia shown in his gait, both facts suggestive of tumor. Early in the history of his symptoms there was considerable pain, though the patient always magnified his symptoms. He seems even to have had a girdle sensation. The presence of the knee-jerks, the extraordinary polyuria, the cutaneous vaso-motor paresis, were all somewhat peculiar though not unique symptoms. Fragility of the bones is a curious and interesting symptom, and it adds to the links between Friedreich's ataxia and the locomotor ataxia of adults.

Pathological Anatomy.—Fourteen autopsies have been made upon cases of Friedreich's ataxia, including one by Dr. J. P. Crozer Griffith, not yet published. The disease in most of the cases had lasted over ten years; in one, only two years; in two, eight years. The examinations have led to quite uniform results. The lesions of importance were found in the spinal cord and medulla only. The cord was usually small, flattened, and apparently congenitally imperfect in development. In some cases two central canals have been seen. A sclerosis exists through the whole length of the posterior and lateral columns, sometimes extending to the anterior columns. The sclerosis is most marked in the postero-median columns, which are always affected *in toto*. The postero-external column is less involved, and there is often a narrow strip of healthy tissue between the posterior horn and the sclerosed area, also between the posterior gray commissure and the diseased parts. The posterior column sclerosis is usually most marked in the lumbar region. In the lateral columns the sclerosis always affects the crossed pyramidal tracts. The direct cerebellar tracts and the so-called ascending antero-lateral tract are diseased in some cases, but apparently not in all. In a few instances the anterior median columns are involved. A zone of healthy tissue is often found between the sclerosed pyramidal tracts and the posterior horn. (See Plates I. and II.)

As to the gray matter, it often appears small in amount; there may be proliferation of cells around the central canal; the cells of the anterior and posterior horns and Clark's columns are at times few in number or somewhat atrophied. There is, however, no important or specific change in the gray matter.

Some chronic lepto-meningitis, especially on the posterior surface, has been noted.

The medulla shows some traces of extension of the sclerosis, but the involvement of the cells of the hypoglossal nucleus is probably the most significant change. The brain shows no changes of importance in relation to the symptomatology of the disease.

The posterior nerve-roots are extensively sclerosed, the anterior roots less so, and the peripheral nerves show some degenerative changes.

Pathology.—The disease is essentially a primary degenerative one. The persons affected by it are born with nerve-tracts which have not sufficient vitality to develop in accordance with the needs of the individual; consequently they atrophy, and connective tissue takes the place of nerve-fibre. This is the same process that takes place in locomotor ataxia of adults, which is a degenerative atrophy also, but involving only the posterior column, as a rule. The distinction between Friedreich's ataxia and true tabes dorsalis is etiological and clinical, but not a pathological one.[1]

A clinical peculiarity which especially distinguishes Friedreich's ataxia is the freedom from cutaneous anæsthesia, pain, and involvement of the organic spinal centres. The lesions show that cutaneous sensations must pass up either in the external part of the postero-external column or in the lateral column just external to the posterior horn. The absence of involvement of the sphincters may be due to the fact that the lowest part of the lumbar and the sacral cord are not excessively involved until late in the disease.

Diagnosis.—The distinguishing features of Friedreich's ataxia are the hereditary or family history, the age of the patient, the peculiar rolling ataxic gait, the absence of severe pain, of cutaneous anæsthesia, and of bladder or rectal troubles, the peculiar thick speech, the oscillations of the head, the spinal curvature, the talipes, and the absence of optic atrophy, or Argyll-Robertson pupil.

Disseminated sclerosis may counterfeit Friedreich's ataxia, but in the former there are exaggerated reflexes, spastic phenomena, tremor, paralysis, and often apoplectiform attacks.

My own case closely resembles cerebellar tumor, but is distinguished from it by its long course (five years), freedom from headache, from vomiting, and from eye-symptoms, involvement of the arms in the ataxia, oscillations of the head, thick speech, and fragility of the bones.

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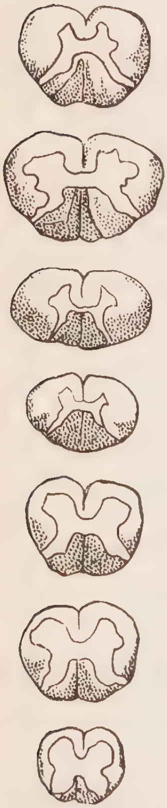
[1] Some assert (Blocq and Marinesceo) that Friedreich's ataxia is a primary degenerative disease of the spinal vessels, and that the nervous tissue is secondarily involved. All agree that the posterior columns and lateral tracts are first and most affected, because they are latest in being developed and are the most highly differentiated and specialized.

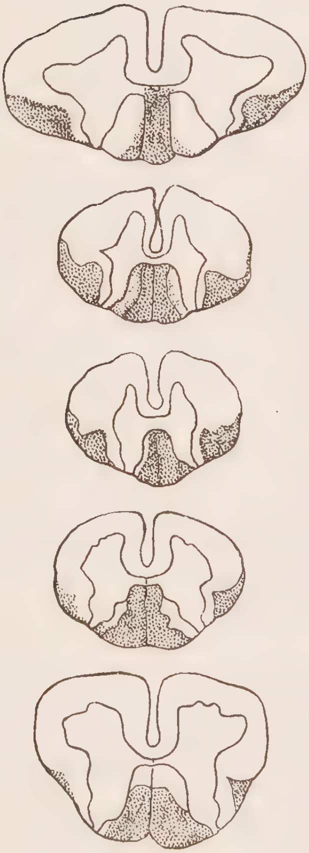
PLATE I.

Fig. 5.



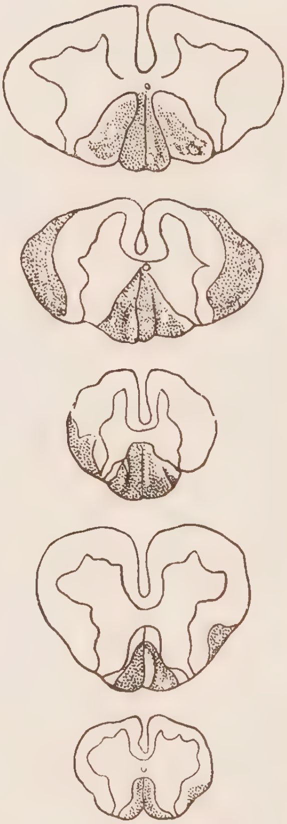
Friedreich's Case II., C. L., Virchow's Archiv, Bd. lxx. Duration of disease, eighteen years.

Fig. 6.



Case I., Rutimeyer and Quincke, Virchow's Archiv, Bd. xci. H. K., fifteen years' duration.

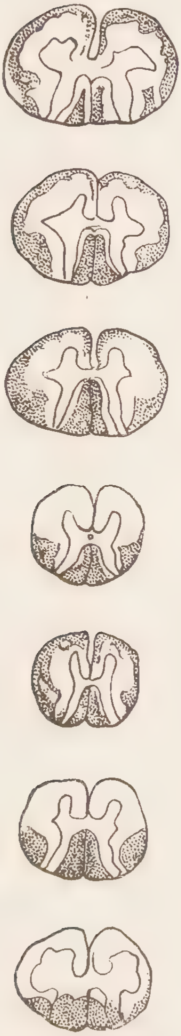
Fig. 7.



Case II., Rutimeyer, Virchow's Archiv, ibid. B. K., ten years' duration.

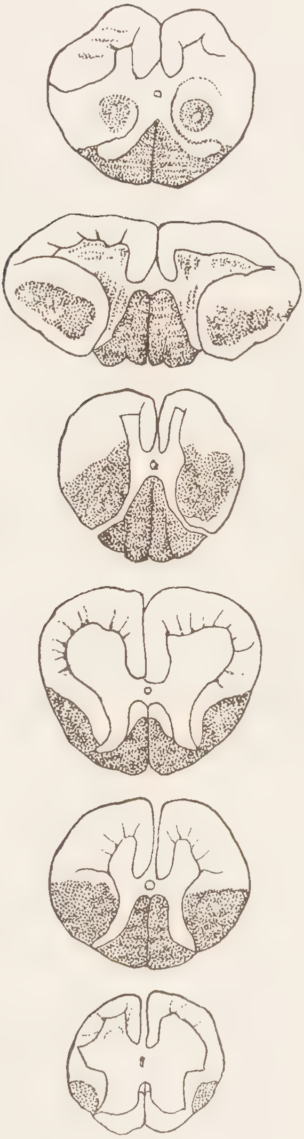
PLATE II.

Fig. 8.



Gowers and Pitt, Guy's Hosp. Reports. R. S., ten years' duration.

Fig. 9.



Erlitki and Ribalkin, Archiv f. Psych., 1886. J. W., two years' duration.

Duration and Prognosis.—The disease is a progressive one, though it may be stationary for a long time, and may even show temporary improvement. The longest period of duration of the disease on record is forty-six years, and the shortest two years, the average being fifteen or twenty years. Death occurs from some intercurrent disorder.

Treatment.—A quiet life, good food, and favorable hygienic surroundings are the main therapeutic helps. Arsenic and various nerve-tonics may be of temporary benefit. My case and some of the French cases were benefited by suspension by the neck in a Sayre's apparatus. If the disease appears in one member of a family, effort should be made to prevent its appearance in others. The infant should not be nursed by its mother; and special care should be taken to prevent it from getting any infectious fevers, and from receiving any falls or blows. Its life should be exceptionally quiet, so far as physical exertion goes.

HEREDITARY ATAXIC PARAPLEGIA IN CHILDREN.

There is a primary degenerative disorder of the spinal cord occurring in children and characterized by symptoms of ataxia and some cutaneous anæsthesia and spasm. The disease develops usually about the time of puberty. It progresses slowly, and takes upon itself the characters which I have described under the name "spastic ataxia," but to which the name ataxic paraplegia is more often given. Such cases may for a time be considered of functional or hysterical character. A longer acquaintance with them, however, reveals the progressive and organic character of the disease.

Hereditary ataxic paraplegia belongs to the same group as Friedreich's ataxia. It is a primary degenerative disorder, involving, no doubt, the lateral and posterior columns of the spinal cord, but with a preponderance of the lesion in the lateral columns.

Two cases of this disease were reported by me in the *Medical Record*, July 2, 1887. The following is a synopsis of the second case:

Female, aged nineteen; family history good; patient of nervous temperament; supposed hysterical attacks between ages of eight and ten; severe mental and emotional strain at sixteen; ataxia and anæsthesia, with spastic symptoms affecting different extremities for three years, but mainly lower limbs; temporary visual trouble; diplopia and vesical paresis; no neuralgic pains. When seen by the writer at the end of three years, there was spastic ataxia with cutaneous anæsthesia, mainly in left arm and right leg; some improvement, with shifting, of symptoms; mental depression; no historical crises.

In the three years subsequent to the above report the patient's condition has varied, but, on the whole, there is a progression of symptoms, and, while at first the disease was considered functional, I think that now there is no doubt of its organic character.

LOCOMOTOR ATAXIA IN CHILDREN.

The spinal cord of persons under the age of puberty is not subject to primary systemic degenerative changes. This is a general law to which, aside from the cases of Friedreich's ataxia, there are hardly any exceptions. Du Castel[1] has reported a case of primary sclerosis of the columns of Goll. There are several cases of amyotrophic lateral sclerosis in young children. Cases of lateral sclerosis are not rare, but they are always secondary, and dependent generally upon brain-lesions. About twenty cases of disseminated sclerosis are on record, but here such systemic degenerations as occur are also secondary.

The posterior columns alone seem sometimes to be primarily affected, as already indicated; but in almost all instances the disease takes the type known as Friedreich's ataxia, and it may be seriously doubted whether tabes dorsalis ever shows itself in young children in any other way than as hereditary syphilis or as the peculiar form known as degenerative or Friedreich's ataxia. Erb asserts, however, that he has seen three cases of locomotor ataxia in children between the ages of eleven and twenty. Eulenberg reports one case beginning at the age of nine; Leubuscher, one beginning at the age of three; Bradbury, one beginning at the age of nineteen; Remak describes three cases of tabes in children aged respectively nine, thirteen, and fifteen,[2] and refers to six others previously reported. Thomas Freyer reports three cases occurring under the age of ten.[3] Strümpell reports a case with tabetic and paralytic symptoms in a child of thirteen. Babez refers to five cases occurring between the ages of sixteen and twenty.[4] Jos. Eichberg reports a doubtful case of a girl aged fifteen, with optic atrophy and lightning pains.[5]

Gombault and Mallet have reported a doubtful case of tabes with paralysis developing in childhood.[6]

The histories of some of the cases of Friedreich's ataxia—such, for example, as Dreschfeld's and Seguin's cases—are more like those of cases of tabes dorsalis in the adult. Still, many of the foregoing cases are far from showing typical symptoms of locomotor ataxia, and some are probably instances of disseminated sclerosis, chronic myelitis, neuritis, or hereditary syphilis.

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[1] Gazette Médicale, 1874, No. 3.

[2] Berlin. Klin. Wochenschr., 1885, No. 5.

[3] Ibid., 1887, No. 7.

[4] Progrès Médical, 1888, Nos. 23, 24, 30.

[5] Cincinnati Lancet and Clinic, 1888, p. 354.

[6] Arch. de Méd. Expér. et d'Anat. Path., May, 1889.

Locomotor ataxia, therefore, when it occurs in children, takes, as a rule, the type of Friedreich's ataxia. So far as it diverges from that type it approaches in its symptomatology to the locomotor ataxia of adults. The patients then suffer from pains, anæsthesia, bladder-troubles, and visual disturbances. The etiology in such cases is often obscure. Sometimes there is evidence of direct heredity, as in Eichberg's case, or of injuries, or of rheumatic influences. Most often there is a history of hereditary syphilis.

STRUCTURAL ABNORMALITIES OF THE BRAIN AND CORD.

By SARAH J. McNUTT, M.D.,

AND

SARAH E. POST, M.D.

Abnormalities of the brain and cord may be divided into two classes, —namely, those of greater and those of lesser clinical importance. Among the latter may be placed anencephalus, cyclops, absence of the prosencephalon, and absence of the cerebellum,—gross defects which are, with the exception of the latter, incompatible with prolonged extra-uterine life.

Anencephalus is always associated with a considerable degree of cranial defect. Bauer has classified cases of anencephalus into those with and those without an occipital fossa, the vault of the cranium being absent in all cases. In a typical case there are present of the skull, at the most, only the lower portions of the occipital, temporal, sphenoidal, and frontal bones. The basis cranii is occupied by a mass of connective tissue and blood-vessels formed from ingrowths of the pia, constituting the pseudencephalon or fungus cerebri of the older writers. Rachischisis and amyelia are accompaniments of this condition.

Absence of the prosencephalon may be found with a complete skull. The cases of Starr[1] and of Dana[2] are still fresh in mind. Of Dana's case it is said that the skull was proportionately large and long in its antero-posterior diameter, and that the sutures were open and the bones freely movable. Both of these children were born at term and lived a number of days. A rudimentary thalamencephalon was present in both cases. The cerebellum, medulla, and pons were reduced in size, but otherwise normal, the difference in size being apparently due to absence of the fibres which depend upon the prosencephalon for development. The main interest of these cases has been found in the complete elimination of the pyramidal fibres and the consequent simplification of problems connected with these and other spinal and intracerebral tracts.

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[1] Journal of Nervous and Mental Diseases, July, 1886.

[2] Ibid., January, 1888.

Cyclops is characterized by an undivided anterior cerebral vesicle, which is occupied by but a single ventricle, and in marked cases gives rise to but a single optic nerve and a single eye. The malformation of the eyes is, however, but one of a series of defects to be found in these cases, as there are also imperfect development of the other parts derived from the anterior cerebral vesicle, and extensive changes in the bones of the skull and face. The olfactory nerves are absent, and the nose is but a rudimentary organ. The deformity of cyclops may exist in the case of an otherwise well-formed child. In a case recently described by William Craig, of Edinburgh,[1] the child was otherwise well developed and lived for twenty minutes after birth. Cases partaking partially of the nature of the cyclops have lived to a considerable age. In a case reported by Richter the subject was a male who died of phthisis at the age of nineteen years. The legs were completely paralytic, there was but slight use of the arms, and the intelligence was of low grade. When younger the patient is said to have been able to talk, but later he became stupid and apathetic. Upon autopsy the posterior third of the corpus callosum was found present, but there had been no further division into hemispheres. The optic nerves were well developed.

Another case is described by Hadlich,[2] in a child living but eight days, where, with hare-lip and other deformities, the surface of the brain was furrowed between the frontal lobes, but showed no further effort at division. There were no traces of the corpus callosum in this case. A similar case has been reported by Wille.[3] Difficulty in nourishing the child has appeared to be the direct cause of death in these cases. An interesting point connected with the condition is the fact that cortical convolutions develop independently of a corpus callosum or other transverse commissure.

A trilobular brain has been reported by Heydenreich,[4] from the St. Petersburg Findelhaus, the middle lobe being occupied by the third ventricle. There was no trace of ossification in the skull, yet the child lived a number of days. One other similar case had been reported from the same institution in 1850, the total number of inmates during the interval having been two hundred and thirty-nine thousand eight hundred and twenty-eight. Geoffroy-Saint-Hilaire mentions a similar case. Double brain, or four hemispheres, is perhaps less infrequent. A recent case has been reported from Italy by Martinotti and Sperino.[5]

In all cases of this class the deformity is supposed to date from a very early stage of intra-uterine life. According to Richter,[6] the deformity of cyclops has its origin before the eighteenth day.

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[1] Edinburgh Medical Journal, vol. xxxii. No. 3.

[2] Hadlich, Arch. f. Psych., 1880, Bd. x. p. 97.

[3] Wille, Arch. f. Psych., 1880, Bd. x. p. 97.

[4] Heydenreich, Virchow's Archiv, iv., 1885, Bd. c. p. 24.

[5] Internat. Monatschr. f. Anat. u. Phys., 1888, p. 11.

[6] Virchow's Archiv, Bd. cvi. p. 390.

Kölliker tells us that the closure of the medullary canal begins a little caudad to the head and proceeds in both directions. This observation is cited as a reason for the fact that in these early deformities the sacral region and the prosencephalon suffer so severely, the part last developed being that which is most apt to be malformed.

The remaining conditions which we have to consider are not inconsistent with a somewhat prolonged extra-uterine life. This class may be subdivided as follows:

1. Abnormalities accompanied by defects in the envelopes of the part:
   - *a.* Cranioschisis,
     - Encephalocele.
     - Hydrencephalocele.
     - Meningocele.
   - *b.* Rachischisis,
     - Myelocele.
     - Meningocele spinalis.
     - Spina bifida occulta.
2. Abnormalities in which the envelopes are entire:
   - Hydrocephalus.
   - Microcephalus.
   - Porencephalus.
   - Aberrant arrangement of fissures and convolutions.
   - Agenesis of cortical elements, commissures, and associative tracts.

Encephalocele is characterized by protrusion or hernia of a part of the brain through an opening in the skull. According to Bruns, cerebral hernias are, with few exceptions, located in the median line of the skull. The majority are found at the posterior extremity of this line,—namely, at the occipital protuberance,—extending upward to the posterior fontanel or downward to the occipital foramen. Much less often the aperture is at the anterior extremity, between the cribriform plate of the ethmoid and the frontal bone or between the halves of the frontal bone at its lower and anterior portion. Exceptional cases are found in which the opening is between the halves of the frontal bone at its upper portion, the anterior fontanel, and the sagittal suture. A few cases have been observed in which it was between the temporal and parietal bones.

Congenital cerebral hernias are found between bones, not, as a rule, through openings or foramina in the substance of the bones. There are but few exceptions. The occurrence of such a hernia in the middle of the occipital bone is no exception, because at a certain period of development the halves of this bone are separate. Hernia outside of the median line is so rare that only a single case has come to the writer's notice.

Encephalocele appears as a round or egg-shaped tumor covered with skin, unaltered if the tumor is small, but destitute of hair in those of larger growth. The tumor of encephalocele sometimes shows a furrow dividing it into lateral halves, varying in size from that of a nut to that of a man's fist. In the true encephalocele this tumor is pretty firm. As it contains more water it fluctuates. Its volume is diminished by pressure during deep inspiration and during sleep, while it is increased in volume during expiration and cries. It is semi-transparent when containing water. Impairment of function is not usual where the tumor is small and not disturbed. When disturbed, restlessness, cries, and convulsions follow. Pressure on the tumor produces turning or fixing of the eyes, difficult respiration, weakening of the pulse, nausea, convulsions, and a comatose condition. Exceptionally no change occurs in the tumor, but usually it becomes larger, and the more considerable its size at the time of birth the more rapid is its growth. Most of the cases die within a few weeks of birth, from progressive hydrocephalus or diffuse inflammation of the membranes from the distention of the portions over the tumor. A few cases have, however, lived twenty or thirty years, without functional disturbances.

Meningocele is characterized by the escape of the arachnoid adhering to the dura through an opening in the skull, so that there is formed another vesicle which communicates with the cavity of the arachnoid by a narrow mouth. This vesicle forms a tumor which is filled with fluid and presents a semi-translucent appearance. The case figured below is one exhibited by Dr. McNutt before the Clinical Society of the New York Postgraduate Medical School, April 16, 1887. The history of this case is briefly as follows:

FIG. 1.

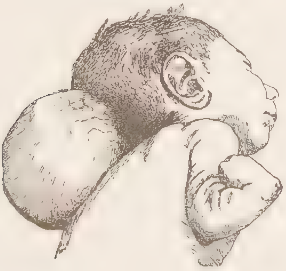
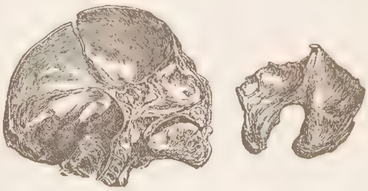


FIG. 2.



The patient was a male, the first child, born at term, of young, healthy parents. There was a history of privation, anxiety, and insufficient food during the pregnancy. The delivery was normal. The tumor was observed immediately after birth. It appeared in the occipital region. The sac of the tumor was, about the convexity, membranous and translucent. Towards the base it was covered with hair. The bones of the skull were soft, the fontanels large, and the sutures open. Left facial paralysis, shoulder-luxation, talipes, and deformities of the wrists and hands were present. The child lived two months. At the time of death the tumor measured nine and three-fourths inches in circumference. The pedicle of the tumor was thick and contained cerebral tissue. The occipital convolutions interlocked, and the cerebellum was absent. A further detailed report of the microscopical conditions in this specimen is being prepared. The bony defect extends from the level of the inferior curved line into the occipital foramen.

A vertical section through the skull in the median line is shown in Fig. 2, with the segment of the occipital bone surrounding the defect, which was disarticulated and removed with the tumor. We shall again refer to this drawing, made from a photograph of the specimen.

Myelocele, or hernia of the cord, is one of the conditions commonly designated as spina bifida. As described by Koch,[1] it is found most frequently in the sacral region. The spine down to the sacrum is normally closed, but the posterior segments of the arches of the three upper sacral vertebræ are lacking, leaving a four-cornered fissure. The number of the vertebræ is normal and the direction of the laminæ correct; they simply do not quite meet. The borders of the fissure are covered with the periosteum of the spinal canal, which blends with the lumbo-dorsal fascia. The dura forms the sac of the tumor.

Normally the cord in the new-born ends at the level of the second lumbar vertebra, the cord being retracted to this level, according to Ranke, with the later growth of the bony canal. In myelocele at the level of the first sacral or the last lumbar the cord is adherent to the dura, the fascia, and the skin, and retraction is thus apparently hindered, because if the cord in a case of myelocele be extended it will reach the level of the third or fourth sacral vertebra. Lumbar, sacral, and coccygeal nerves are found in the tumor. Where the cord is adherent the membrane covering it is often thin and transparent, appearing to have the character of primitive epiblastic tissue. Hydromyelius is present, as a rule, to a greater or less degree in all cases.

Meningocele spinalis consists of a hernial sac formed by the dura externally and lined by the pia. The spinal cord does not take part in the formation of this tumor, but lies even more deeply than usual in the lumen of the spinal canal, or, at most, sends only a few fine nerve-trunks over the tumor. The classic location for meningocele is the occiput. According to Koch (*loc. cit.*), its next most frequent location is coccygeal, at the lower extremity of the sacral vertebræ, the pedicle appearing through the hiatus sacralis. Absorption of the neighboring vertebral segments may occur from pressure, but a true bony defect is not necessary to the formation of this tumor. The diagnosis is obtained from the transparency of the tumor, its fluctuation, its rapid development, and the production of cerebral symptoms when pressure is exerted upon it. The myelocele is, on the contrary, a small, firm, and usually umbilicated tumor, always associated with bony defect.

Spina bifida, or rachischisis posterior, is characterized simply by absence of a larger or smaller portion of the vertebral arch. In total rachischisis the whole canal lies open, the free borders pointing outward. The cord is only partially developed. Cases of anencephalus and occipital encephalocele are allied to this class.

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[1] Beiträge zur Lehre der Spina Bifida.

In partial rachischisis the defect is limited to one region, or even to a single vertebra. According to Koch (*loc. cit.*), the vertebræ are normally formed up to their transverse processes. The intervertebral foramen and the interarticular ligaments also are little altered; but beyond the transverse processes the arch, instead of continuing backward and inward, proceeds in an outward direction.

In total rachischisis the whole number of vertebræ is under the normal; the bodies of the defective vertebræ are smaller than normal, or may be fissured, giving the variety known as rachischisis anterior. With this malformation the whole cord may be defective, absent, or represented by embryonal tissue, or it may appear as a fissured expanse. In other cases, where the medullary plate has united to form the medullary tube, the spinal nerves take their origin irregularly, often being in connection with the membranes only and totally detached from the cord.

Spina bifida occulta is a minor allied condition discovered by Virchow and since described in a number of cases.[1] The defect here is in the lumbar region, it is covered with skin, and hypertrichosis of the part is characteristic of all cases. There have been uniformly present also club-foot, a degree of anæsthesia, and perforating ulcer of the foot. These two varieties of rachischisis were observed by Dareste in his studies upon the artificial production of deformities, and were referred to by him before the Academy in 1877. In one, all the tissues from the laminæ outward were wanting in the median line; in the other, bone only was wanting, the skin and subcutaneous tissues being formed.

With a high grade of rachischisis, ventral hernia, imperforate pharynx and rectum, etc., usually are present. J. Bland Sutton finds the origin of this coincidence in the primitive neurenteric canal, the neural canal and the alimentary canal being continuous at a very early date, and hence conditioned by the same lack of formative force.

The older writers regarded meningeal effusion as the primary etiological factor in the production of cerebro-spinal deformities. In the class of cases which we have just considered it was supposed that the envelopes rupture; in the class yet to be considered the envelopes resist the excentric pressure, and embarrassed growth is the result. The more recent literature has not, however, given to meningeal effusion the same prominent place. Dareste and, later, Lebedev,[2] from studies of the chick embryo, Ackermann,[3] from a study of the crania associated with hernia of the brain, and William Koch,[4] from a study of spina bifida, arrive at a common conclusion,—namely, that bony defects at least do not necessarily depend upon hydro-

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[1] Conrad Brunner, Virchow's Archiv, Bd. cvii. p. 494.

[2] Virchow's Archiv, Bd. lxxxvi. p. 263.

[3] Die Schädeldifformität bei der Encephalocele Congenita.

[4] Beiträge zur Lehre der Spina Bifida.

cephaloid conditions. Koch considers the bony defect in myelocele and encephalocele alike due to failure of separation between the cutaneous and the medullary layer after the formation of the medullary canal. A septum thus continues to unite the neural tube with the skin and prevents union between the segments of the osseous and muscular layers. These finally, by increasing in thickness, may exert traction upon the neural organ and lift it from its bed in the canal. Increase in size, gravity, and subsequent meningeal effusion complete the work thus begun. We have already referred to the fact that encephalocele and myelocele are apt to be umbilicated or furrowed. The place of umbilication or furrowing is supposed to locate the septum and primitive defect.

Koch finds the shortening of the spinal column, the absence of some vertebræ, and the dwarfing of others in pronounced cases of rachischisis, also the occurrence of minor degrees of rachischisis without neutral involvement, all opposed to the theory of rupture from distention. In hydromyelius he would expect the spinal axis to be lengthened rather than curtailed.

Ackermann cites further the condition of the crania in cases of encephalocele as evidence opposed to the theory of rupture by the distention of a hydrocephalic sac. The peculiarity of these crania is that known as kyphosis[1] of the base of the skull. In this deformity there is steepness of inclination of the whole base of the skull, and, in addition, sharpening of the occipito-sphenoidal angle, or the angle formed between the basilar process of the occipital bone and the body of the sphenoid. In the five skulls examined by Ackermann the angle made by the basilar process of the occipital bone with the plane of the occipital foramen was diminished from 146 degrees to 112, 104, 100, 119, and 132; and the angle made by the basilar process of the occipital bone with the body of the sphenoid, from 151 to 133, 130, 129, 144, and 143.5. Prognathismus, with its receding forehead and diminished cranial capacity, accompanies this basilar deformity. In cases of hydrocephalus the first angle was little altered; the second was, however, found flattened in all of six cases. In hydrencephalocele also this angle was, as in hydrocephalus, increased to 157, 175, and 178 degrees in three cases. Ackermann finds kyphosis an argument in favor of a primitive bony defect with a resulting diminished intracerebral pressure. Normally, he explains, intracerebral pressure must somewhat exceed that of the surrounding medium. With a bony defect this balance of pressure will be lost, and crushing together of the immature bones will result. In hydrocephalus, on the contrary, the intracerebral pressure is supposed to be increased, and flattening of the basal angles is a consequence.

Fig. 2, representing a vertical section of the skull in Dr. McNutt's case of meningocele, is manifestly an example of this deformity. Further observations not only upon the brain but also upon the skull in these cases are to be desired. The practical importance of the investigation is easily seen. If prenatal hydrocephalus leaves its impression upon the base of the skull, this base may be interrogated in regard to a number of disputed points.

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[1] Post, Basilar Kyphosis, New York Medical Record, December 12, 1889.

To construct the angles referred to by Ackermann, first divide the skull vertically in the median plane, then upon this plane draw lines representing its intersection with the plane of the occipital foramen, with that of the longitudinal axis of the basilar process of the occipital bone, and with that of the longitudinal axis of the body of the sphenoid. The two latter extend midway between the inner and outer surfaces of the bones, the inequalities of these surfaces being disregarded. We present Ackermann's observations at some length, on account of their interest and apparent value, also as being in their conclusions harmonious with the more recent experimental results.

While Koch, Lebedev, and others make meningeal effusion secondary to a primitive medullary defect, Sutton, on the other hand, ascribes the tumor of occipital meningocele to primary closure of the canals connecting the lateral recesses of the fourth ventricle with the subarachnoid space. It is associated with a rudimentary or absent cerebellum if developed at an early stage. This variety of meningocele has the same relation to true meningocele that syringomyelocele has to spinal meningocele. Hydrencephalocele would be produced, according to Sutton, by dilatation of the central canal and the mechanical withdrawal of cerebral tissue, the agent being the traction exerted by the growing tumor upon the adjacent cerebral mass; Bruns thought that a meningocele became a hydrencephalocele by the going over of cerebral substance into the tumor under the combined influences of growth and the cranial defect; while Koch, on the contrary, holds that the primary encephalocele becomes a hydrencephalocele by the loosening of the band of connection between the neural tube and the skin and the consequent sinking back of the hernia into the canal. The question as to which existed first, the tumor or the defect, and the question whether the meningeal effusions are primary or secondary, cannot thus be said to have yet received a completely decisive answer.

Hydrocephalus may be internal or external. Internal hydrocephalus is characterized by effusion into the ventricles. In external hydrocephalus the fluid is found in the subarachnoid space. Hydrocephalus may be due to stasis of the circulation, meningitis, shrinkage of the brain, or congenital rachitis. In hydrocephalus due to inflammatory action the ependyma is found thickened, red, and granular.

Microcephalus is characterized by narrowing of the brain in all its diameters. Premature synostosis of the bones of the skull has been established by Virchow as a more or less constant concomitant of this condition. The convolutions are simple in arrangement, approaching, according to some authors, the cetacean type. In a case reported by Dr. Mary Putnam Jacobi,[1] in a child of three months, the skull measured but twelve and a half inches in its greatest circumference. There was effusion into the ventricles, but the ependyma was not injected. The convolutions were simple, little curved, narrow, and flattened. The occipital lobe was least developed. The corpus callosum was small and terminated "prematurely."

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[1] New York Medical Record, January 11, 1881.

Inoccipitie is a term used to distinguish microcephalic brains in which the defect in the occipital lobe is especially marked. This abnormality, according to Richter, dates from about the fourth month, at which time the frontal, parietal, and temporal lobes have been differentiated, but the occipital lobe has not yet been formed. Following Virchow, Richter finds the origin of occipital agenesis in premature occipital synostosis. Schroetter in conditions of idiocy found the corpus callosum abnormally short in one hundred and nineteen cases.[1]

Porencephalus is characterized by the presence of depressions or atrophied portions of the surface of the hemispheres. In some cases the pit communicates with the lateral ventricle. The surrounding convolutions then converge towards it, and the lesion is more profound and presumably of earlier origin. In the majority of cases the defect is unilateral; in others it is bilateral and even symmetrical. An example of bilateral symmetrical porencephalus is shown in Figs. 3–7. The clinical condition is one of paraplegia of cerebral origin, the development of the parts supplied by the atrophied cortex being impaired. In the case here referred to[2] there was a history of difficult labor, with an after-coming head. The child suffered with convulsions for nine days after birth. It lived two years. Voluntary motion never was obtained, the reflexes were exaggerated, and contractures were present in all the extremities, in the lower extremities the rigidity being most marked. The larynx also was badly developed, and the child had the crowing respiration and the tendency to regurgitation noted in similar cases. The cortical defect was found in the parts adjacent to the sulcus of Rolando and the præcentral sulcus, there being complete absence of gray matter in the bottom and along the sides of these sulci. The examination of this brain, made by Dr. William H. Welsh, shows results which are in some respects unique. We refer particularly to the escape of the crest of the convolutions from the atrophic process. Dr. Welsh[3] refers to this peculiarity as follows:

"The margin of sclerotic tissue can be traced up for a certain distance along the sides of the anterior central convolution, and then there appears rather abruptly a cortex two or three millimetres in thickness, which can be traced over the top of the convolution where it has its greatest thickness. The sclerotic tissue, however, does not disappear, as it can be traced along the deeper parts of this cortex near the junction of white and gray matter. At the top of the anterior central convolution can be made out, in normal succession and of about normal thickness, the molecular layer, the layer of small and the layer of large pyramids, and then comes the sclerotic tissue, with its abundant nuclei, fibrous texture, and dilated lymph-spaces. In following the cortex down the sides of the convolution it is seen that sclerosis invades from the deeper parts more and more of the cortex, the layer of large pyramids first disappearing, then that of small pyramids, while the molecular layer, although abnormally rich in nuclei, can be traced all the way down the sides of the convolution. The impression is not that of a sclerosis invading the cortex from the surface, but rather that of invasion from the deeper layers of the cortex or from the medullary substance."

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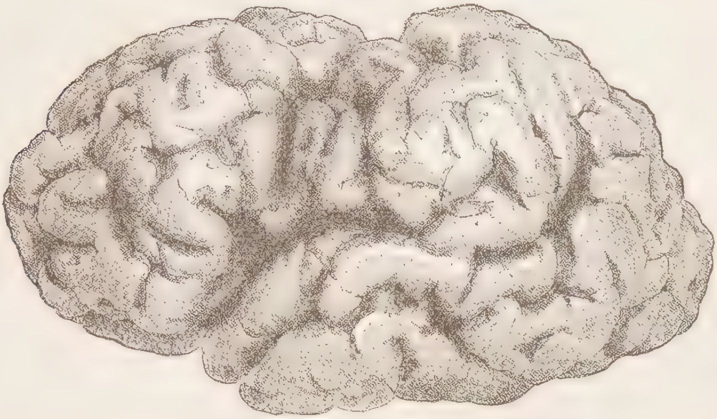
[1] Neurolog. Centralblatt, October, 1886.

[2] McNutt, American Journal of the Medical Sciences, January, 1886.

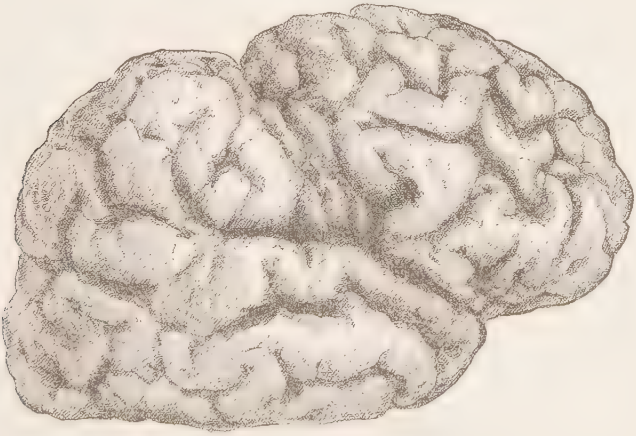
[3] Ibid., January, 1885.

Fig. 3.



Left hemisphere. Atrophy of the ascending frontal convolution, atrophy of the ascending parietal convolution, atrophy of the paracentral lobule, and possibly atrophy of the anterior part of the first temporal convolution. (About two-thirds actual size.)

Fig. 4.

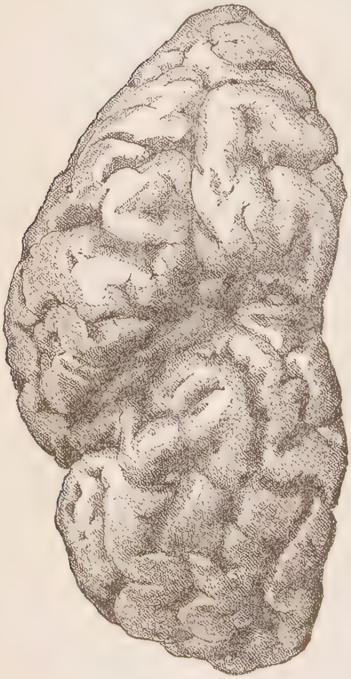


Right hemisphere. Atrophy of the ascending frontal convolution, atrophy of the ascending parietal convolution, and atrophy of the paracentral lobule. (About two-thirds actual size.)

Figs. 3, 4, 5, 6, and 7 are taken from this brain.

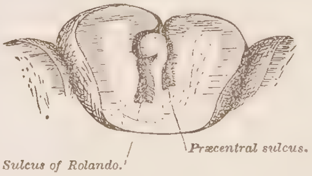
The lesion in these cases would appear to be identical with that of general cortical sclerosis, from which it often cannot be differentiated during life. Records of sixteen autopsies of spastic bilateral hemiplegia collected by Osler have shown five cases of general cortical sclerosis, seven cases of extensive partial sclerosis, including the central convolutions, and four cases only of sclerosis limited to this region. Paraplegia, rigidity, and idiocy were present in all, the essential lesion in every case being apparently the atrophy of the central convolutions. The disability in Dr. McNutt's case was as profound as in the cases where the lesion extended over a larger surface of the hemisphere.

Fig. 5.



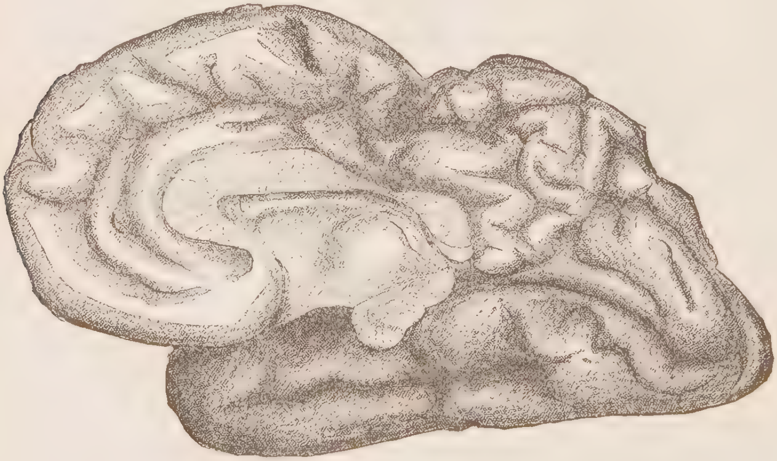
The right hemisphere viewed from above.

Fig. 6.



Vertical section showing atrophied præcentral convolution.

Fig. 7.



Internal aspect of the right hemisphere, showing the atrophied paracentral lobule with the atrophied band in the callosum.

The etiology of porencephalus has given rise to many interesting hypotheses. The hemiplegia with which it is associated has in many cases appeared after an acute illness characterized by fever, convulsions, and other evidences of disturbed cerebral action. In connection with this fact the production of symmetric atrophy of the cortical motor centres has suggested a systemic inflammatory affection,—the polienecephalitis of Strümpell, supposably analogous in character and development to anterior poliomyelitis. Satisfactory as it may appear, this theory has not been substantiated by post-mortem evidences, and must be said to be losing ground in the general estimate of this condition. In other cases, as in the one here referred to, the paralysis has dated from birth, and there has been a history of difficult labor, with convulsions following delivery. An hypothesis fitted to these cases has been meningeal hemorrhage at the time of birth. So probable does this etiology appear that Gowers has styled such double hemiplegias birth-palsies. Not all cases of even biplegia have, however, this history. In eight of the nineteen cases observed by Osler the labor was normal.

Richter's observations, already quoted, have included cases of porencephalus. In these cases he has divided the skull vertically upon each side of the longitudinal sinus, so as to leave the sinuses and falx entire. With such a section of the skull he claims to have unmistakably demonstrated kyphosis[1] of the base of the skull with undue projection of the sella turcica into the cranial cavity. To accommodate itself to this deformity the brain is more than usually inclined, and the corpus callosum comes into collision with the falx. The result is failure in the development of the restricted portion of the callosum and symmetric atrophy proceeding centrifugally in both directions. Pressure of the falx upon the venæ Galenæ produces circulatory disturbances and ventricular effusion. Symmetrical atrophy of the cortex is thus explained as primarily "tabes of the corpus callosum."

In the case of double porencephalus here referred to, unfortunately, the brain was removed so precipitately that even a considerable amount of basilar deformity might have escaped unnoticed. Excepting the post-natal convulsions, therefore, we have no evidence connected with the case which would disprove Richter's hypothesis; while the location of the atrophy of the callosum just in front of the splenium, the part which Richter finds the most frequently encroached upon by the basilar deformity, with the symmetry of the cortical lesion, would be in its favor.

A curious point in connection with this observation of Richter is the fact that he found the condition of kyphosis and collision with the falx in a case where the inception of the paralysis dated from the sixth year. His explanation is that the deformity was present, but not sufficient to interfere with the brain until it reached this stage of its development. The possibility of the development of basilar kyphosis after birth might also be suggested. There was delayed ossification of the intersphenoidal articulation in Dr. McNutt's case of meningocele, also in all of Ackermann's cases. The fact of persistent cartilaginous intersphenoidal union suggests rachitis as the possible origin of the deformity. In Dr. McNutt's case of biplegia, the base of the skull was not examined, but the vault was rachitic, and the child's whole condition had a rachitic aspect.

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[1] Post, loc. cit.

In regard to unilateral porencephalus, apparently interruption of the pyramidal tract in any part of its extent will result in a porus if the injury be received during the period of growth. Wigglesworth reports the case of a man dying at the age of fifty-six years in which the left upper extremity had been amputated at the age of four years. Upon autopsy there was found atrophy of the right post-central convolution. Also we are reminded that in Heubner's case of double porencephalus the symptoms dated from convulsions and fever at the age of fifteen months. An embolus was found in the right middle cerebral artery in this case. It is probable that basilar kyphosis is at the most but one of a number of conditions capable of producing this deformity.

From even this short and incomplete review of the recent literature of our subject it will be apparent perhaps that interest has tended to centre in the collateral etiological evidence to be gained by interrogation of the skull and spinal canal, upon the one hand, and, upon the other, in the revelations afforded by the natural atrophy method of agenesis. In the presence of such observations as those of Richter all doubt as to the commissure system of the corpus callosum should be, it would appear, permanently put to rest. The value of this natural atrophy method in demonstrating the cerebro-spinal fibres has also been referred to. An abnormal product is in the light of such investigations not a mere monstrosity to be immediately put out of sight, but rather a blunder by which Nature reveals the secrets of her workshop.

The arrangement of convolutions and the degree of development in the nerve-elements have received attention in connection with the brains of imbeciles and criminals, particularly from Spitzka,[1] Mills,[2] and Sachs.[3]

It is evident that in this line of investigation, also, there is a wide field for research.

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[1] American Journal of Neurology and Psychology, 1882, p. 386.

[2] Journal of Nervous and Mental Diseases, September and October, 1886.

[3] Ibid., September and October, 1887.

THE SURGICAL TREATMENT OF ABNORMALITIES OF THE BRAIN AND SPINAL CORD.

BY P. S. CONNER, M.D.

THE congenital or early-developed abnormalities of the brain and spinal cord that may demand and with advantage receive surgical treatment are meningocele, encephalocele, spina bifida, and hydrocephalus; in other words, herniæ and dropsies.

Abnormal protrusion of a portion of the brain or its coverings may be due to too slight resistance on the part of the membranous or bony cranium, to increased pressure from within because of ventricular dropsy, or to a dragging force from without consequent upon early attachment to the fœtal envelope. As a result of the action of these causes, the presenting tumor may be a portion (1) of the meninges, containing fluid,—*i.e.*, a *meningocele*, which, if it occur at all (and this is much doubted), does so very rarely; (2) of the brain,—*i.e.*, an *encephalocele;* (3) of both brain and covering,—*i.e.*, a *meningo-encephalocele*, which is the most frequently observed condition; or (4) of the brain with included ventricle distended with fluid,—*i.e.*, a *hydrencephalocele.*

As shown by Berger, there occurs at times a hyperplasia of the meningeal envelope simulating a neoplasm, and when brain-substance is present it may be of such mixed character as to indicate that the protrusion dates back to an early period in the development of the nervous system.

The size of the hernia varies greatly, at times almost equalling that of the head itself. As might be expected from the manner of its production, its location is along the suture-lines, being in the great majority of instances somewhere on the median line, most commonly in the occipital region, but not seldom in front, at or near the naso-frontal junction. Very rarely has it been found in the temporal region. When located posteriorly it may be just above the foramen magnum, at the fontanel, or, more usually, between the two at the meeting-point of the several parts of the occipital bone. That brain is or is not present cannot in many cases be absolutely determined except by actual inspection, or, it may be, careful microscopic examination, and it should therefore always be regarded as entering into the composition of the tumor.

When other than quite small, its diagnosis is ordinarily easy, if due consideration is had to its location, its more or less yielding and fluctuating character and translucency, its reducibility, and the effect of pressure in producing general disturbances (nausea, convulsions, or even coma). It may be confounded with simple serous cyst, but in the great majority of cases such cyst may properly be regarded as having been originally a hernia, the connection of which with the interior has been cut off. If small, especially when located near the inner angle of the eye, it may be and has been mistaken for a wen or an erectile tumor, which latter an encephalocele may much resemble in general appearance. In at least one case it has been thought to be a nasal polyp, and operated on accordingly, with a fatal result. The great difficulty at times, not to say impossibility, of clearly diagnosticating the affection makes it necessary to view with much suspicion any early-noticed tumor situated in or near the median line, especially in front.

In all the abnormalities under consideration treatment is either palliative or radical, the former consisting in the application of a protecting pad, and, if permissible, of moderate pressure. Radical treatment has been by puncture, by injection, and by incision followed by either replacement or excision. Puncture, which until quite recently was the only operation proper to be done, since the others were almost certain to be followed by fatal septic inflammation, has proved unsatisfactory, having ordinarily to be frequently repeated, and being liable, even though all antiseptic precautions are taken, to give rise sooner or later to meningitis.

Injections are not to be advised, because of the great likelihood of the production of destructive irritation and inflammation.

Shall a cutting operation be done? It is as yet too soon, the cases are too few, to warrant the expression of a positive opinion. Septic meningitis has up to a recent date carried off the majority of the patients. If an operation is to be done, the sac should be freely opened, the protruding brain-substance replaced if possible, the sac excised and its edges sutured, and the skin-flaps brought together and united. Only when the brain-hernia is small can it be put back, the obstacle to reduction being either the size of the opening in the skull or the intolerance of the brain to the necessary pressure.

How shall an irreducible mass be treated? Either by letting it alone, closely covering it in with sac and skin-flaps, or by excising it. The latter procedure will certainly add to the gravity of the operation, particularly if the case is one of hydrencephalocele, when some part of the lateral ventricle must of necessity be opened. Further experience must determine how much of the brain may be cut away without resulting death or marked mental impairment.

When the tumor is very small, is not enlarging, and seems to be producing no disturbance, it should not be interfered with; nor should any operation be done when the general condition is bad, or when there is present other malformation dangerous to life, or of such nature as to render it undesirable that life should be prolonged.

Spina Bifida.—Hernia of the spinal cord or its envelopes, or both, is commonly known to English and American physicians and surgeons as spina bifida, because of the prominence attaching to the associated cleft condition of the spinal column, which only in the most exceptional cases is of the vertebral bodies. In the great majority of patients the non-closure of the posterior arches is in the sacral, lumbo-sacral, and lumbar regions, occasionally in the dorsal, and much less often in the cervical. In a few instances the entire canal has been found open posteriorly. Like the corresponding brain-hernia, that of the cord may be (1) of the meninges, *meningocele*, the dropsy occurring in the arachnoid cavity and pushing the cord forward against the bodies; (2) of the cord and its coverings, *meningo-myelocele*, the fluid being in the subarachnoid space; or (3) *syringo-myelocele*, from distention of the central canal, the cord being flattened and spread out. The second variety is much the more common: 63.2 per cent. of the cases examined by the Clinical Society Committee were of this class. Ordinarily, if not always, congenital, the tumor may, though very rarely, manifest itself only some time after birth. In a few cases there is present a thick covering of skin, but usually the integument is very thin and closely adherent, generally entirely absent over the central part of the mass. In Ranke's opinion, it was a growing together of the membranes of the cord and the external skin that prevented closure of the canal. Instead of the ordinary reddish color of the tense shining tumor, the sac may be bluish white, as in a case of Crew's. In other than simple meningoceles the cord or a part of it is found within the sac, more or less closely and extensively attached to it or even passing over in its wall. Not seldom a dimple or depressed furrow indicates the point or line of adhesion. Existing septa may make the tumor multilocular.

Other malformations, especially hydrocephalus and club-foot, are often present. Paraplegia and sphincter-paralysis may exist at the time of birth, in which case death speedily occurs. In many cases paralysis is developed after a short time. Spontaneous rupture often takes place, in labor or after a few hours or days, with the result ordinarily of causing death, either quickly from shock, or more slowly from septic meningitis. If the opening is a very small one, such inflammation may not be developed.

The only other conditions likely to be mistaken for spina bifida are fatty tumors and congenital cysts; but mistake is not apt to be made if regard is had to the consistency and probably lobulated character of the former, and to the absence of the effect of pressure, of changes of position, of crying, etc., in altering the size of the latter. The cyst may be and often is an original spina bifida in which the vertebral opening has become closed. Fatty, cystic, and even fœtal tumors have been noticed coming from within the spinal canal and preventing union of the arches.

Though the majority of the subjects of this abnormality die early, largely from marasmus, a few live for many months or years, and individuals have survived to the age of thirty, fifty, even seventy-four years. Of Demme's thirty-two patients, however, who were untreated, all were dead within two years. Occasionally it happens, as in a case of Whitehead's, that after years (twenty-one in the case referred to) of quiescence the tumor enlarges and becomes troublesome, even dangerous. Spontaneous closure of the opening may occur.

As in cases of brain-hernia, so here, treatment is either palliative or radical, intended to protect by pad or other mechanical covering and secure moderate compression, or to remove the tumor and effect closure of the opening. When the protrusion is small, is not enlarging or that but slowly, and is covered with healthy skin, the palliative treatment should always be adopted. The application of collodion has been found of service. There is much wisdom in the suggestion that, as a rule, no radical treatment should be instituted for at least two months after birth, as not seldom by that time indications of spontaneous healing will manifest themselves, and a large proportion of the children unfit for operation will have died.

The radical treatment has been by puncture, by injection, by ligation or clamp-compression, by excision and plastic operation.

Puncture, which has usually had to be repeated owing to the refilling of the sac, has occasionally been followed by cure, but ordinarily has resulted in failure, commonly in death, due to a developed meningitis.

The success following the application of a ligature, the écraseur-wire, or the clamp has not been such as to give much encouragement for their use.

Puncture followed by simple iodine-injection, advised by Velpeau and first practised and highly commended by Brainard (iodine gr. v, iodide of potassium gr. xv, water ℥i), was a favorite method of treatment until Morton proposed the use of glycerin in place of water (iodine gr. x, iodide of potassium gr. xxx, glycerin ℥i), since which time such solution has been ordinarily employed; the glycerin being thought to lessen the liability of the diffusion of the irritating iodine to the parts outside the sac. Many successful cases have been reported (twenty out of twenty-six treated by simple injection, thirty-five out of seventy-one by the Morton fluid, of the cases examined by the Committee of the Clinical Society of London), but too much regard should not be had to the statistics that have been accumulated and published, as they are not very extensive, and there is good reason for believing that the large majority of the unsuccessful cases are not put on record.

Great care must be taken, in withdrawing the fluid of the sac, not to remove too much,—not more, certainly, than one-half,—and to inject slowly the iodized glycerin in small quantity (ʒss–ʒiii), as otherwise fatal shock may be produced or violent inflammation rapidly excited. I have once seen an infant several months old die immediately upon being turned over upon its back, after removal of a large part of the fluid in the sac and injection of perhaps forty drops of the glycerin solution.

The existence of paralysis is a contra-indication to the use of iodine, unless, as suggested by Ashhurst, it can be thrown into the coverings without perforating the sac itself.

When a successful result is to follow the injection, the sac becomes filled with new connective tissue, which later undergoes contraction and solidification. Whenever puncture is made, with or without associated injection, the needle should be entered at a considerable distance from the median line, to lessen the risk of injuring any part of the cord which may be in the sac: as has been said with truth, we have "no means of determining in the living subject whether or not the cord is in the sac." Electrical stimulation has been proposed as a method of making such determination.

Though the injection treatment in "its mode of action most nearly resembles the natural mode of cure," and its employment has not seldom been followed by a satisfactory result, yet within the last few years direct operation has much engaged the attention of surgeons, and in a number of cases the sac has been removed and the adjacent soft parts brought together as closely as was possible. In one case of spontaneous rupture, Davidson (of Glasgow) used a thin layer of sponge cut to fit the opening in the sac as a framework for granulations, and found after a time that it had entirely disappeared; a cure followed. To close the vertebral gap Mayo-Robson introduced a piece of rabbit periosteum, without, however, securing the desired result; and Dollinger has done an osteoplastic operation, breaking down, bending in, and suturing together the everted arches.

Too rapid and excessive escape of fluid should be prevented by the position of the patient rather than by the use of clamp or ligature. Any nerves that may be in the sac should, if possible, be dissected out and returned within the canal.

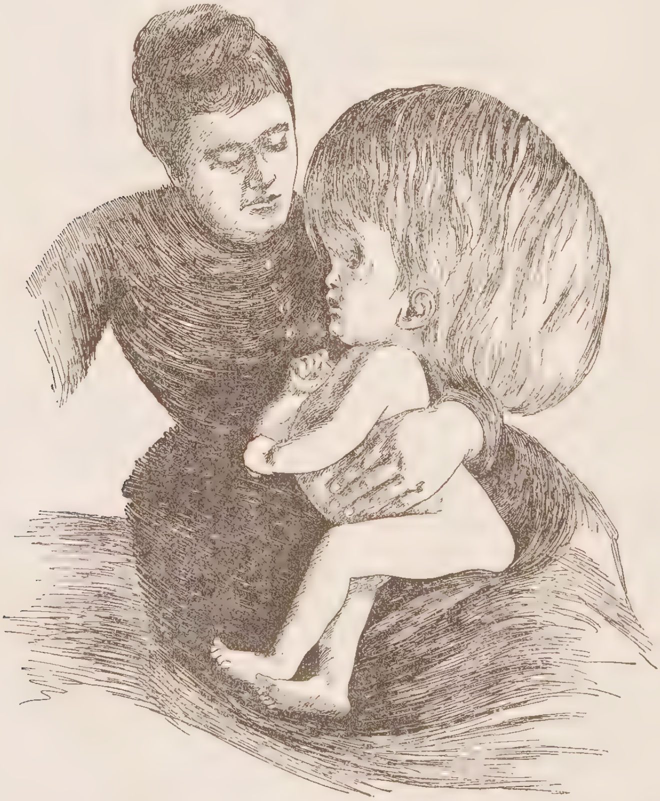
Zenenko has stated that removal of such nerves apparently causes no discernible disturbances in the functions of any organs, yet such a case as the one reported by Barton shows that, even if no speedy ill consequences result, convulsions, followed by death, may later occur.

In closing the wound, the several layers should be separately united by buried sutures. Parkes, who has reported three operations, with two recoveries, attributes the fatal result in the third case to infection introduced along the tracks of two silk sutures passed through the skin and the deeper parts to strengthen the union, septic meningitis developing very quickly after removal of the stitches, though primary union of the wound itself had taken place. It is of interest to notice that the two children who recovered were very young (four and seven weeks old).

Though these excisions and plastic operations have but seldom been done, though it is exceedingly difficult to keep the wound aseptic, and though the results thus far have not been very encouraging (*e.g.*, three out of five patients operated upon in the Göttingen clinic and reported upon by Hildebrand died), yet future experience, and that on an extended scale, can alone determine the actual value of the really operative treatment of this abnormality.

Hydrocephalus.—Consequent upon interference with the outflow of blood through the Galenic veins or closure of the communication between the ventricles and the subarachnoid space, or because of some disturbance of the balance between secretion and absorption the cause of which may not be recognizable, there occurs a dropsical accumulation in the ventricles or subarachnoid space, or both, apparent at birth or manifesting itself soon after. Not seldom various members of the same family are affected, and coexistence of other abnormalities (spina bifida, club-foot, hare-lip, etc.) is frequently observed. Those congenitally affected are, as a rule having few exceptions, short-lived, but when the disease is developed later and the enlargement is of slow production the patient not very infrequently lives to adult, it may be, though rarely, to old age. The size to which the head may attain is at times monstrous : in the Warren Museum is a skull twenty-seven and a half inches in greatest circumference ; in the Cruikshank Museum was one of fifty-one inches. Cardinal's head was thirty-two and a quarter inches around, Esquirol's patient's thirty-six inches. In a child twenty-eight months old whom I saw (Fig. 1), the distance from the root of the nose to the occipital protuberance was forty-two inches, and that from one ear to the other over the vertex twenty-seven and a half inches.

Fig. 1.



Occasionally the enlargement is asymmetrical, even one-sided. The great disproportion between the size of the head and that of the face gives a peculiar and characteristic appearance to the child. When, as is usually the case, the dropsy is intraventricular, the eyes are carried downward and outward, while their position is unchanged when the enlargement of the head is due to fluid in the arachnoid cavity only. In other than the milder cases the brain is, as a rule, feebly developed, and often will be found spread out as a thin layer lining the inner surface of the sac, or resting upon the base of the skull. Henoch reports a case in which the hemispheres had almost entirely disappeared.

Spontaneous bursting of the sac is usually followed by death, though recovery has been known to take place. Extensive cellulitis and formation of abscesses have been reported.

Of treatment little can be said. The internal administration of drugs is seldom or never of any value, and local applications are practically useless. The only operative treatment as yet resorted to has been puncture, at times employed since the days of Le Cat, occasionally with success, especially in cases of arachnoid dropsy. Only a moderate amount of fluid should be drawn off at a time, and that through a puncture a little to one side of the median line near the anterior fontanel. The aspirator-needle should be preferred to the trocar and canula.

When closure of the sutures has taken place yet symptoms of intraventricular dropsy are present (indications afforded by ophthalmoscopic examination are of great value), the ventricles may be tapped through an opening made in the skull, as has been done by Ayres and Keen. The latter advises lateral trephining, and directs to make a large opening one and a quarter inches behind the meatus and one and a quarter inches above Reid's base-line. Puncture towards a point two and a half inches directly above the opposite meatus. The puncture will traverse the second temporo-sphenoidal convolution and enter the normal lateral ventricle at the beginning or in the course of the descending cornu at a depth of about two to two and a quarter inches from the surface. In the case operated upon, the ventricle was reached at a depth of one and three-quarter inches from the dura mater. The same objection holds against this operation as against tapping in ordinary cases: reaccumulation of the fluid is almost certain to occur.

THE OPERATIVE SURGERY OF THE BRAIN AND SPINAL CORD.

By CHARLES B. NANCREDE, M.D.

As this article is strictly confined to the consideration of operative technique, diagnosis and indications for or against operative treatment must be sought in other sections of this work.

The fact that " no injury of the head is too trivial to be despised, and none too severe to be despaired of," should unceasingly influence the surgeon's opinion and control the treatment of head-injuries at all ages, but especially those occurring during childhood. As the most trivial injuries of the scalp may be complicated with severe cranial and cerebral injuries, or as secondary diseases of the soft structures may initiate fatal intracranial trouble, the proper therapeusis of scalp-wounds must first be briefly considered. An insignificant scalp-wound, *if it become infected,* may lead to periostitis, purulent osteitis, osteophlebitis, and encephalitis ; or, again, from the periosteum the infective process may, by continuity of tissue, through osteophlebitis, initiate thrombosis of the cerebral sinuses, with consequent pyæmia. Although such complications, as well as tetanus, are uncommon, they are possible, and must not be ignored.

INCISED, LACERATED, AND CONTUSED WOUNDS OF THE SCALP.

Incised Wounds.—In view of the just-mentioned facts, and lest the wounds of the soft tissues be only the smaller part of a severe head-injury, the following precautions should always be observed before any examination of the wound is made.

The hair should be carefully removed from the edges and immediate neighborhood of the wound by the razor or scissors, after a thorough drenching with carbolized water, and the scalp carefully scrubbed with a nail-brush wet with a mixture of two parts of turpentine and fourteen parts of alcohol, followed by soap and carbolized water, after which the wound should be thoroughly irrigated with mercuric bichloride solution ; now, and only now, can the wound be safely explored with the disinfected finger or probe. Careful search revealing neither foreign bodies nor fracture, any hemorrhage which cannot readily be controlled by compression must be checked by twisting or tying both ends of the divided vessel. When the ends retract into the dense fibro-cellular tissue, they can be best secured by passing a needle armed with a ligature around the vessel, including some of the surrounding tissues. Ligatures will probably never be necessary in children except when the wound involves the lower part of the temporal fossa, in which case the bleeding may prove difficult to arrest. "Should the bleeding recur or become dangerous, notwithstanding all our local means, the question of applying a ligature to the external or common carotid may arise."[1] I quote the foregoing sentence not because in my practice any such contingency has arisen, but because so experienced a surgeon as Hewitt has evidently known of some such question arising.

All bleeding having been arrested except that which is to be checked by the compression of the dressings, the wound, if it have not penetrated the aponeurosis of the occipito-frontalis muscle, should be closed by fixing one end of a strip of fine aseptic gauze with iodoform-collodion[2] upon one side of the cut, when the wound can be accurately coaptated, and held so by painting the other end of the gauze strip with more collodion, the drying and consequent fixation of the dressing being hastened by fanning. This expedient is preferable to the use of adhesive plaster, because the latter, by the rapid growth of the hair, soon becomes loosened, and, above all, asepticity of the wound is rendered difficult, if not impossible.

When the wound extends through the occipito-frontalis aponeurosis, catgut or horse-hair sutures will often become necessary from the gaping of the wound, but before their passage the centre of a bunch of fine catgut must be secured by a suture of the same to the deepest portion of the wound, three or four strands being carefully brought out between each pair of sutures. In either variety of scalp-wound, dusting with iodoform,[3] laying a piece of protective along the line of the wound, and the adjustment of appropriate aseptic gauze compresses to secure contact of the flap with the deeper parts, must be resorted to, covering all in with about eight thicknesses of bichloride gauze the innermost layers of which have been thoroughly moistened with the mercuric solution. My preference is to retain all in place by the application of a moist carbolized gauze roller, which, when dry, forms a firm unyielding dressing. For appearance' sake a muslin bandage may be applied outside the gauze roller.

I have entered into these minute details because after compound fractures, the operation of trephining, etc., the same method of dressing should be adopted, with the exception of the drainage, which in certain instances must be with the tube, as will be indicated in its proper place.

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[1] Hewitt, Holmes's System of Surgery, 3d ed., vol. i. p. 570.

[2] Plain collodion will do very well, while shreds of absorbent cotton can be substituted for the gauze.

[3] Notwithstanding the experiments of laboratory investigators, I still cling to the use of this drug, my clinical experience apparently contradicting that of the experimenters. Probably both are correct, recent observations going to show that iodoform destroys the virulence of the ptomaines resulting from the multiplication and growth of certain of the micro-organisms.

Should suppuration beneath the aponeurosis occur, from neglect of the proper aseptic precautions or from infection of the wound, capillary drainage will fail to remove the pus, and a tube or tubes must be substituted, with the application of such compresses as will prevent all purulent accumulations. The wound will rarely be situated so as to afford drainage at the most dependent portion of the accumulations, under which circumstances counter-openings are indicated, to be kept patent by the introduction of small drainage-tubes. If the surgeon will remember that this aponeurosis is attached behind to the superior curved lines of the occipital bone, to the mastoid process of the temporal bone and the zygoma laterally, and that it becomes continuous with the tissues of the upper lid and those over the root of the nose, together with the position in which the disease will compel the patient to keep his head, the proper places to make the counter-openings can readily be determined in each case. The most strenuous efforts must now, of course, be made, by the occasional use of the stronger antiseptic solutions, to render the pus-cavities aseptic, and all bagging of pus must be prevented by proper compresses, for by such means oftentimes a rapid diminution in discharge, pain, and constitutional symptoms will result. Sloughing may occur, but is rarely productive of any osseous necrosis, especially if strict asepsis be secured. Should the surgeon fear to trust to the unaided powers of nature, he may invoke the osteogenetic powers of the diploë by penetrating the outer table with a fine awl, making the perforations as close together as convenient. In a few days granulations will spring up from the diploë and coalesce with those of the soft parts. It can hardly be expected that extensive or more massive necrosis will occur in childhood from traumatisms, with which we have chiefly to deal in this section, but, should this occur, it must be remembered that separation of the sequestra will probably occupy months or years, so that the subsequent discharge and exhaustion may at times compel the surgeon to resect large portions of the calvarium.

Constitutional Treatment.—As this is the form of inflammation too commonly mistaken for erysipelas and alleged to be due to the use of stitches, and as the disease may in some cases either be of a genuine erysipelatous nature or become complicated with erysipelas, attention to the digestive tract by the exhibition of calomel and soda in small doses, followed by a saline, will prove useful, when indicated by the condition of the tongue and bowels. Quinine, tincture of the chloride of iron, and stimulants when necessary, should then be prescribed, with abundance of nutritious, non-irritating food.

Lacerated and Contused Wounds: Local Treatment.—After the preliminary antiseptic cleansing previously described, these wounds, if not penetrating the aponeurosis, should, when needful, be coaptated by gauze (or shreds of absorbent cotton) and collodion, while division of the aponeurosis, with consequent gaping of the wound, calls for sutures as a retentive measure, even if complete coaptation cannot be effected: they should be drawn just tight enough to effect this end, while drainage must be provided for as in incised wounds. Under no circumstances should rags of bruised, apparently hopelessly-damaged scalp-tissue be cut away, for even in the adult they rarely slough, much less in children, and, should they die, no harm will accrue, provided asepsis be secured: this is a cardinal rule.

CONTUSION OF BONE, WITH SUBSEQUENT OSTEO-MYELITIS, OSTEO-MYELITIS FROM MIDDLE-EAR DISEASE, ETC.

This complication, which probably always results from infection, should but rarely occur when proper aseptic methods, such as suggested, have been conscientiously pursued. When the constitutional or local symptoms, or a combination of both, indicate the necessity for operative interference, after the usual preliminary antiseptic precautions, a large flap, its outline being a shallow curve so planned as to avoid the main scalp-vessels and favor drainage, should be raised, all the tissues, including the periosteum, being divided at one cut, which must be vertical to the skull. Preferably under constant antiseptic irrigation, the bone should be removed with the gouge, trephine, chisel, saw, or burr of the dental engine, including some of the apparently healthy bone for a short distance around the diseased area. Appropriate tube-drainage being instituted, the flap may be now laid down and carefully sutured with silkworm-gut, silk, or prepared horse-hair, and an antiseptic dressing applied over all. That under the most unfavorable circumstances success is still attainable in cases of this nature, which have resulted even in thrombosis of the lateral sinus and septic embolism of the heart and lung, is proved by a case of Horsley's, the disease originating, as is most usual, from old middle-ear disease.[1] Horsley has suggested that when "the first indication of embolism appears, the internal jugular vein should be tied in the middle of the neck," though, as he points out, a serious argument against the performance of this operation "lies in the, at present impossible, task of discovering how much thrombosis there is, and, further, what risk there is of embolism from the same."[2]

SIMPLE FRACTURES OF THE VAULT.

Should operation be indicated,—and I must reiterate that in this article I have nothing to do with anything beyond the mere technique of the various operations required for injuries and diseases of the head,—the usual antiseptic preliminaries must be attended to with peculiar care, for here, unlike the case of a compound fracture, the "surgeon's acts determine the fate of a fresh wound," and "infection and suppuration are due to his technical faults of omission and commission." Since septic infection and inflammation of the meninges and brain constitute the only immediate risk to life of trephining, no arguments are needed to enforce unusual care. The preferable form of flap, that which has been just described in the preceding section, should always be employed where the previous damage to the soft parts admits of it, which in the injuries now under consideration, of course, cannot interfere. The bone being exposed, the depressed incarcerated fragments must be released by as limited a removal of the overlapping sound bone as will admit the introduction of the point of the elevator: if extensive comminution of the internal table exists, sufficient sound bone must be removed to extract all the fragments of the internal table.

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[1] Medical Press and Circular, 1886, N. S., p. 495.

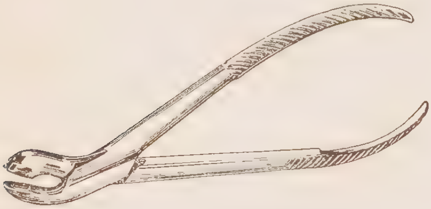
[2] The Operations of Surgery, p. 197, W. H. A. Jacobson, F.R.C.S., Philadelphia, 1889. This suggestion has been recently successfully carried into effect by another surgeon.

Oftentimes the use of a Hey's saw, by removing the overlapping edge of the external table of the skull, or by merely widening a fissure, will render elevation possible, but, when the whole thickness of the skull is driven down, the bone can be more rapidly and satisfactorily removed by the use of W. B. Hopkins's modification of the rongeur. Again, in many cases the trephine must be used, especially where other instruments are not available.

METHOD OF PERFORMING THE OPERATION OF TREPHINING.

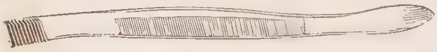
The special instruments required are an elevator, a knife with raspatory attached, trephines of various sizes, a trephine-brush, a pair of Hey's saws, a small pair of cutting bone-forceps, the gouge-forceps of Hopkins, and perhaps a lenticular, with which some surgeons still prefer to elevate and smooth the rough edges of bone; but I have never seen any necessity for its use, as an elevator in careful hands is perfectly safe, and its rough serrated edges serve admirably to file away, as it were, any inequalities left by fragments of the brittle inner table remaining at the periphery of the aperture.

Fig. 1.



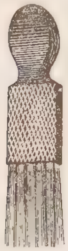
Hopkins's rongeur.

Fig. 2.



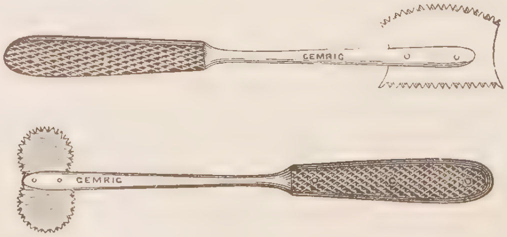
Elevator.

Fig. 3.



Trephine-brush.

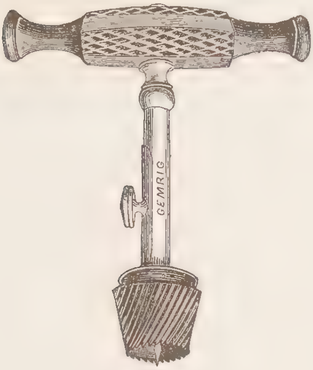
Fig. 4.



Hey's saws.

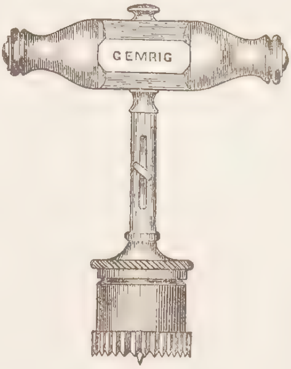
Hopkins's rongeur (Fig. 1) is an admirable instrument, and, where it can be used, serves a better purpose than the trephine or Hey's saw (Fig. 4) in removing an overlapping shelf of bone, while it is an instrument which can be used with more safety and rapidity. Of the two forms of trephine, the conical, commonly called Galt's (Fig. 5), is the safer, since from its form, *when properly constructed*, it is almost impossible for it to injure the brain if, as the last portions of the inner table are divided, undue pressure be made; while under these circumstances the old form of instrument (Fig. 6) has occasionally been accidentally plunged into the brain-substance. Horsley[1] has devised a trephine with a peculiar handle, with such a thin cutting edge that the buttons of bone when replaced fit much better than they would if cut out with the ordinary form of instrument.

FIG. 5.



Conical trephine.

FIG. 6.



Old-pattern trephine.

All the instruments, including, in addition to those mentioned, a flat-ended probe or toothpick to measure the depth of various portions of the trephine-cut, a tenaculum, several hæmostatic forceps, a pair of scissors, etc., should be thoroughly boiled[2] in plain water, and then with *aseptic hands* removed and placed upon a towel wrung out of a five per cent. carbolic acid solution, another similar towel covering them until needed.

A convenient method of supporting and steadying the head is the use of sand-bags. Anæsthesia must be carefully induced, and, in my judgment, except for brain-tumors and perhaps epilepsy, ether should be the agent employed.

The scalp surrounding the proposed site of operation, having been shaved and disinfected as previously described, where there is no wound, should be incised down to the bone with one stroke of the knife, forming a large flap whose outline is a shallow curve so planned as to avoid the main scalp-vessels and favor drainage in the recumbent posture; this should be rapidly reflected. Any bleeding which does not spontaneously cease in a few moments should be arrested by the hæmostatic forceps, or possibly by ligatures, but these are rarely necessary. The periosteum usually readily strips off with the rest of the flap, but this manœuvre may be aided by the use of the periosteal elevator of Mr. Victor Horsley's, depicted in Fig. 7. When merely an overlapping edge of bone has to be removed, this separate removal of the periosteum is hardly necessary, the membrane being readily scraped aside by the raspatory. All these manœuvres, with those presently to be described, should be executed under a constant flow of tartacid mercuric bichloride solution, one to two thousand or three thousand.

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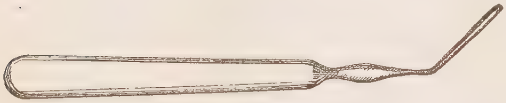
[1] At least one other surgeon claims the peculiar form of handle figured, and I cannot say that Mr. Horsley does more than state his *preference* for this form of instrument.

[2] Boiling in plain water will render aseptic any instrument if kept up for not less than five minutes, and has the advantage of not injuring the edge of cutting instruments. For this reason solid steel instruments are desirable; but even ivory or ebony handles will often stand this treatment: if not, they must be cleansed by *long immersion* in the five per cent. carbolic acid solution.

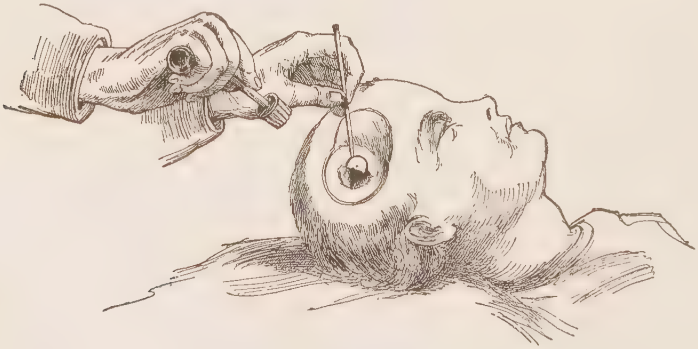
FIG. 7.



Horsley's periosteal elevator.

The trephine, with its centre-pin protruded about one-sixteenth of an inch and firmly screwed in this position, should now be applied to the portion of bone which it is intended to remove, the periphery of the trephine somewhat overlapping the depressed bone at one point, as indicated in Fig. 8. The instrument should be worked with a light, sharp, quick movement from left to right and from right to left, care being taken not to press unevenly, and the pressure being chiefly exerted as the hand is carried from left to right. As soon as a sufficient groove has been cut to steady the trephine, the centre-pin should be withdrawn, and fixed so as to avoid injury to the dura mater. When the outer table of the skull is cut through, if irrigation is not used, the bone-dust, which up to this time has been dry, becomes soft and bloody, as the instrument penetrates into the diploë.[1] Both the sound and the feel are also different. When the use of the centre-pin seems undesirable, Dr. P. H. Watson, of Edinburgh, has suggested that the instrument should be steadied by applying it through a perforated

FIG. 8.



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[1] This is absent in early life, and at all ages over a large part of the squamous bone and in the occipital fossæ.

piece of pasteboard firmly held against the bone. In view of the great difficulty of rendering pasteboard aseptic and of its softening under irrigation, I would suggest the substitution of a perforated piece of thin splint-wood.[1] If the irrigation does not keep the trephine-teeth free of bone-dust, or when this measure is not employed, the instrument must be removed from time to time, and cleansed in the carbolized water, either by the brush or a sponge.

The detritus lying in the bony groove should also be removed by the toothpick or flat end of the probe, advantage being taken at the same time to measure the depth of the cut at various portions of the circumference, to ascertain whether the bone is being evenly divided. If one segment of the groove be deeper than another, the trephine must be inclined towards the shallow side, and pressure made at that point alone until the groove is of equal depth throughout. It must be remembered that the bone is frequently of unequal thickness at various parts of the circumference of a trephine-cut, so that, while the osseous disk may be firmly held fast at some points by portions of the inner table, the teeth of the instrument may at other points be tearing the dura mater, the chief thing to be avoided. Even in the adult the skull-cap varies from one-fifth of an inch upon the average, to as much as three-fourths of an inch at the occipital protuberance. In young children the diploë may also be absent, so that in them great caution must be exercised. I can give no better working rule than that of Holden: "Think that you are operating on the thinnest skull ever seen, and thinner in one portion of the circle than in the other."

When the instrument reaches the inner table, both the sound to the ear and the sensation conveyed to the hand usually give warning. Great care should now be exercised: each turn must be made cautiously and with very light pressure, frequent resort being had to the probe or toothpick to determine at which points, if any, the bone has been completely divided, that the teeth may be so inclined as to avoid cutting the dura mater. By a slight rocking movement of the trephine, the looseness of the piece can be ascertained; but it is better to lay the instrument aside, and, seizing the disk in the grasp of a stout pair of forceps inserted in the groove, gently move it from side to side. If loosened at one edge, the trephine-teeth must be made to cut upon the opposite, attached part, for a turn or two, when the forceps should again test the stability of the disk. When loosened, the bone may come away in the crown of the trephine; but I think it safer to remove it by tilting the piece out with the forceps, using a rocking movement, and always drawing it out towards the side where any portion of the inner table remains unsawn, as then the dura mater escapes the slightest injury from the other thoroughly sawn and perhaps splintered edge of bone. Should any portions of the inner table be left behind, they can be removed by the rongeur, elevator, or lenticular. When a very large disk is being removed, as one from one and a half to two inches in diameter, the bone had perhaps better be removed in the following manner. When the disk is loosened, the elevator may be gently used to lift one edge, after which the dura mater should be carefully stripped off with Horsley's instrument depicted on page 754 (Fig. 7), or by a smooth, blunt elevator. The trephine-disk, as well as any other fragments subsequently removed, should at once be transferred to a warm solution of mercuric bichloride, one to two thousand, placed in a china receptacle,[1] and carefully maintained at a temperature of from 100° to 105° F.

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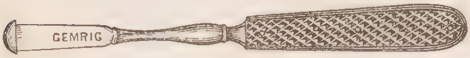
[1] In sets of trephining instruments manufactured over fifty years ago, there was a similar steel instrument with two handles constructed for this special purpose, as well as segmented trephines, such as were reinvented by Dr. J. B. Roberts a few years since.

Fig. 9.



Lenticular.

When dealing with a punctured fracture, a trephine large enough to include the starring of the outer table with a small area beyond should be used, when most of the fragments of the inner table will come away with the button. Great care must be exercised in removing splinters of the inner table, lest the dura mater or venous sinuses be wounded. When dealing with an ordinary depressed fracture, the trephine should be applied so that only about two-thirds of the circumference will be located upon the sound bone, and the crown of the instrument should be no larger than will admit of the easy introduction of the elevator. If the removal of one button will not allow of elevation of the fragments, more bone can be readily removed by the rongeur or Hey's saw; if not, a new segment of bone must be removed with the trephine. Certain precautions must be observed when the fracture is near a sinus which we suspect has been wounded by the depressed fragments. Thus, the trephine-cut must be planned so as to give free access to any bleeding point, rather than merely to admit easy elevation of the fragments. One is often tempted to draw out a long fragment driven some distance beneath sound bone, whose concealed extremity lies in close proximity to a large sinus, possibly wounding it; but in such a case this would be a dangerous practice, since without removal of bone access to any bleeding point cannot be obtained. It is far safer to trephine over the site of the concealed extremity of bone, when, if its withdrawal is followed by hemorrhage from a wound of the sinus, instant compression may be effected, as will presently be indicated: neglect of this rule has been followed in the practice of two eminent Philadelphia surgeons by almost instant death, in one case from an extensive wound of the lateral sinus, in the other from a wound of the superior longitudinal sinus. Large instruments are indicated when operating for intracranial hemorrhage, pus, cerebral tumors, or epilepsy, or after traumatism when cerebral localization suggests the gradual involvement of one centre after another.

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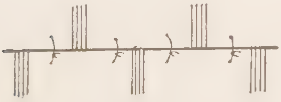
[1] I specially mention this because I have known of a surgeon placing the bone in a mercuric solution contained in a *metal* basin, which, of course, decomposed the antiseptic, subsequent necrosis of the replaced button resulting, compelling its removal.

Whenever trephining is performed, all loose fragments of bone should be removed, to make sure that no spicules have wounded the dura mater or brain, and depressed pieces elevated, but allowed to remain *in situ*, unless evidently *infected* by the vulnerating body, when, unless the damaged portion can be disinfected while in position, the whole piece had better be removed, cleansed, and replaced, as I shall describe. All the fragments should be placed in the warm antiseptic solution, as before mentioned. If the sinus-like veins of the diploë bleed, the bone-tissue around the orifice should be crushed into the sinus-mouth from all sides by some blunt instrument, as the elevator, or they must be plugged with a small fragment of antiseptic gauze, which may be removed at the end of the operation or allowed to remain for twenty-four hours.

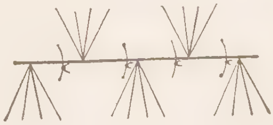
Any hemorrhage from bleeding points in the dura mater must be arrested by fine catgut ligatures, passed, if requisite, through the membrane with a curved needle. Should a careful search indicate neither subdural blood nor wound of the dura mater, after smoothing off the edges of the bone-opening with the rongeur or file-edge of the elevator to lessen the risk of ulceration of the dura mater, the bony fragments should be replaced as nearly *in situ* as possible, when, if they are too small to fill up the gap, they should be perforated with the centre-pin of the trephine, placed with one portion of their periphery in contact with the sound bone, and sutured to the scalp by passing a stout catgut thread through the perforation, as suggested by W. W. Keen. Where a large gap is still left, osteogenesis may be secured by carefully mincing the smaller bone-fragments and dusting them over the exposed dura mater. After a final irrigation with mercuric bichloride, a bunch of fine catgut drain should be secured by its middle to the deeper parts of the wound, and the flap then laid down, and sutured by medium silk or, what I prefer, silkworm-gut, placing the stitches about one centimetre apart, bringing out between each pair three or four catgut threads, which must be kept carefully in contact with one another (Fig. 10) and not sprayed out (Fig. 11). Horse-hair or fine silk may be used between the other stitches if deemed necessary. Dusting with iodoform,[1] protective to keep the catgut drains moist,—without which precaution they will

FIG. 10.



Proper method of arrranging gut threads for capillary drainage.

FIG. 11.



Improper method of arranging gut threads for capillary drainage.

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[1] This drug should be carefully washed with bichloride, repowdered, and kept in aseptic receptacles.

fail to drain,—and a voluminous mass of wet corrosive gauze will complete the dressing, all kept in place as directed under the caption Scalp Wounds.

When the dura mater has been wounded, it should be carefully sutured with fine catgut when possible. If this be not feasible, a rubber drainage-tube had better be used for twenty-four to forty-eight hours, for a reason which will be explained when speaking of the removal of cerebral tumors. Certain modifications of operative methods, drainage, and after-treatment will be described in the sections treating of brain-abscess, brain-tumors, and epilepsy.

The after-treatment will be pointed out in a separate section at the end of this article.

It is advisable to avoid certain regions of the skull in trephining, such as over the course of sutures, because the dura mater usually adheres at these points with exceptional tenacity, rendering tearing of the membranes a probable accident, and also because of the great difference in the thickness of the bone: thus, along the sagittal suture, where lies the groove for the longitudinal sinus, at one portion of the periphery of the trephine-cut the teeth would almost certainly touch the dura mater, while at other portions the bone would be only half sawn through. In traumatic cases, where it seems imperative to apply the trephine over a sinus, there is, fortunately, very frequently a separation of the dura mater produced by the injury, which removes it out of harm's way. Where operating for disease, a trephine-cut of medium size should be made upon each side,—say, of the superior longitudinal sinus,—when the intervening bridge of bone in contact with the vessel can be carefully cut away with the rongeur, its smooth, thin, blunt lip safely separating the sinus previous to each cut; when this instrument is not available, the bony bridge should be cautiously divided by a Hey's saw. The risk of injuring this sinus in the adult is great, owing to the existence of Pacchionian bodies whose perforations of the dura and adhesions to the cranial walls are often undoubtedly the cause of injuries at this point: fortunately, this anatomical disposition is practically absent at the age of which I am writing.[1] Where a sinus that is *freely* exposed has been wounded, plugging a small orifice with a bunch of catgut has proved effective in the hands of more than one surgeon, as has also lateral ligation, but in my own experience pressure by a small antiseptic pad or plugging with a strip of gauze has been perfectly effective and reliable: very much less pressure is required than is used to arrest bleeding after venesection.

Trephining over the anterior inferior angle of the parietal bone is likewise to be avoided, because the middle meningeal artery so often runs in a bony canal at this point that it must then of necessity be wounded. Should this vessel be wounded, it can be secured by passing a needle armed with a fine gut ligature through the dura beneath the vessel ; if lying in a bony canal, this can be plugged with a bunch of catgut, the vessel can be sealed by a touch of a probe heated to a dull-red heat, and antiseptic tamponnade can be resorted to; or in some cases no attention need be paid to the hemorrhage until the operation is completed, as in an operation for penetrating gunshot wound recently occurring in my practice, where all bleeding had permanently ceased by the time the wound was ready for closure. As an exceedingly rare late complication of trephining, I would mention secondary hemorrhage from this vessel. Thus, Mr. Jackson reports a secondary and uncontrollable bleeding from this vessel, which required, six weeks after trephining, ligature of the carotid artery ; the patient recovered. This could occur only in a septic wound, which it is the surgeon's business to prevent.

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[1] They are said never to have been observed before seven years of age, and after that but rarely before puberty.

Trephining over the frontal sinus has been in times past a bugbear, but, as this cavity does not exist before the fifteenth to the sixteenth year, we have nothing to do with it in this work.

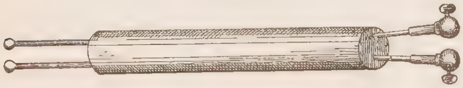
The hiatus left by removal of portions of the cranial bones is usually closed by fibrous tissue, sometimes by fibro-cartilage, and very rarely by a more or less complete, thin plate of bone. Sometimes the bony margins thicken, forming a raised ring, while at other times they become thinner, and blend with an imperfect ring of osseous tissue which extends into the fibrous membrane, thus closing the opening to a varying extent.

OPERATIVE TREATMENT OF EPILEPSY.

This consists, in the first place, in thorough excision of the scar in the scalp, should the disease have resulted from traumatism, which is all the more imperative if pressure on the scar, as in a case of my own, produces the paroxysm *on the side* of injury. If this prove abortive, or if during the operation depressed or thickened bone be detected,—provided the case be not of the Jacksonian form,—trephining, with removal of depressed or thickened osseous tissue, including some of the surrounding healthy bone, is the next step. If a scar in the dura mater, or even distinct evidences of local disease, such as thickening, exist, the diseased portion must be excised, with such underlying portions of the involved brain-substance as are included in the cicatrix, when possible, of course, dissecting the membrane off the surface of the convolutions, thus dividing only such vessels of the pia mater as necessity compels, in view of the fact that these vessels are for the most part terminal. Should a distinct cicatrix, tumor, cyst, or localized portion of cortex damaged by disease, *not by the knife*, be detected, it should be excised, the incisions being clean-cut, vertical to the surface of the hemisphere and at right angles to the long axis of the convolutions. Where nothing beyond removal of bone has been required, its reposition as just directed may be made, using capillary drainage. When the operation requires exposure of the cortex, no antiseptic solution must be allowed to touch it, since, if electricity be necessitated for locating the centres, the brain will, as Horsley has pointed out, remain sensitive only in spots, which will prove misleading or render all efforts at localization abortive. In one such case I resorted to the use of plain aseptic—*i.e*, boiled—water after the dura mater was incised, with perfect aseptic results. Where the membranes have been removed or a portion of cortex has been excised, one or more small rubber drainage-tubes should be inserted at the most dependent portions, which should not be allowed to remain for more than twenty-four hours, as will be explained under the section on the removal of brain-tumors.[1] In other respects the dressings should be applied as for an ordinary trephining.

In the Jacksonian form of epilepsy,[2] previous to operation the location of the discharging lesion must be carefully determined, and at the operation—all evidences of bone and meningeal disease having been removed—this portion of the cortex must be determined by a delicate sterilized electrode, such as that depicted in Fig. 12, using as weak a current as is possible, in view of the dangers of passing strong currents through the cerebrum. The removal of a portion of the cortical centre where the discharging lesion exists will only for the time destroy movements, "compensation," so far as the *coarser movements* are concerned, commencing within a few days to ten or more days after operation, a fact which has been observed by all operators, including myself; nevertheless, the patient or friends must be informed of the possibility of permanent loss of function, especially as the necessary dissection of the membranes may damage the outer layer of cells in regions other than those removed, or by destruction of their blood-supply may cause paresis or destruction of function.

FIG. 12.



Brain electrode.

The danger to life both of simple trephining and of operations for epilepsy is surprisingly small, provided strict asepsis be secured. With the older methods of asepsis, which too often proved a snare, by deluding the operators into a belief that they could with slight risk open the cranium, undoubtedly the mortality after trephining was *per se* 10.69 per cent.;[3] but with proper precautions this mortality can now be reduced to between two and three per cent. For the proportion of recoveries from the epilepsy the reader is referred to the article on Epilepsy in this Cyclopædia, and also to the author's article on Injuries of the Head, vol. v. of the "International Encyclopædia of Surgery."

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[1] See page 779.

[2] For the preparation of these cases for operation, arrest of hemorrhage, etc., see section on removal of brain-tumors, pp. 774, 777, 778.

[3] See International Encyclopædia of Surgery, vol. v., art. "Head-Injuries."

OPERATIONS FOR COMPOUND AND COMPOUND-DEPRESSED FRACTURES OF THE VAULT.

Nothing need be added here beyond the facts that the formation of the flap will probably have to be modified because of the wound in the soft parts produced by the vulnerating agent, and that there is the utmost risk that the wound has become infected before coming under the surgeon's hands; this renders imperative extra precautions in disinfecting the wound, and presents the most important indication for operating even when nothing more than a fissure exists,—viz., that *it is an infected wound*, and cannot be rendered aseptic without elevation of fragments, with their removal perhaps, for disinfection, even paring with a chisel having been found necessary, to get rid of dirt, etc. The slightest fissure, as by imprisoning a hair,[1] has proved the starting-point of sepsis: so that where there is the slightest doubt, all cracks should be carefully chiselled out and thoroughly irrigated with the bichloride solution.

CONSIDERATION OF THE DIFFERENT METHODS OF DRAINAGE.

The rules as to drainage in brain-surgery, in the present state of our knowledge, cannot be better formulated than as follows:

For trephining in epilepsy, for insanity, or for fractures where no lesion of the dura mater exists, catgut, and catgut only, seems indicated. Where large portions of bone have been removed and replaced *in toto*, no other method of drainage for the deeper parts is available. With lesions of the dura mater which are reparable by suture, the same kind of drainage is indicated, except when a brain-tumor has been removed.

Wounds of the dura mater which cannot be sutured indicate tube-drainage, or, better, Horsley's method (see p. 779). Trephining done for intracranial abscess calls for the use of the tube very gradually shortened, with the strictest watching of the case for some weeks after final withdrawal. If the first perforation made does not effectually drain a blood- or pus-cavity within the membranes or brain *when the patient is in the recumbent position*, either a second opening at the most dependent point should be made and through-drainage instituted, or, better still in many cases, the first button of bone should be replaced after removing a small segment of its periphery, while the second opening is utilized for the drain.

FRACTURES OF THE BASE.

In addition to the various precautions already advised, including the elevation, disinfection, and removal of fragments, in all fractures of accessible portions of the base, certain special measures must be resorted to if good is expected to result. Thus, the external auditory canal of the ear traversed by the line of fracture must be cautiously but effectually disinfected, filled with iodoform, and lightly plugged with antiseptic gauze. If the ethmoid be fractured, iodoform should be carefully insufflated into the upper nasal cavities, after a previous disinfection with a douche or free spraying with a boro-salicylic solution, which latter may be repeated from time to time. When both walls of the frontal sinus are broken, its anterior boundary must be removed with a large trephine, and the cavity most thoroughly disinfected, after which the posterior wall must be perforated, while at the conclusion of the operation a careful plugging with iodoform gauze should be resorted to to prevent infection of the wound through the nasal passages, remembering that secondary cerebral compression may result from swelling of the tampon by imbibition of wound-fluids. If requisite to remove infected materials or foreign bodies, the orbital margin should be unhesitatingly resected.

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[1] See report of a case of the author's read before the Philadelphia County Medical Society, May 8, 1889.

I have thought best to mention these points, for, although most anatomists deny that the frontal sinuses exist in the young, others insist that they are occasionally present before puberty, while the students of this book will doubtless draw the line of childhood at very different ages. Moreover, although the sinuses may not be present, any resection involving free removal of bone at the inner extremities of the supra-orbital ridges or of the orbital margins may perforate the upper portion of the nasal cavities, thus opening a route for infection of the wound if this contingency be overlooked. Where the vault of the pharynx seems to have been traversed by the line of fracture, and the patient is in a condition to stand the requisite manipulations, the cleansing of the parts with boro-salicylic solution by means of the douche, the anterior or posterior nasal spray, or the posterior nasal syringe, with, perhaps, occasional insufflations of iodoform, is indicated: caution must always be exercised lest a dangerous amount of iodoform be used. Thymol, aluminium acetate, and numerous other non-poisonous antiseptic solutions may be substituted for, or serve as adjuvants to, those already mentioned. Wagner (*op. cit.*) has shown that by a resort to these and similar measures twenty-three cases of fractured base which survived for the first forty-eight hours subsequent to the accident recovered. Recently in my own practice I have resorted to similar measures so far as was possible, with the most favorable results, but whether *post hoc propter hoc* or merely *post hoc* it is difficult to say.

TREATMENT OF ENCEPHALITIS.

Since this complication is the chief danger of head-injuries and after operations, its treatment must be here considered,[1] for, although asepsis in operations and antisepsis in injuries will in the vast majority of instances avert intracranial inflammation, yet carelessness on the part of assistants, improper application of methods, imperfect after-dressings, or deeply-planted infection before the injured person comes under the surgeon's hands, will too often bequeath to us this dangerous disease for treatment. It will be noted that no distinction has been made between meningitis and cerebritis, because in practice no such diagnosis can be made, and whichever structure is attacked first the others will sooner or later become involved, if life be sufficiently prolonged; although of intracranial symptoms those indicative of inflammation which commence within a few hours of an injury usually mean meningitis; those supervening four or five days after laceration or incision of the brain with no precedent brain-symptoms probably indicate cerebritis; while symptoms of cerebral abscess usually do not become pronounced until the twenty-fifth day, upon the average.

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[1] For a more elaborate treatment of this subject consult the author's article in the International Encyclopædia of Surgery.

Prophylaxis occupies the first rank. Thus, as there is a variety of meningitis arising directly from the intense congestion of the brain and its membranes from concussion, even without any cerebral contusion, which sets in very early, sometimes even within a few hours, I am in the habit in such cases, unless the patient has lost much blood or is from any cause peculiarly feeble, of enforcing the strictest antiphlogistic diet for the first forty-eight hours: that is to say, nothing but water, or perhaps a very small amount of milk, is allowed. Dry cold should be applied over the dressings and to the exposed portion of the head, which should be shaved if the injury have been a severe one.[1] The room should be darkened, the head kept slightly elevated, and a calomel purge,[2] followed by a saline, at once administered. After the first forty-eight hours, or from the outset if the patient be feeble or have lost much blood, a full milk diet must be substituted for the starvation plan, although only the smallest quantity of nutriment compatible with supporting the patient's strength should be indulged in until after the fifth or sixth day,—*i.e.*, the usual time for the onset of intracranial inflammation. This treatment may seem to many out of vogue, but, when judiciously resorted to, I am satisfied that it is the safest course to pursue, and on this plan I have treated many head-injuries and operations without any indications that I had better have resorted to other measures. *Per contra*, an increase of diet has been repeatedly followed by vertigo, increased headache, and fever, all of which symptoms have spontaneously subsided upon returning to a more meagre allowance of food.

At the first inception of pronounced intracranial inflammation in vigorous patients, wet cups to the nape of the neck, or leeching, will often prove useful, with free purging, repeated from time to time as indicated, always remembering that the patient under treatment is to be considered not as a supposititious "case" of encephalitis for which such and such remedies have been recommended, but as a person whose condition may vary from day to day,—nay, from hour to hour.

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[1] I need hardly point out that in any important operation upon or injury of the head the whole scalp had better be shaved, for many and obvious reasons.

[2] Calomel will remain upon the irritable stomach when other purgatives will be at once rejected.

In times past I have thought that bringing the patient rapidly, but slightly, under the influence of mercury has been of decided benefit, and, with the modern views as to the germicidal action of this drug as an internal remedy in diphtheria, my practice seems all the more likely to have been a correct one.

Sleeplessness or the furious delirium which at times supervenes within a few hours of a head-injury is best treated by the cautious administration of opium. When the heart's action is strong and frequent, aconite and veratrum viride may be found useful, either with or without opium. Chloral and the bromides, as supplementary to opium, or to replace it, will prove useful in cases of excessive wakefulness, and when there is a marked tendency to convulsions.

Blisters and counter-irritants are of doubtful value, and, if resorted to at all, should be used only late in the disease.

A gradual return to a more liberal or to an ordinary diet must be instituted only when, in the judgment of the surgeon, the patient is becoming exhausted, or when all probable danger of a relapse is past.

When stimulants seem required, digitalis, ammonia, and perhaps chloride of barium are better than alcohol. In the later stages iodide of potassium, to promote absorption of inflammatory exudates, and ergot, to contract the paretic and therefore dilated vessels, have proved serviceable in my hands.

In those later and insidious forms of traumatic encephalitis to which the terms subacute or chronic may be applied, where the patient after injury or operation is allowed to continue at or return to school, or even to resume at an early date the boisterous plays of childhood or youth, depletion is out of place, while a more generous diet with judicious laxatives will prove the best treatment, aided by bodily and mental rest.

After any attack of intracranial inflammation, the patient must regard himself as an invalid for a long period, entirely abstaining from school, much reading, or work of any kind, and only gradually resuming these occupations, to relinquish them at once upon the slightest sign of cerebral irritability.

GUNSHOT WOUNDS.

So far as the bone-lesions are concerned, the same operative measures are indicated as have been already detailed for compound skull-fractures. The ultimate evils attendant upon the retention of a foreign body in the brain have been so conclusively shown by Wharton[1] that, if the operation can be done without fatal injury to the encephalon, balls should always be removed, since, even primarily, such injuries are certain to be followed by suppurative inflammation, which too commonly is of the diffused form, when death is inevitable. *Free drainage*, rather than the removal of the foreign body, should be the principle most prominently kept in view, although to effect perfect drainage, both theoretically and practically, *the course of the ball must be followed throughout and disinfected,* when the missile, being accessible, and capable years hence of producing serious trouble, should be removed. The main difficulty in carrying out these indications is a safe method of search for the ball, one which will render it unlikely or impossible to make a false passage, which a resort to the use of any ordinary probe will inevitably result in. Where the ball has done little beyond penetrating the bone and lies close to the osseous perforation, no doubt the ordinary probe in careful hands has proved both efficient and innocent; but even then the aid of *gravity* and that alone must be invoked. Far preferable to the ordinary probe is the use of a small French or Nélaton catheter manipulated in the gentlest manner. Wherever possible, Fluhrer's aluminium probe (Fig. 13) should be used, which is so light that, if allowed to pass along the track of the ball by its own *gravity*, it is absolutely incapable of penetrating the brain-substance. If it can be avoided, this instrument should never be curved, but, when bent, the other extremity should be curved in the opposite direction: this is imperative, since it is the only means of indicating what course the instrument is taking.

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[1] Philadelphia Medical Times, 1879.

FIG. 13.

Aluminium probe.

Method of Searching for and Extracting Bullets, etc., embedded in the Brain.—The importance of a careful examination of ball-wounds of the head is taught by a case of Larrey's. A soldier received a musket-ball wound in the middle of the frontal bone near the sinus, which passed between the cranium and dura mater and along the longitudinal sinus to the occipital suture. It produced all the symptoms of compression. Larrey, inferring the location of the ball from the patient's complaints of pain at a point diametrically opposite to the wound, introduced a gum-elastic sound until he touched the bullet. By measurement, the portion of cranium beneath which the ball lay was exposed, a large trephine was applied, pus was evacuated, the ball was removed from between the dura mater and the bone, and recovery ensued.[1] Here apparent perforation of the brain had taken place, which proper search demonstrated to be an error, the ball being readily and safely removed. The same author reports a second case. A ball penetrated the left parietal eminence of a soldier, passed obliquely along its inner surface, and was arrested about one centimetre from the occipital suture. The presence of a slight ecchymosis near this spot, the symptoms, and the employment of a small soft bougie, induced Larrey to lay bare the bone, when he found a small fissure, applied a large trephine, and removed a piece of the bullet; the patient did well for five days, and then died of a fever.[1]

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[1] Author's article, International Encyclopædia of Surgery, vol. v. p. 73; also Larrey's Memoirs of Military Surgery, Amer. ed., 1814, vol. i. p. 307.

After the usual antiseptic preliminaries, the opening in the bone must be exposed by the reflection of a proper flap, and all loose bony fragments must be removed after enlargement of the opening by trephine, forceps, or Hey's saw. The patient's head must then be placed so that the track of the ball is as nearly vertical as possible, when the probe, gently introduced into the brain-opening, must be allowed to *gravitate* along the ball-track.

When the whole cerebral mass has been traversed without penetration of the opposite cranial wall, the ball usually, according to Fluhrer, although not always, rebounds at an angle equalling that of incidence, and becomes embedded in the brain about one inch above, below, in front of, or behind the point of bone struck, according to the direction pursued by the ball. If the probe shows that the cerebral mass has been completely traversed and that the ball has struck the bone, a large counter-opening is to be made, the membranes carefully incised,[2] and the ball first sought for in the positions which the above-mentioned rule of Fluhrer would indicate. If neither ecchymosis nor a deeper-seated hardness indicate the presence of the missile at this point, careful, methodical search must be made in various directions, remembering the possibility of the ball having lost so much of its initial velocity that, instead of penetrating the brain by its rebound, it may be merely lying *between* the dura and the cerebrum, from which point it may gravitate out of reach if the brain be carelessly depressed or manipulated: in other words, the periphery of the space exposed by the elevation of the membrane should be carefully scrutinized unless local ecchymosis or manifest penetration of the brain exists. When the ball has been found and extracted, and its track and surrounding carefully disinfected by means of the minimum of mercuric solution requisite, or by the boro-salicylic solution, there remain only the drainage and disinfection of the track made by the passage of the missile, which are to be effected in the following manner. A fine antiseptic silk thread is to be attached to one end of the gravity probe, when the opposite extremity must be introduced at the wound of entrance and the instrument withdrawn through the trephine-opening, leaving the thread in the ball-track: by attaching a fine rubber drainage-tube to this thread the tube can be readily drawn through the track, after which its lumen must be cleared by careful syringing with the mercuric or boric solution; nothing is now required but the reposition of flaps and an antiseptic dressing, as indicated under the head of Trephining.

Suppose that it prove impossible to find the ball, then the passage of the drain and the counter-opening will have effected what I have insisted upon as the chief indications,—viz., drainage and disinfection,—and will have done all that is possible towards averting the most fatal complication of these injuries,—viz., diffuse suppurative cerebritis.

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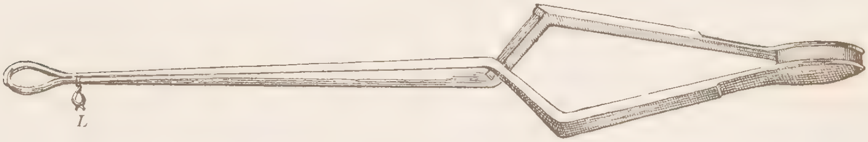
[1] Op. cit., loc. cit.

[2] Any hemorrhage at this or any other stage of the operation must be arrested as indicated in the sections on Trephining and Brain-Tumors, pp. 756, 757, 759, 777, 778.

In the event of the ball being deeply embedded, but not having entirely traversed the whole cerebral mass, the following method of extraction has been suggested by Fluhrer, but, so far as I can ascertain, has not yet been put in practice. The ball having been located by the gravity-probe manipulated as previously described, the instrument should be pushed onward, *alongside* the ball, until it strikes the cranial walls,—provided, of course, no vital cerebral structures intervene,—when its point must be exposed by trephining, and the probe drawn through, leaving two silken threads, which have been attached to its other extremity, in its track. By means of one of these, threaded through the eyes, a small (No. 2 or 3) English catheter with straightened stylet must be made to follow the course of and replace the probe. The distance of the ball from the wound of entrance having been again ascertained by the probe, a delicate pair of self-closing rat-toothed forceps (see Fig. 14), upon one branch of which is securely fixed a loop of fine aseptic silk, must be passed down to the ball by means of the loop *L*, which must travel freely along the catheter. When the site of the ball has been reached, it must be sought below the catheter; if not found there, the forceps should be withdrawn and passed down again to one side, and so on until the whole circuit of the guide has been made, when, if the ball has been previously correctly located, it will be found, and can be removed by the forceps, which must be withdrawn *with the guide.* A rubber drain should now be drawn through the wound by means of the remaining silk thread, and the dressing completed as described for complete penetration of the whole cerebral mass. While seeming a most severe procedure, in appropriate cases I cannot but think that in careful hands the *ultimate results,* if the patients survive, fully warrant the additional risk incurred.

FIG. 14.



Self-closing rat-toothed forceps.

Of course, where the ball is but a short distance beneath the skull, a free removal of bone, incision of the membranes, location with the gravity-probe, and a careful use of the forceps just described, or any other suitable instrument, followed by suturing of the membranes, partial replacement of the bony fragments if deemed necessary, and such a method of drainage as seems indicated, will be all that is requisite.

MALIGNANT GROWTHS OF THE SKULL.

These may primarily affect the bones, or secondarily spread from the overlying soft parts, but the operative technique will be similar in either event. After the usual preliminary antiseptic precautions, an appropriately-formed flap or flaps must be reflected, to obtain the freest possible access to the parts, in those cases where the soft parts are involved cutting wide of the apparent limit of the infiltration, so as to remove the diseased soft parts with the osseous tumor. Having perforated the bone at the most convenient point with a medium-sized trephine, to ascertain the thickness of the skull-case, the remainder of the bony sections requisite to circumscribe the neoplasm can be most rapidly completed by means of the dental engine, which with a small circular saw will rapidly cut nearly through the bone, when strong cutting pliers may be used to complete the section. If the dental engine is not at hand, after the preliminary trephining, a series of repetitions of this procedure, and the use of a Hey's saw, the chisel, or the rongeur, will equally effectually, but more slowly, enable the surgeon to remove the tumor.

If the growth or the necessary bony incisions overlie one of the large cerebral venous sinuses, an opening must be made such as will give free access to any bleeding point or will admit of ligation of the sinus before it is divided; this latter can be readily effected by a trephine-cut on either side of the line of the endangered vessel, followed by the removal of the intervening osseous bridge by the rongeur or chisel *from without inward*, so that a sufficiency of bone can be removed to permit the passage of a needle armed with a double ligature, although a careful adoption of this method of exposing the sinus will in many cases enable the surgeon safely to remove the entire bony surfaces covering the sinus without its ligation. In all these operations the general principles governing the operative treatment of similar neoplasms situated elsewhere should control the surgeon's actions,—viz., early removal and with a free hand. If the latter part of this advice be not strictly adhered to, such operations had better not be attempted, for if timidity gains the upper hand the danger to life is the same, with no reasonable prospect of good accruing. Large portions of scalp, of bone, of the dura mater with its venous sinuses, or even of the cerebral cortex, may require removal.

I cannot do better than briefly relate what some surgeons have done in cases of malignant growths of the cranial bones. Paully[1] "removed the frontal bone from the root of the nose to the zygoma and up to the hair-line; in a month the wound was quite healed." Kronlein[2] removed—successfully, so far as life was concerned—a cancerous ulcer two and one-half inches wide, "extending from the root of the nose beyond the line of the hair," with a piece of the frontal bone and dura mater about three-fourths of an inch in diameter, and, in addition, nearly half an inch of the falx cerebri. Of sixteen cases operated upon with full antiseptic precautions, thirteen recovered from the operation, and three died, one from air-embolism,—an accident which may be prevented by the previous ligation of an endangered sinus, keeping the head low, and irrigating the wound constantly,—*i.e.*, keeping the wound full of fluids, which will be drawn into the wounded sinus instead of air. The dura mater was incised or excised in nine cases; in four operations the falx cerebri was cut; the longitudinal sinus was twice divided; and in two instances portions of brain-substance were removed. In all considerable portions of bone were excised, and in five the gap left measured transversely three inches. These sixteen operations were done upon thirteen patients, in all but two the growth occupying the frontal region. Recurrence took place in only one instance before healing, and rapid recidives in but four instances.

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[1] Verhandlungen der deutsch. Ges. f. Chirurgie, 1883.

[2] Deutsche Zeitschrift f. Chirurgie, 1885, Bde. xxi u. xxii.

ABSCESS OF THE BRAIN.

Since nearly all, if not all, operable cases will be found to result either from a traumatism, with perhaps lodgement of a foreign body, when the site of the injury and the progress of the symptoms will indicate the location of the pus-focus,[1] or from chronic suppurative middle-ear disease, it will be necessary only to detail the operative technique advisable for the latter class of cases, and to emphasize the proper methods of drainage which are imperatively demanded for cerebral abscess wherever situated. Pus in these cases is more often found in the temporo-sphenoidal lobe than in the cerebellum or elsewhere, because the tympanic roof is the thinnest portion of the middle-ear cavity, while more vessels pass from the tympanum and along the squamoso-petrosal suture to the dura mater than from the posterior portion of the petrous bone. Nine-tenths of these abscesses in the temporo-sphenoidal lobe occupy a space three-fourths of an inch in diameter, whose centre is one and a half inches above and the same distance behind the centre of the cartilaginous external auditory meatus.[2] Cerebral abscess is far the most frequent, since, of purulent encephalic collections resulting from chronic ear-disease, three will be found in the cerebrum to one situated in the cerebellum or in other portions of the encephalon.[3]

Operation for Cerebral Abscess.—After the usual antiseptic preliminaries, with, in addition, an attempt to render the tympanum and mastoid cells as aseptic as possible, and the reflection of a proper flap, the crown of a medium-sized trephine should be applied one and one-fourth to one and one-half inches behind and the same distance above the centre of the cartilaginous external auditory meatus (Fig. 15, $C \times$), and worked cautiously, owing to the thinness of the bone here; the disk removed should not show any of the groove for the lateral sinus. After incising the dura mater to a limited extent, the pus must be sought by passing either an aspirator-needle or a grooved director downward, inward, and slightly forward, towards and down to the petrous portion of the temporal bone. When the pus is found, the track, if favorably situated for drainage, must be enlarged by a small Volkmann's spoon, and, a drainage-tube, either of rubber or of silver,[1] having been introduced, the abscess-cavity must be very cautiously irrigated with a weak mercuric or a boro-salicylic solution, or carefully filled with ethereal solution of iodoform.

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[1] Since the operation under these circumstances will be indicated upon localizing and other grounds, I have not mentioned the usual symptoms or the fact that the dura mater does not pulsate and bulges into the trephine; yet this is not a certain sign of anything but increased intracranial tension, and there may be a distant focus of pus with a pulsating brain.

[2] Barker, British Medical Journal, 1887, vol. i. p. 407.

[3] Barker, ibid., December 11, 1886, p. 1155.

If the trephine-opening is unfavorably situated for drainage, in certain instances it is better to make a second bone-perforation opposite the most dependent portion of the abscess-cavity, when the first opening can be closed by its own bone-disk, notched on one side to prevent accumulation of pus or wound-secretions, after which a director must be passed into the abscess from the second trephine-cut, and the enlargement of the track, the placing of the tube, and the disinfection conducted as before suggested: to insure patency of the drainage-opening through the soft parts, a small portion of the edge of the flap should be excised or a piece punched or cut out opposite the trephine-opening, after which careful suturing and the usual dressing must be employed, since primary union of the soft tissues is aimed at, as preventive of the formation of hernia cerebri, a sequel to be dreaded. The tube must be very gradually shortened, and after its final removal the case must be jealously watched, lest a reaccumulation of matter occur.[2] I have given the site selected for perforation by Mr. Barker the preference over that of Mr. Caird (above and in front of the external auditory meatus) and others, because it is safer anatomically, since the middle meningeal artery is out of harm's way, the pus can be evacuated through a comparatively callous portion of the brain, which possesses no motor functions, and, as before stated, the point selected is almost directly over the location of the vast majority of the abscesses of the temporo-sphenoidal lobe.

Operation for Cerebellar Abscess.—In all cases where pus is supposed to be located in the cerebellum, the foramen for the mastoid vein should be examined before perforating the bone, because, "if there be inflammation on the posterior aspect of the petrous bone, it can hardly reach the cerebellum without forming a layer of pus under the dura mater of the lateral sinus. If this is so, the pus will escape by the mastoid foramen if the latter be exposed."[3] When this condition is found,—*i.e.*, escape of pus from the mastoid foramen,—a spot beneath and slightly posterior to the mastoid foramen, below the inferior curved line of the occipital bone, will be the proper spot at which to apply the trephine: in all other respects the directions given for cerebral abscess are strictly applicable here. (See Fig. 15, *E.*)

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[1] Mr. Barker prefers the metal tube, as being easier both of introduction and of retention.

[2] Fenger and Lee, Transactions of the American Surgical Association, vol. iii. p. 65 *et seq.*

[3] Barker, op. cit.

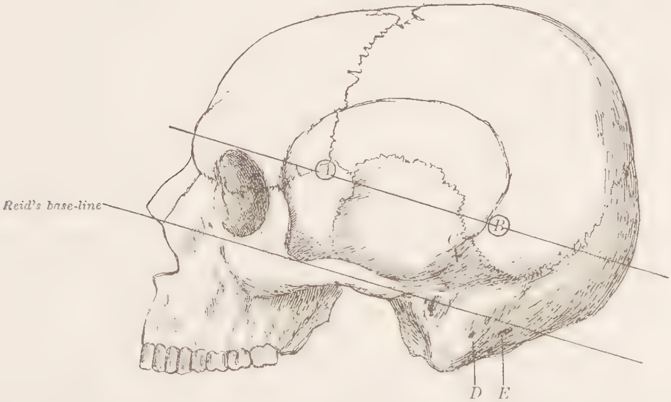
OPERATIONS FOR INTRACRANIAL HEMORRHAGE.

These are practically restricted to cases where the blood-collections—either fluid or clotted—arise from the rupture of the middle meningeal artery or some of its subdivisions, since traumatic intracerebral and subdural hemorrhages in the present state of science cannot be distinguished from diffuse multiple laceration of the encephalon, etc.

In this class of cases I believe, with Mr. Godlee, that chloroform is contra-indicated, while I cannot but believe that ether is likewise prejudicial, and certainly is usually unnecessary, from the semi-comatose or completely comatose condition of the patient.

When a fracture or an external injury exists, this must first be explored, and will in most instances reveal the site of the hemorrhage and its source; but, in cases where neither fracture nor external injury exists, where shall the skull be perforated? Unless specially contra-indicated by some *decided* localizing symptoms which can be explained only by a collection of blood compressing the centre indicated, as the middle meningeal artery or some of its branches are almost invariably the source of the blood, after the reflection of an appropriate flap, the trephine should be first placed from one inch and a quarter to one inch and a half—according to age and size of head—behind the external angular process of the frontal bone, on a horizontal line drawn around the skull at the level of the upper margin of the orbit, parallel with "Reid's base-line" (see Fig. 15). Should this opening reveal neither a clot nor a bleeding vessel, a second opening on the same line just below the parietal boss must be made,—*i.e.*, where a vertical line carried directly upward behind the mastoid process bisects the horizontal line.[1]

Fig. 15.



A, anterior point for application of trephine for intracranial hemorrhage; *B*, posterior point for application of trephine for intracranial hemorrhage; *C*, point to perforate for cerebral abscess following chronic suppurative ear-disease; *D*, foramen for mastoid vein; *E*, point to perforate for cerebellar abscess following chronic suppurative ear-disease.

In one successful case of Kronlein's these two openings were made and a drainage-tube passed through both, while in two cases where the second opening was not made the same surgeon reports, as does also Wetherle,[1] that post mortem a clot was found which might easily have been removed by means of a second trephine-cut. The clot must be carefully removed by the forceps and antiseptic irrigation, after which all accessible bleeding vessels, if in the dura mater, should be secured by ligatures passed with a curved needle, by acupressure, or by touch with a red-hot knitting-needle: where the main trunk of the middle meningeal artery lies in a bony canal, as it often does, a pellet of antiseptic wax or plugging the canal with a sharpened, disinfected wooden match will serve admirably. Where an extensive surface-oozing persists which hot water will not check, I have successfully resorted to antiseptic tamponnade.

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[1] Kronlein, Deutsche Zeitschrift f. Chirurgie, 1886, Bd. xxiii. Hefte 3 und 4.

Where the trephine has been applied at other situations or for hemorrhages which arise from a lacerated sinus or from vessels of the pia mater, —in both instances probably the operation having been instituted for a depressed fracture and not for the evacuation of blood,—the damaged sinus must either be ligated or compressed, while the bleeding vessel of the pia should always be tied with fine catgut when possible, the thread being drawn only sufficiently tight to occlude its lumen, as otherwise its fragile walls will be divided: when the ligature repeatedly cuts through, a plan suggested by Fluhrer, and carried out in one case by myself, will prove effectual,—viz., small serres-fines left on for several days or until spontaneously separated.[2]

Unless through-drainage is clearly necessary, when two openings have been made, one orifice had better be occluded by the replacement of its own notched bone-button, and the more dependent one utilized for drainage. Where neither of the openings is well situated for drainage, I would advise that another bone-perforation, for this special purpose, be made at the most dependent portion of the cavity, or that the nearest opening be enlarged by the rongeur, if sufficiently close, in order more perfectly to effect this all-essential indication of drainage. The reposition of the flap and the use of drainage-tubes, etc., must be regulated by the principles laid down elsewhere.

If after trephining severe secondary hemorrhage occurs, Jacobson recommends first the application of a freezing mixture over the dressings, then placing the patient in the upright posture, next compression of the common carotid artery, and finally, if this fails, ligation of the same vessel.

The latest statistics of this operation[3] show that of one hundred and forty-seven cases of intracranial hemorrhage treated by the expectant plan one hundred and thirty-one died, or about nine-tenths, while of one hundred and ten operated upon, only thirty-six died, or about one-third; moreover, it must be borne in mind that in the majority of those who died after operation the extravasation was not reached, and, of course, not removed; had this been done, the mortality would undoubtedly have been less.[1]

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[1] Northwestern Lancet, November 15, 1885.

[2] Fluhrer, op. cit.

[3] Weisemann, Ueber die Indicationen zur Trepanationen mit besonderer Berücksichtigung, etc., Deutsche Zeitschrift f. Chirurgie, 1885, Bde. xxi. u. xxii.

METHODS FOR THE DETERMINATION OF THE POSITION OF THE FISSURES OF ROLANDO AND OF SYLVIUS.

As a preliminary to the consideration of the methods of operating for brain-tumors, I have thought it best to indicate briefly the readiest methods for determining the position of these two important reference-points, leaving all minuter details for the neurological articles of this work.

The fissure of Rolando is situated about the middle of the outer surface of the hemisphere, commencing at or near the great longitudinal fissure one-half inch behind the mid-point between the glabella and the external occipital protuberance, whence it runs downward and forward at an angle of sixty-seven degrees for its upper two-thirds; it then forms a knee-like angle, becoming more vertical, and terminates a little above the horizontal limb of the fissure of Sylvius: upon either side of the Rolandic fissure lie the ascending frontal and parietal convolutions.

While the plans devised by Broca, Lucas-Championnière, and others are reliable, the readiest method for determining the fissure of Rolando is a simple combination of Hare's, Thane's, and Horsley's as modified by Dr. Morris J. Lewis, of Philadelphia. Hare has shown that, measuring backward from the glabella (or nasion), the upper extremity of the fissure lies 55.7 per cent. of the whole distance between the glabella to the inion (external occipital protuberance) behind the glabella, while Thane has shown that the commencement of the Rolandic fissure is accurately to be determined by taking a point one-half inch behind the mid-point of the line drawn from the glabella to the inion.

The upper extremity of the fissure having been determined by one of the above methods, a line drawn at an angle of sixty-seven degrees (Horsley) will indicate the upper two-thirds of the fissure.

The lower limit lies about one-half inch above the horizontal limb of the fissure of Sylvius, which may be determined in the following manner. By causing the patient alternately to contract and relax the temporal muscle and by tracing the temporal ridge with the finger, the point where the ridge for the temporal fascia crosses the coronal suture (the superior stephanion) must be determined, thence a line must be dropped which shall fall vertical to the middle of the zygoma; the course of the fissure of Sylvius will correspond to a line drawn from the mid-point of the vertical one just described, passing upward and backward (nearly straight for its anterior half, more curved posteriorly) to within about one-half inch of the centre of the parietal protuberance.

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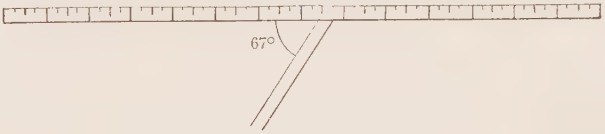
[1] This is a far better showing than that of my statistics collected in 1883, and is unquestionably due to the prevalence of antiseptic methods. Indeed, Kronlein maintains that this operation, aseptically performed, is perfectly safe, death resulting simply from complications unconnected with the operation.

Having determined the mid-point of the bi-aural line and the fissure of Sylvius, provide a half-inch strip of parchment-paper from ten to fourteen inches in length, graduated into inches and fractions of an inch in *both directions from its middle point* (Fig. 16), half an inch behind which is fixed, at an angle of sixty-seven degrees, the anterior margin of a second graduated strip, and proceed as follows. Place the median edge of the long graduated strip against the mid-point of the bi-aural line, and shift it forward and backward until the same figures indicate the glabella (nasion) and the external occipital protuberance. Thus, if the graduated strip indicates five and one-eighth inches at the glabella, it should read five and one-eighth at the occipital protuberance. When these measurements coincide, it will be clear that the anterior margin of the angular strip will correspond to a point one-half inch behind the mid-point between nasion and inion, or 55.7 per cent. of the whole distance between nasion and inion, *behind* the nasion. Nothing now remains but to draw a line along the anterior margin of the oblique arm (carefully held *in situ*) to within one-half inch of the already-indicated Sylvian line, remembering that the lower third of the Rolandic fissure bends somewhat backward from the line indicating the course of its upper two-thirds.

FIG. 16.



Simple form of cyrtometer.

The locations of the different centres with reference to these fissures must be sought in the articles in this work dealing with the localization of the cerebral functions.

OPERATIONS FOR BRAIN-TUMORS.

Although what follows is in part a reiteration of details already given, the importance of attention to minutiæ in such operations warrants their repetition.

The head must be carefully shaved the day before operation, and washed thoroughly with soft soap, followed by ether, or a thorough scrubbing with a nail-brush wet with turpentine one part, alcohol seven parts, may be used instead. The situation of the growth should now be localized and marked on the scalp,[1] after which the head must be covered with lint wet either with carbolic-acid lotion, one to twenty, or with tartacid sublimate solution,[2] one to three thousand or four thousand, with oil-silk and a layer of cotton bandaged over all. A purgative should be exhibited the night previous to, with an enema on, the morning of operation.

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[1] An aniline pencil will sometimes suffice; iodine will not do if carbolic acid be used; while, although it may blister, nitrate of silver exposed to the sunlight is the only *sure* agent.

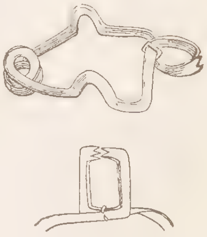
[2] Tartaric solution of mercuric bichloride.

About one hour previous to the administration of the anæsthetic a hypodermic injection of morphine should be given, graduated according to age, or a proper dose may be exhibited by the mouth. Chloroform is preferable to ether, being less apt to produce cerebral excitement; but the following quotation from Mr. Horsley teaches us that great caution is requisite. "But, like all kinds of anæsthesia, it has certain special risks, to which it seems necessary to draw attention. In the first place, the remarkable proclivity of children to the effect of morphine must be properly discounted." . . . In one patient, four years old, one-twentieth of a grain of morphine "was amply sufficient" to contract "the cerebral arterioles. With respect to the administration of chloroform, the one additional fact here to be noted is the extremely important one that it is very easy to give too much in a brief space of time. Thanks to the great care of Drs. Wilson and Stedman, who have assisted me in this respect, I have seen no accident; but I have been very deeply impressed with the startling rapidity with which a patient who has roused up in the middle of the operation is sent off again in a moment with only a few whiffs of the drug. It is perfectly comprehensible, therefore, that an anæsthetist, however careful, might be induced to prolong the administration beyond the point of just sending the patient off again, as, indeed, he would naturally do under ordinary circumstances. Such prolongation, under the present circumstances, is evidently exceedingly dangerous."[1]

The object of administering morphine is twofold,—*i.e.*, less chloroform is requisite, and Schaefer and Horsley's experiments show that morphine contracts the arterioles of the central nervous system, thus lessening hemorrhage.

The strictest attention to asepsis, as regards instruments, hands, and assistants, must be maintained. In addition to the ordinary instruments required for trephining, such as at least two large-crowned trephines, cutting bone-forceps, bone-elevators, Horsley's enucleator, scalpels, hæmostatic forceps, Hey's saws, rongeurs, etc., a pair of blunt-pointed scissors, curved needles threaded with catgut, both to secure vessels in the dura mater and to suture this membrane, a needle-holder for the same, and a few wire serres-fines with threads attached, must be provided. When a dental engine with proper saws, burrs, etc., is attainable, and if the surgeon is accustomed to its use, it may supersede almost all the other bone-instruments.

FIG. 17.



Wire serres-fines.

The patient being anæsthetized, the dressings put on the previous day should be carefully removed, and the localizing points determined, and now marked *in the bone* by driving in a disinfected tack at each extremity of the Rolandic line, or at the same spots cutting a groove or nick with a gouge, or drilling a little with the centre-pin of a trephine. This is an essential preliminary, as otherwise all landmarks will disappear with the reflection of the flap.

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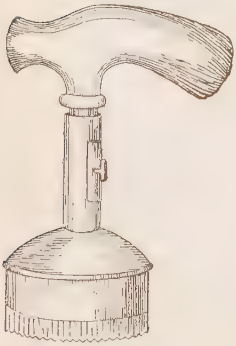
[1] British Medical Journal, April 23, 1887.

Since most of these operations are prolonged, and sometimes entail the loss of considerable quantities of blood, which children bear badly, the encircling of the head with an Esmarch tube should always be tried; but it is not infallible. In one morning I have seen it act perfectly in one operation for epilepsy, in a friend's hands, and in a similar operation of my own conspicuously fail to diminish hemorrhage,—nay, more, it actually increased the loss of blood.

The chief risk being septic encephalitis, Horsley thinks that the spray ought to be used; but I regard it solely as a cleanser of the atmosphere, and think that its use is not absolutely necessary. A free irrigation with bichloride solution just before commencing and from time to time during the operation is all that is requisite.

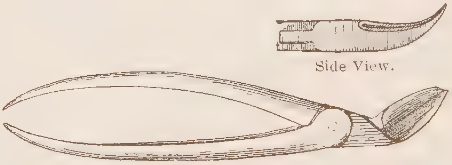
All incisions of the soft parts should be vertical to the skull, and include all the layers at one stroke, not excepting the pericranium.[1] A single flap should thus be raised, its outline being a shallow curve, in order not to divide collateral vessels, and so planned as to avoid the main scalp-arteries. These indications can readily be fulfilled without interference with drainage, since the patient will be in the supine position. All bleeding that does not quickly cease should be arrested by pressure-forceps, by ligature, or by needles armed with gut passed around the vessels in the scalp-tissues, since much blood lost means an unnecessary risk for a child to run. If more bone has to be removed than was originally planned, the periosteum must be carefully dissected back off each new portion.

FIG. 18.



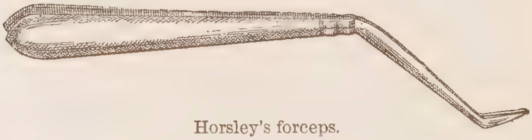
Horsley's trephine.

FIG. 19.



Horsley's forceps.

FIG. 20.



Horsley's forceps.

If the surgeon proposes to use the ordinary trephining instruments, he should now proceed as follows. The trephine-cuts, using an instrument of the diameter of one and one-half to two inches,[1] should be made at the two extremes of the bone-area to be removed, when the intervening bridge can be partially sawn through with a Hey's saw, and the division completed with the bone-forceps. (Figs. 19 and 20.) A far safer and almost as rapid plan for the inexperienced operator—since the bone over some brain-tumors is not thicker than card-board—is to remove the bone with the Hopkins rongeur after one or more preliminary trephine-cuts of the ordinary size. All bone-fragments should be at once placed and kept in a warm antiseptic solution,[2] as it is best to replace them, where it is possible to preserve the dura mater intact.

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[1] This stripping off of pericranium involves no interference with the nutrition of the bone.

Next the dura mater must be cut around four-fifths of the osseous orifice, one-eighth of an inch from the bone, so as to leave room for stitching; start the incision with a scalpel, but complete it with blunt-pointed scissors. Any arteries lying in the line of the proposed dural incision must be tied, by passing ligatures through the dura mater, beneath and around the vessels, with a curved needle, before incising the membrane.

The brain being now exposed, note first if it bulges into the wound,—*i.e.*, is the intracranial tension increased? Next look for a yellowish tinge or possibly lividity, which will denote a tumor in the corona radiata beneath the cortex.

Now closely scrutinize the vessels and perivascular lymphatics, noting especially "any yellowish-white patches in the walls of the latter, indicating old mischief." Finally ascertain whether the brain has undergone any alterations in density, although in most subcortical tumors the diagnosis can be made certain only by an exploratory incision.

Hemorrhage has been much dreaded in the removal of brain-tumors, but the fear is unfounded, for the arteries, and especially the arterioles, which are chiefly concerned in such operations, run perpendicularly to the cerebral surface, and sponge-pressure soon checks all bleeding.[3] Should this fail, ligature of the larger vessels must be tried, the catgut being drawn only tight enough to close the lumen of the vessels. If their walls are too fragile to bear ligation, forci-pressure is alone available. In one case at least,[4] continuous pressure with iodoform-gauze packing failed to prevent fatal recurrent hemorrhage. Fluhrer[5] has shown that the ligature often fails to hold on the vessels of the pia mater, and advises leaving Nunneley's artery-forceps on any bleeding points until the instruments separate of themselves. Preferable to these, as being far lighter, are the wire serres-fines with an attached thread. (Fig. 17.) As the cerebral arteries are terminal, avoid every vessel, as far as possible, this end being often attainable by lifting them out of the sulci between the convolutions, and, after removing the subjacent brain, replacing the pia mater; if any thrombosis occurs it will probably be only temporary.

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[1] See p. 756 for special directions for using these large trephines.

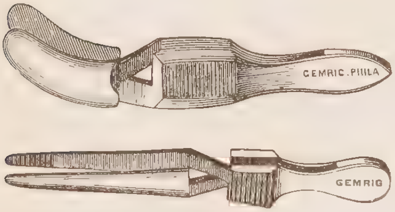
[2] Or between warm aseptic sponges (Horsley).

[3] Clotting is also favored by the presence in quantity in the brain of the lecithin proteid compound discovered by Dr. Woodridge to play a very important part in the process of coagulation.

[4] Birdsall and Weir, Annals of Surgery, August, 1887, p. 149.

[5] New York Medical Journal, March 28, 1885.

FIG. 21.



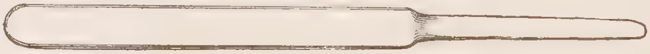
Nunneley's forceps.

If one of the large venous sinuses be wounded during an operation, its ligation is a perfectly safe procedure, healing occurring as in veins, and collateral vessels enlarging alongside of the occluded channel. Sponge-implantation or antiseptic tampons will always temporarily, and often permanently, arrest the bleeding from a wounded sinus, as I have twice seen.[1]

The risk of air-embolism[2] is not imaginary, but can be obviated.[3]

Incisions into the brain must always be clean-cut, vertical to the surface, and directed into the corona radiata, when necessary, so as to avoid damage to the fibres coming from other portions of the cortex or those surrounding the seat of operation. The paths pursued by the fibres from the cortex must be kept constantly in mind when incising the brain, and, where possible, portions of each centre involved should be left, as under such circumstances the coarser movements of the part governed will often be regained.[4] Finally, it is of the utmost importance to be thoroughly familiar with the encephalic blood-supply, in deciding where to make the incisions, in order to have a full understanding as to what portions of the remainder of the organ certainly will be deprived of their blood-supply, and what portions may possibly be rendered avascular. The growth must be enucleated by means of Horsley's flexible knife (Fig. 22), or by the enucleator, aided by the fingers,[5] but not by cutting instruments. "Malignant growths must be excised very freely from among the white fibres," since they chiefly recur in this portion of the brain. In all cases adherent or altered dura mater must be freely cut away.

FIG. 22.



Horsley's flexible knife.

When a portion of brain is excised, the underlying cerebral tissue soon bulges up almost to a level with the cortex, while the cut edges evert, and, if "less brain than bone is removed," protrusion—*i.e.*, an acute hernia cerebri—forms. A persistence of this dangerous condition is prevented by the weight and primary union of the scalp-flap : the advantages afforded by the large one recommended are thus apparent.

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[1] See author's article "Head-Injuries," International Encyclopædia of Surgery; also Hopkins, Annals of Surgery, July, 1885, p. 65.

[2] See Senn on Air-Embolism, or Berlin. Klin. Wochenschr., 1881, p. 673.

[3] See page 769.

[4] See Hughlings Jackson, British Medical Journal, 1886, vol. ii. pp. 670–675; Keen, American Journal of the Medical Sciences.

[5] See Birdsall and Weir's case; Nancrede, Medical News, November 24, 1888.

After all oozing has been arrested by gentle sponge-pressure or by ligatures, the flap of dura mater, if the operative procedures have left it intact, should be carefully sutured with chromic catgut to the margins of the membranous opening, leaving space for the easy passage of a drainage-tube at the most dependent point. Next the bone-disks—*if the dura be intact*—should be arranged and secured, after Keen's method (see p. 757), or minced up, after the manner of Macewen, and strewn over the membrane. The scalp-flap should then be laid down and secured by stitches of medium-sized silk placed about one-third of an inch apart, with horse-hair sutures between, after which the whole head must be enveloped in a voluminous antiseptic dressing.

When brain-substance has been removed, drainage should not be kept up more than twenty-four hours, because firm union must be secured within four or five days, and a certain degree of pressure should in the mean time be exercised upon the brain which tends to protrude, in order to avoid hernia of the organ. Again, there should be some protective between the brain and the scalp, in the form of soft—*i.e.*, non-inflammatory—connective tissue. Briefly, all these desirable results are obtainable by the following measures :

1. Place a tube at the most dependent portion of the wound when the patient lies supine, for twenty-four hours only, to drain all blood and serum. Horsley now says that he sews "the wound all around closely except for one inch at the most dependent part, where any tension of wound-discharge can relieve itself by escape between the edges ;" but I still think the original plan the safer, especially for inexperienced operators.[1]

2. After twenty-four hours remove the tube, and redress, as at first, antiseptically, making firm but gentle pressure over the centre of the flap.

3. If on the third day pain and throbbing in the wound be complained of, and the flap, on exposing the wound, be found "distended in the centre, the periphery being firmly united," undue collection of wound-fluids has taken place. If it appear probable that the pressure will break down the union of the flap, the track of the drainage-tube must be gently opened up with a probe, and some of the pent-up fluid let out ; if the union seem safe to hold, let the accumulation alone, since after the evacuation of the fluid the advantages of supporting pressure are lost. The tension at most usually requires to be relieved but once. The liquid cushion, until absorbed, represses the tendency to hernia, promotes absorption by the meningeal lymphatics of all inflammatory exudates, thus favoring rapid union, and serves as a scaffolding for the formation of normal connective tissue, which is developed in a few days, at least in the lower animals.

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[1] Horsley's second paper.

After from five to seven days, boric acid cotton, and collodion for support, are better than the ordinary antiseptic dressing. The stitches can be removed any time after the first week.

After-Treatment.—The diet for the first three days should be liquid, which is sometimes productive of a furred tongue and an unpleasant condition of the mouth. Horsley recommends for this thorough brushing of the mouth several times a day with strong solution of chlorate of potassium. Solid food may be given as soon as the patient asks for it. In a comparatively trifling case the patient, Horsley thinks, can be safely allowed to get up at the end of a week, ten days being a good average for the stay in bed, with two weeks for severe cases. I cannot but think that children had better be restrained of their liberty for at least three weeks, unless something contra-indicates such confinement. Where large portions of skull have been removed and not replaced on account of excision of the dura mater, if deemed necessary, a moulded, perforated celluloid cap may be worn over the part.

Should a second operation become necessary, or a primary one for brain-injuries which have resulted in scars of the cortex, especially those produced by traumatic losses of brain-substance which have healed after free suppuration, great caution must be exercised, since the cicatrices often displace large vessels, and are traversed by veins of considerable size.

CYSTS.

These should, after the same precautions as advised for cerebral tumors, be carefully curetted and drained, preferably by the tube, but recognizing the risk of hernia cerebri by this method of treatment.

HERNIA CEREBRI.

In this connection the question of prevention of hernia cerebri should be considered, since this is one of the complications quite possible after any operation which compromises the integrity of the dura mater, such as compound fractures, ball-wounds, etc., but is still more likely to occur after the removal of a brain-tumor. As has already been pointed out, if less brain than bone be removed, in a short time the excavation in the brain partially or totally disappears, in which latter event there is actual eversion of the edges, forming an acute hernia cerebri. All further tendency to undue protrusion is, however, repressed by the method of drainage, the suturing of the dura mater, the heavy flap, and the after-method of dressing[1] in a properly-conducted operation for brain-tumor or epilepsy. In like manner, after trephining for compound fractures, suture of the lacerated membranes, and in all varieties reposition of the bony fragments, with modified drainage, as already pointed out, will in most instances prevent any protrusion; but there are certain cases, as ball-wounds and two accidental conditions which may complicate any head injury or operation, which must be mentioned. The first complication is when, for any reason, the flap of dura mater and scalp fail to unite, or their adhesions are purposely broken down to release accumulated wound-fluids, as in a case reported by Keen,[1] and a decided hernia cerebri occurs, which, if properly treated, is, in my experience, of very little moment, but which will terminate disastrously to the patient if the therapeutics in vogue a few years since are resorted to. The strictest attention to antiseptic or rather aseptic measures, with the gentle support afforded by the dressings, combined with the recumbent posture and non-stimulating diet, will in the vast majority of instances result in the gradual subsidence of the protrusion and cicatrization of the wound.[2]

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[1] See p. 779.

The second accident is one which may or may not be beyond the control of the surgeon,—viz., septic inflammation of the brain. If the operation be one for epilepsy, brain-tumor, intracranial hemorrhage without fracture, etc., it is almost invariably, if not invariably, the surgeon's fault, either during the operation or at some subsequent dressing; while a compound fracture, a ball-wound, an abscess, or a suppurating malignant growth may render it impossible to keep the wound aseptic. The importance of preventing any form of cerebritis will be apparent from a moment's consideration of the pathology of hernia cerebri. I have already pointed out the fact that when the support afforded to the brain-tissue by the bone and membranes is removed there is a distinct tendency to protrusion, especially if the slight barrier afforded by the pia mater is removed by excising a portion of the cortex; in other words, a brain of normal consistence is forced into and partially *through* any bony hiatus in the skull by the normal intracranial tension. Let this normal tension become abnormally great,—all the more if the texture of the brain-tissue is softened by the same cause,—and at once all the conditions for a hernia cerebri are present, and it will inevitably form, provided the soft parts, by their union or by their weight, do not mechanically prevent it. Finally, the growth is checked, and recession and cure of the hernia are effected, by the formation of a layer of granulation-tissue, which by its transformation into cicatricial tissue so compresses the protrusion as to force it within the brain-case. It hardly seems necessary to point out that cauterization, ligation, or excision will remove the only barrier—the granulation-tissue—which prevents further protrusion, and which must form again before a cure can be effected. Strict asepsis, or antisepsis when infection has occurred, is the best preventive treatment for this complication, in conjunction with the suturing of membranes, modified drainage, and support before advocated, while absolutely *nothing* should be done to the protrusion, beyond its disinfection, antiseptic dressing, and *gentle* support, combined with those constitutional and local measures suggested when describing the treatment of intracranial inflammation.

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[1] See Transactions of the American Surgical Association, vol. vi. p. 293.

[2] My subsequent much-extended experience more than justifies the favorable opinion which I expressed more than six years ago, in the article on Injuries of the Head, in the International Encyclopædia of Surgery, vol. v. p. 67, bearing on this subject.

TAPPING FOR HYDROCEPHALUS.

I have been in the habit of giving a dose of opium, proportionate to the age, about one hour before this operation. The head should be surrounded with a many-tailed bandage, which must be drawn upon as the fluid is evacuated, in order to afford support to the intracranial circulation; the various tails should be snugly pinned at the conclusion of the operation, so as to keep up the support, supplemented, if needful, by compresses held down by a few broad strips of adhesive plaster crossing the vertex, to afford more equable pressure. An elastic bandage may be used instead of the many-tailed bandage, both during and after the operation, but it must be put on with the minimum of tension, be most carefully watched, and be removed upon the slightest evidence of undue intracranial tension.

With an elastic or the many-tailed bandage, after due aseptic precautions, an ordinary fine exploring trocar and canula may be used, but, when possible, the aspirator-needle should be preferred, as making a slighter wound while evacuating the fluid with equal, or, indeed, greater, certainty. The amount of fluid to be withdrawn at any given tapping must be judged of for each case at the time of operation. I have removed many ounces, and have stopped only when I thought that, the bones being incapable of further approximation, it would be difficult to keep up due intracranial pressure on the blood-vessels; if this is not done, of course a rapid re-accumulation of the fluid will occur. After the withdrawal of the needle or canula, nothing but a little cotton soaked with iodoform or ordinary collodion will be requisite to heal the puncture.

PERMANENT DRAINAGE OF THE VENTRICLES.

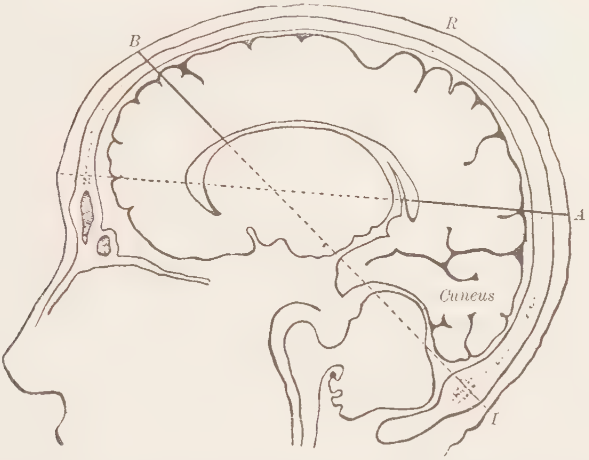
This operation, recently proposed by Dr. W. W. Keen, has as yet been too seldom tried to decide as to its value. Still, as one likely to be suggested and attempted by bold operators, I have thought it best to give its details according to its originator.

The motor zone of the brain must be avoided,—the neighborhood of the Sylvian fissure,—because of the middle meningeal and cerebral arteries, and also because a puncture here would involve the island of Reil and the basal ganglia,—and all known sense-centres, utilizing, as far as possible, only the so-called "latent zones." From these considerations, Dr. Keen has been led to propose the three following routes:

"i. Trephine half-way from the inion (the external occipital protuberance) to the upper end of the fissure of Rolando, one-half to three-quarters of an inch to either side of the middle line. Puncture towards the inner end of the supraorbital ridge of the same side (Fig. 23, *A*). The puncture will pass through the precuneus, and the normal ventricle will be struck at some point in the posterior horn at from two and a quarter to two and three-quarters inches from the surface of the scalp.

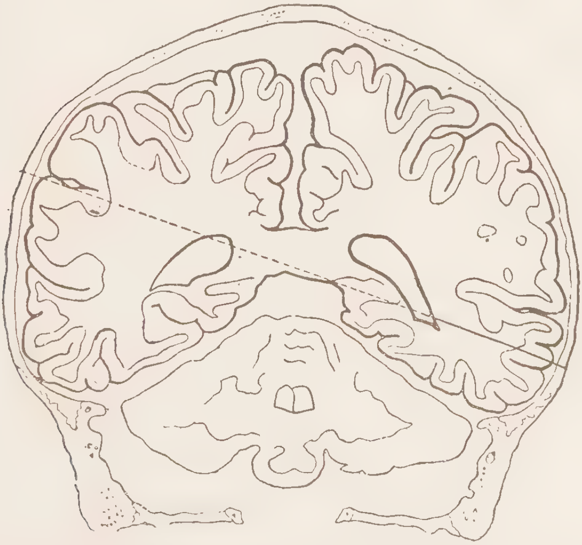
"ii. Trephine at one-third of the distance from the glabella to the upper end of the fissure of Rolando, and half to three-quarters of an inch to either side of the middle line. Puncture in the direction of the inion (Fig. 23, *B*). The puncture will traverse the first frontal convolution well in front

FIG. 23.[1]



ANTERO-POSTERIOR SECTION OF THE HEAD ONE-HALF INCH FROM THE MIDDLE LINE.—*R*, fissure of Rolando; *I*, inion; *A* and *B* (solid), the lines of puncture, the dotted lines showing their imaginary continuation to the opposite fixed points. (Drawn by Dr. J. M. Taylor.)

FIG. 24.[2]



Transverse section of the head one and a quarter inches behind the meatus. The continuous line shows the line of puncture, the dotted line its imaginary continuation to the opposite side of the skull. (Drawn by Dr. J. M. Taylor.)

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[1] From Dr. Keen's paper.

[2] Ibid.

of the motor zone, and the normal ventricle will be struck in the anterior horn at about two to two and a quarter inches from the scalp.

"iii. Trephine one and a quarter inches behind the meatus, and one and a quarter inches above Reid's base-line. Puncture towards a point two and a half inches directly above the opposite meatus (Fig. 24). The puncture will traverse the second temporo-sphenoidal convolution, and enter the normal lateral ventricle at the beginning or in the course of the descending cornu at a depth of about two to two and a quarter inches from the surface. . . . In this third route the measurements are for an adult skull, and are to be somewhat reduced for children. In a distended ventricle from effusion the distances would be proportionately less.

"An inch or a half-inch trephine opening having been made, the dura should be examined. If it pulsates, bulges in the wound, and is tense and elastic to the touch, it will confirm the diagnosis; should it be tense, elastic, and bulging, but not pulsate, abscess or tumor should be suspected and sought for. The diagnosis of dropsy of the ventricles having been confirmed, the dura should be incised crucially and the grooved director now be introduced in the direction and about the depth above indicated, unless fluid is found more superficially. If the first puncture does not reveal fluid, a second or a third may be made. When found, it should be evacuated by the introduction of a dressing or hæmostatic forceps. A drainage-tube should then be introduced and retained for from twenty-four hours to such time as the surgeon sees fit to attempt its permanent removal."[1]

The rest of the treatment will be such as before indicated after operations involving incision or excision of portions of brain-tissue.

CRANIECTOMY.

This operation has been recently devised and put successfully into practice by Prof. Lannelongue for microcephaly. He believes that the partial idiocy and various paretic symptoms common in such cases are the results of cerebral lesions due to pressure. An antero-posterior median incision, extending from the frontal to the occipital suture, should be made down to the bone, the soft parts separated, and a strip of bone about one-half inch in width removed from the left parietal bone, close to, but avoiding wounding, the superior longitudinal sinus. Of course this bone-excision may be extended forward into the frontal bone, or backward into the occipital, if deemed advisable. Great care must be exercised not to wound the dura mater, the bone being removed, after a preliminary trephining with a small instrument, by means of a Hey's saw, the chisel, or the rongeur. The superficial wound must be closed by sutures, and an antiseptic dressing applied, as elsewhere indicated, no drainage being requisite. If preferred, capillary drains may be introduced.

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[1] Medical News, December 1, 1888.

SURGERY OF THE SPINAL CORD.

This practically resolves itself into trephining—so called—for injury, and a similar operation for extra- and intra-dural tumors or possibly for other forms of pressure on the cord. With regard to the advisability of these operations I have nothing to say, as this point is discussed elsewhere.

TREPHINING OF THE SPINE FOR FRACTURE.

This must be done with the strictest aseptic precautions. The patient had better be turned only three parts over during the anæsthesia. An incision about four inches in length, with its centre opposite the most obviously damaged spinous process, must be made, and the muscles separated by the knife-handle, elevator, and division by the knife of resisting tendinous slips until the laminæ are reached. The lips of the wound should be kept apart by retractors, and each apparently-damaged spinous process seized with the forceps and gently rocked to ascertain if it is loose, when it must be carefully dissected out. If no spine is loose, the laminæ must be divided, by strong cutting forceps or a Hey's saw, on either side of the spinous process, the laminæ on both sides being cleared of their muscular coverings, and the severed bone carefully elevated. Should a fragment of a vertebral body be found projecting backward into the spinal canal, efforts may be made, with a strong director or a female catheter, to force the bone into place. All easily-removable blood should be got rid of, and, after the flushing of the wound with an antiseptic solution and the arrest of all hemorrhage, tube-drains must be used.

OPERATIONS FOR EXTRA- AND INTRA-DURAL TUMORS; INTRA-DURAL SECTION OF ROOTS OF NERVES; INTERNAL PACHYMENINGITIS, ETC.

I cannot do better here than quote the details of two cases operated upon by Dr. Abbe, of New York, and one operated upon by Dr. J. W. White, of Philadelphia.

Extra-Dural Tumor.—" An incision was made, six inches long, close to the spinous processes, from the sixth to the twelfth dorsal. The laminæ being uncovered, some thick broken-down tissue and pus were scraped away. The laminæ were already bare of periosteum, and the spines and laminæ of the eighth, ninth, and tenth were cut away by rongeurs. This revealed a dense mass of tissue and desiccated pus, occupying the entire calibre of the canal and extending up and down for a distance in all of two and a half inches. This compressed the cord tightly against the anterior wall. It was rapidly and thoroughly removed by a sharp Volkmann spoon, until sound bleeding tissue was left on every side. The cord was not seen to pulsate. The cavity was packed with iodoform gauze, after bichloride irrigation, and an antiseptic dressing and plaster-of-Paris jacket applied."

SECTION OF POSTERIOR ROOTS OF NERVES FOR INTRACTABLE NEURALGIAS.—I shall again quote a case of Dr. Abbe's, which in all its essentials would do equally well for the removal of an intra-dural tumor.

After laying bare and removing the laminæ on the right side, the dura, pulsating, as it should in health, was punctured with a knife; a fine director was slipped in, and the spinal fluid was slowly let out until it ceased to run. "Then I slit up the dura for one and a half inches. The cord and membranes looked sound." The roots of the nerves requiring section were then hooked up and divided. "The slit in the dura was now sutured with fine catgut." In similar cases the wound may be either packed for the first few days with iodoform gauze and then closed, or closed and drained by the tube from the first, the former, however, being the preferable practice.

Dr. White, after careful preliminary antiseptic precautions, operated with the patient in the semi-prone position, a small flat pillow under the sternum serving to throw out prominently the dorsal spinous processes. A median incision was carried down to the bone; "the ligaments and muscular masses occupying the vertebral gutter" on either side were rapidly separated by means of the knife and a curved periosteal elevator. All hemorrhage from the muscular branches was arrested by hæmostatic forceps, and free access was obtained to the deeper parts without any transverse division of the deep fascia by forcibly separating the tissues by means of flat rectangular retractors with blunt serrated edges. The bases of the spinous processes of the fourth and fifth dorsal vertebræ were next cut through by angular bone-forceps, "greatly enlarging the field of operation."

With other angular forceps the laminæ of the fifth dorsal were cautiously divided by "small bites," first on one side, then on the other, and as near as possible to the transverse processes, "after which the attachments of the vertebræ to the fourth and sixth were divided by the same forceps, cutting transversely to the axis of the spinal column." A few touches of the knife and scissors curved on the flat, while the fragment was held by the lion-jaw forceps, served for its removal, which gave fair access for exploration with the tip of the little finger to the lateral and antero-lateral aspects of the cord. The dura mater was then seized with toothed forceps, nicked, and divided with scissors to the full length of the incision. At places, particularly towards the upper angle of the wound, it was very adherent by new fibrous tissue to the pia mater and cord. All accessible adhesions having been gently separated, the dura mater was united with interrupted catgut sutures, introduced at intervals of about one-fourth to one-third of an inch by means of long-handled staphylorrhaphy-needles; "a medium-sized rubber drainage-tube was then laid in the wound, its ends projecting at each extremity. The muscles, including the deep fascia, were then brought together by chromicized catgut stitches, and the skin and subcutaneous structures by silver wire," after which an ordinary antiseptic dressing was applied, requiring at first daily renewals, because of the free oozing of cerebro-spinal fluid.

The severe venous bleeding which may occur during these operations from the bone or large spinal veins, when the bleeding vessel cannot be ligated or secured by forci-pressure,[1] requires careful packing with iodoform or other antiseptic gauze.

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[1] See pp. 777, 778.

DISEASES OF THE PERIPHERAL NERVOUS SYSTEM.

By JOHN VAN BIBBER, M.D.

It will be unnecessary, in considering the peripheral diseases of the nervous system in children, to describe or dwell upon the anatomy and physiology of the special part of this system under consideration. It is essential, however, to the full understanding of this subject to call attention to the great increase of nervous diseases among children of all ages, and to the increased irritability of this part of the system at a very tender age, and, in fact, to notice the development of diseases which heretofore have not been observed as prevalent during the early years of life. Hence disease of the nervous system in childhood is at the present time a most interesting subject, for we are now called upon to see an increased number of little patients suffering from nervous diseases of types and severity before unknown, and the question at once confronts us as to whether the tendency is inherited from more nervous parents, or whether the present mode of life and education produces this increased sensitiveness directly in the children themselves.

In an article "On the Increase of Nervous Diseases among School-Children,"[1] I came to the conclusion that the strain and worry of school-life were in a measure responsible for the increase of nervous diseases among children, and I then contended that over-pressure in school-life had much to do with the early development of migraine and other neuroses which were heretofore noticed only in adult life. Among the conclusions reached in the paper, I quote the following: "It has been alleged that an enthusiasm for hygiene has led the medical profession to attribute many of the ills of childhood to the effects of education and confinement in badly-ventilated schools. But I do not think any impartial observer, professional or lay, can study the statistics collected in this paper without being impressed by the increase of nervous diseases in children and the apparent connection between the development of these unusual maladies and the gradual extension of the public-school system. These two facts stand to me in the light of cause and effect, and, though I should be the last one to cast any aspersion on the ennobling influence of education, still I cannot shut my eyes to the very decided evil consequences which undoubtedly follow a too early and strict enforcement of the present system. . . . These children are to be the men and women of the future, they are to represent the average health of our population, and, if on the threshold of life they are forced into a form of disease which leaves a lasting impression on their constitutions, I do not think the national type can escape decided deterioration."

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[1] Archives of Pediatrics, vol. ii. No. 2, February, 1885, p. 95.

It will be seen in the following short summary of peripheral nervous diseases that the literature of the various subjects is full and interesting, and that this special branch of nervous diseases is receiving an adequate share of attention from the thinkers of the profession. In a general way I think it will be found true that in children of tender age the central system is more sensitive than the peripheral, and that morbid processes are more likely to be developed in the central nervous system and in the meninges. Yet from the following table of peripheral nervous diseases the reader will see that the list is quite formidable, and much too long for the limited space which can be allowed to its consideration in this volume:

- Neuralgia . . . .
  - Growing-pains,
  - Migraine,
  - Intercostal neuralgia.
- Peripheral Neuritis.
- Facial Hemiatrophy.
- Peripheral Spasm . . . . . .
  - Eclampsia,
  - Tetany,
  - Nodding spasm,
  - Saltatory spasm,
  - Habit spasm,
  - Torticollis,
  - Tremor.
- Peripheral Paralysis . . . .
  - Facial paralysis,
  - Extensor paralysis,
  - Reflex paralysis.
- Diphtheritic Paralysis.

NEURALGIA.

Neuralgia is comparatively rare in infancy and childhood, and, when it occurs, usually presents the same features as in the adult. It will suffice in this article to draw attention to two only of the neuralgiæ,—migraine and intercostal neuralgia.

The so-called growing-pains in young children might perhaps be classed here, although but little has been written to elucidate their real nature. According to Jacobi,[1] they are probably referable to anæmia and imperfect nutrition. Apart from this condition they are most probably due to the great activity at this time of life, and are more pronounced after prolonged and violent exercise. The use of warm alkaline baths—at 98° F.—alleviates this form of neuralgia temporarily, and will be of permanent benefit if systematically kept up for some weeks.

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[1] Pepper's System of Medicine, vol. v. p. 1217; Anæmia of Infancy and Childhood, A. Jacobi, Archives of Medicine, vol. v., 1881.

MIGRAINE.—**Synonymes.**—Hemicrania, Megrim, Sick-headache.

Definition.—Migraine is a paroxysmal neurosis, characterized by attacks of unilateral headache, nausea and vomiting, disturbed sensation, special and general, and vaso-motor disturbances.

Etiology.—It is of frequent occurrence in childhood, in a large proportion of cases commencing about puberty. According to Gowers,[1] one-third of all cases arise between the ages of five and ten, and two-fifths between ten and twenty. Henoch[2] finds that in Berlin it has become much more frequent of late years, which he attributes to the increased exactions of modern education. All authors agree as to the predominant influence of circumstances that lower the general health, and particularly those that act upon the nervous organism. Over-study is a frequent source of the disease, especially when connected with over-crowding in school-houses and with insufficient recreation. It prevails more in the city than in the country. Children who are its subjects usually have a nervous temperament and are often very "bright." The nervousness may be inherited, or acquired through improper training. The influence of heredity is generally noted: Gowers (*op. cit.*) found evidence of it in more than one-half of his cases. This may be shown either by a direct inheritance—a member of the family, not infrequently a parent, having suffered from the disease—or indirectly by the occurrence of other forms of nervous disease in the family. Several children in a family may be affected simultaneously. Among the affections especially associated with migraine are epilepsy, neuralgia, and insanity. Anæmia favors its development, and it is part of the hysterical diathesis. Gowers (*op. cit.*) notes a connection with the gouty diathesis, and says that it sometimes becomes transformed in the adult into gout. Henoch (*op. cit.*) has seen it disappear after the passage of round worms. Irritation of the genital organs, as from masturbation, is a possible etiological factor. In those subject to the disease, individual paroxysms are readily excited by over-exertion, either physical or mental, mental excitement, and indigestion. Any sudden impression upon the special senses, as a bright light, a loud noise, or a strong odor, will often induce an attack. Individual experience shows that certain articles of diet are specially causative of attacks in certain persons.

Pathology.—There are no known anatomical changes. The hypothesis most prevalent regarding the nature of the disease is that it is seated in the sympathetic nervous system; but Strümpell[1] points out that the presence of the vaso-motor symptoms may be but a reflex of the pain. According to this hypothesis, the pallor and superficial contraction of vessels indicate an irritation of the sympathetic (spastic form), and the superficial congestion, etc., a paralysis of the same (paralytic form). Another view, which has been especially maintained by Liveing,[2] is that the derangement is in the nerve-cells of the brain, the vaso-motor disturbance being secondary. Strümpell (*op. cit.*) believes the pain to be situated in the membranes of the brain. Flint[3] supposes that there is a toxic agent in the blood. Gowers (*op. cit.*) lays stress on the close resemblance in some of its features to epilepsy, particularly *petit mal.*

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[1] A Manual of Diseases of the Nervous System, by W. R. Gowers, M.D., F.R.C.P., Philadelphia, 1888.

[2] Lectures on Children's Diseases, by E. Henoch, vols. i. and ii., New Sydenham Soc. Trans., 1889.

Symptomatology.—The paroxysms recur at varying intervals, usually of from two to ten weeks. They have been known to occur daily; also to exhibit great regularity, as noted by Trousseau. The individual paroxysms last from a few hours to two days. They are preceded by certain prodromes, which often indicate to the sufferer the oncoming of his customary attack, such as a feeling of malaise, fulness about the head, vertigo, tinnitus aurium, yawning, chilly sensations, and spots before the eyes. Often the attack is ushered in by a visual aura, as a bright spot or sudden dimness, or by transient aphasia, or by a tingling sensation in the arm or elsewhere. Frequently the patient awakes in the morning with the headache. This increases in intensity. It is situated in one temple, or in the parietal region; sometimes it is frontal. It may affect the two sides alternately. According to Henoch (*op. cit.*), the unilateral feature is not so commonly observed in the child as in the adult. The pain is continuous,—not intermittent, as in ordinary neuralgia; it is increased by noise and light. The affected side of the head is usually hyperæsthetic. The characteristic vaso-motor symptoms accompany the pain. The face and ear on the affected side are often pale and cold, with dilated pupil and increase of salivary secretion. Less often there are redness, heat, and congestion, dilated arteries, local sweating, and contracted pupil. A condition of the cerebral circulation corresponding to these external phenomena is inferred. But all cases do not conform to these types, which may be mixed, or alternate, or be entirely absent. There is anorexia, with general sensitiveness to light and sound. Ocular disturbances, as bright scintillations and even hemianopsia, are frequent accompaniments. The headache is succeeded after some hours by nausea and vomiting, the sufferer falls asleep, and when he awakes he is entirely free from pain. According to Gowers (*op. cit.*), there may be a considerable degree of pyrexia in the migraine of childhood. The health in the intervals is good. The disease is chronic, lasting for years, and the patient usually becomes accustomed to the attacks. It is sometimes transformed into other neuroses, as neuralgia, gastralgia, laryngismus stridulus, angina, and paroxysmal insanity; but the most important and frequent of its transformations is into epilepsy, which it so much resembles.[1]

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[1] Text-Book of Medicine, A. Strümpell, Amer. ed., 1887.

[2] Megrim, Sick Headache, and Some Allied Disorders, by E. Liveing, London, 1873.

[3] Principles and Practice of Medicine, by A. Flint, 6th ed., Philadelphia, 1886.

Diagnosis.—The chief difficulty in diagnosis will be in connection with organic cerebral disease and *petit mal.* The former is sometimes for a long time characterized only by paroxysms resembling those of migraine, but usually the intervals between attacks are shorter and the relief is less complete. The visual aura which sometimes characterizes cases of epilepsy is brief, lasting only a few seconds, thus contrasting with that of migraine, which is at least several minutes in duration.

Prognosis.—The prospect of cure is poor, but, by prolonged treatment and great hygienic care, relief may be expected, especially if there be a definite cause which can be removed.

Treatment.—The etiology must be taken into consideration. Attention should be directed to the diet, mental strain should be removed, and recreation out of doors insisted on. Change of climate to the sea-side or mountains will often prove very beneficial. More reliance must be placed on hygienic treatment than on the action of remedies. Among those which have proved more or less palliative are guarana, caffeine, bromide of potassium, chloral, antipyrin, emetics, hypodermatic injections of morphine, sedative liniments, and hot mustard foot-baths. The bromide is more efficacious in the paralytic (congestive) form. It may be frequently given during an attack, combined with Indian hemp. Strümpell (*op. cit.*) especially advocates salicylate of sodium in strong *café noir.* In the spastic form Gowers (*op. cit.*) recommends nitro-glycerin in liquid form—one per cent. alcoholic solution—after meals, combined with gastric stimulants, as hydrochloric or phosphoric acid, pepsin, and tincture of nux vomica. The galvanic current has been recommended, but is at best only palliative. A mild current, ten to fifteen cells, passed from forehead to occiput, or through the sympathetic, may reduce the severity of the attack. The main dependence, however, must be on a prolonged hygienic course of baths, frictions of the skin, exercise in the open air, avoidance of worry and fatigue, and careful exclusion of all articles of diet found by experience to produce attacks. The use of pulv. guaranæ, in doses of from twenty to thirty grains, at the onset may abort an attack effectually.

INTERCOSTAL NEURALGIA.—Intercostal neuralgia acquires a special importance in the child from its connection with Pott's disease. Being one of the earliest symptoms of that affection, it possesses great value in the diagnosis at a time when localized symptoms may be lacking,[2] and when remedial measures are urgently needed and are most promising of results. It is characteristic of irritation communicated to the trunk of a sensory nerve at any part of its course, that the sensation is felt not at the point of irritation, but at the extremity, often remote therefrom. Accordingly, disease of different regions of the spine will produce effects in accordance with the distribution of the nerves which go out from it. In disease of the superior extremity of the vertebral column,—for instance, the atlas or axis,—pain will be experienced in the region of the occiput. When seated lower down in the cervix, it will be felt in the shoulders, in the arms, and at the upper part of the sternum. When occupying the dorsal region, it will be located in the middle line of the chest anteriorly, in the epigastrium, or in the intercostal spaces at the sides. In disease of the lumbar vertebræ it will be referred to the pelvis and down the lower extremities. Under all these various circumstances the pain is increased by locomotion, especially if of an active sort, since the jar to the diseased part is then greater, and relieved by a recumbent position. According to Roberts,[1] it is worse at night. Sometimes the pain is fixed, sometimes it is lancinating like that of locomotor ataxia; sometimes it is accompanied by a sensation of constriction. It varies in degree in different cases, and in exceptional cases may be wanting.[2] Sometimes cardiac palpitations and irregular breathing accompany the pains, and indigestion may be present.[3] In lumbar disease there may be tenderness on pressure at the sides of the umbilicus, and abducting the thighs increases the suffering.[4]

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[1] Pepper's System of Medicine, vol. v. p. 1231.

[2] "The pain" (in Pott's disease) "is referred to the spinal column only in very rare instances."—*Cyclopædia of the Diseases of Children*, vol. iii. p. 1024.

The source of these pains is believed to be the irritation and compression of the roots of the nerves as they emerge from the intervertebral foramina. They may be due to an actual neuritis, arising from extension of the inflammatory process from the dura, or from injury inflicted on the nerve. The pains are liable to be misunderstood. If in the abdomen, they may be considered as "stomach-ache," or hepatic or nephritic colic; if in the limbs, mere "growing-pains."[5] In all cases, therefore, of persistent pain about the abdomen in the child, an examination should be made of the spine.

The treatment of this neuralgia constitutes one of the most important prophylaxes in medicine, for if recognized early and treated promptly we may be able to save the patient from a life of suffering and deformity. As the pain is due to disease or irritation of the vertebræ, all treatment, after the diagnosis has been made out, must be directed to this point. The recumbent posture is a *sine quâ non* of successful treatment. In order to make this possible, the use of some sedative is indicated; and I have found codeia the most efficient and harmless drug to use in this condition. Suppositories of gum fœtida will also be of service in quieting the nervousness which always results from prolonged pain. By these means it will be possible to keep the patient in a recumbent position, lying flat on the stomach or back. A board, covered only with a blanket, is the most practical basis for this treatment. After this the lateral swing devised by Barwell[1] may be used in various modifications to suit each case.

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[1] This Cyclopædia, vol. iii. p. 1024.

[2] Maladies du Système nerveux, par A. Vulpian, Paris, 1879, p. 29; Pott's Disease, Shaffer, New York, 1879, p. 5.

[3] Bodily Deformities, Reeves, London, 1885, p. 127.

[4] Ibid.

[5] Orthopædic Surgery, Bradford and Lovett, New York, 1890.

The medical treatment is no less important, and must include various preparations of lime and iron phosphates, hypophosphites, chalybeate baths, coarse food, as Graham bread, cracked wheat, barley, bone soup, and all nutriment rich in phosphates, which may improve nutrition and assimilation. If improvement does not take place under this treatment, some more decided means to prevent pressure and stop the commencing inflammation must be adopted ; and for advice on this subject the reader is referred to the article on Curvatures of the Spine and Pott's Disease, in the third volume of this work.

PERIPHERAL NEURITIS.

The causes which give rise to this affection are not largely operative in the child, and hence it will require but a brief notice here. Neuritis may be acute or chronic, primary or secondary, localized or general. It may arise from a direct injury, as a stab, or indirectly, as after a fracture accompanied by redundant callus compressing an adjacent nerve. The entrance of septic organisms into a wounded nerve gives rise to a form of neuritis known as ascending (*neuritis migrans*) ; tetanus is an instance in point. Neuritis may arise by direct extension from contiguous structures, as diseased vertebræ. Strümpell (*op. cit.*) suggests this as the mode of origin of the atrophy and paralysis of muscles after affections of joints. The majority of the cases of the so-called "rheumatic" paralysis,—as facial,—and certain forms of neuralgiæ, especially sciatica, intercostal neuralgia with zoster, etc., are instances of neuritis. It has recently been discovered that it is a far more frequent condition than was hitherto supposed, and many affections have been found to depend upon it which were formerly ascribed to disease of the cord. Such are the "toxic" paralyses, resulting from lead, arsenic, mercury, alcohol, cases occurring in connection with smallpox, typhoid fever, and some other acute diseases, and diphtheritic paralysis. The latter involves a special parenchymatous form. When many nerves are involved, it is known as "multiple" neuritis. Alcohol is the most frequent cause of multiple neuritis, which is rare though not unknown in the child.[2]

Acute neuritis is characterized by anatomical changes in the nerve similar to those accompanying inflammation elsewhere. They affect chiefly the sheath, which becomes in consequence thickened, compressing the nerve-fibres within and producing pain and interfering with their functions. Restoration more or less complete is to be expected in this form, but it often eventuates in the chronic form. Chronic neuritis is accompanied by fatty and atrophic changes, which permanently impair the usefulness of the nerve. The acute form commences with severe pain in the course of the affected nerve, with marked tenderness on pressure. Soon numbness appears in the parts to which it is distributed, and the limb becomes weak and may be completely paralyzed. In severe cases the muscles undergo atrophy and exhibit reaction of degeneration. There are often œdema and herpetic eruption of the part.

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[1] Lancet, July 9, 1887, p. 59.

[2] Gowers, op. cit.

Primary multiple neuritis, which is becoming more and more rare as cases are being investigated and their dependence upon obvious causes—and especially alcohol—is being traced out, sets in suddenly, with fever, headache, anorexia, and sometimes delirium, followed by severe pain in the limbs and loins, often swelling of the joints resembling rheumatism, and later paresis or paralysis, with diminished reflexes, and reaction of degeneration. The career of the disease may now be arrested, or it may continue in a chronic form, or may prove fatal from involvement of the respiratory muscles. Strümpell (*op. cit.*) believes that multiple neuritis is a definite form of infectious disease.

The diagnosis of neuritis is to be based on the limitation of pain and tenderness in the course of a nerve. It is liable to be mistaken for rheumatism, periostitis, and neuralgia. The localization of tender spots, the intermissions in the pain, and the intact sensibility point rather to neuralgia, whilst the reverse is true in case of œdema, and trophic changes in the skin, nails, and hair. Multiple neuritis must be distinguished from poliomyelitis.

There is nothing special in the treatment of the various forms of neuritis in the child calling for measures different from those used in the adult.

FACIAL HEMIATROPHY.

Synonymes.—Progressive facial hemiatrophy, Neurotic facial atrophy, Prosopodysmorphia; French, Aplasie lamineuse progressive.

This very rare disease, consisting in a wasting of all the tissues, including bone, on one side of the face, frequently begins in childhood, sometimes as early as two or three years, and usually without assignable cause. Of twenty-five cases collected by Mills,[1] seven occurred under ten and eighteen under twenty years. It is a significant fact that the atrophy is limited to the distribution of the fifth nerve, and, taking this in connection with occasional pain, spasm, etc., at the onset, the probability is that it is connected with some organic disease of the fifth nerve interfering with the trophic functions thereof. The disease commences gradually, sometimes at one spot on the cheek, with wasting of skin, subcutaneous tissue, muscle, and bone. Cases are reported in which it has extended to the muscles of the back and arm and involved the opposite half of the face, or the entire corresponding side of the body.[2] The hair drops out, and sometimes the teeth. The sensation, the organs of special sense, the secretions of saliva and tears, are unaffected. The same is true of electrical reaction. After some years the disease ceases to progress and becomes permanent.

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[1] Pepper's System of Medicine, vol. v. p. 694.

[2] P. Davidoff, Thesis, Erlangen, 1875; H. Emminghaus, "Ueber halbseitige Gesichtsatrophie," Deutsches Archiv f. Klin. Med., 1873, xi. 96.

Treatment is futile. The affection can be confounded only with the congenital disparity sometimes met, which, however, differs in being but slight and in the absence of the trophic changes seen in this.

PERIPHERAL SPASM.

Only spasmodic affections non-central in their origin will be considered, and this will exclude hysterical spasms, which are not uncommon in childhood.

In the child motor neuroses predominate largely over the sensory; hence neuralgia and disturbances of sensation are rare, whilst all forms of convulsive disorder are exceedingly frequent, especially during the first three years of life. A large proportion of the convulsive disorders of children, whether local or general, are reflex in their origin, being due to some excentric irritant acting upon the peculiarly impressionable nervous system of the child. As pointed out by Gowers (*op. cit.*) and others, this readiness to react to slight stimuli is probably mainly to be ascribed to the imperfect development of the nervous organism in the infant, in consequence of which the lower centres are further advanced in organization than the upper, and hence are less completely under the control of the latter.

ECLAMPSIA.—Eclampsia is the term used to designate the non-organic convulsions of children, except epilepsy, which it so much resembles that the distinction is often difficult: from this resemblance eclamptic convulsions are often spoken of as epileptiform.

Etiology.—Any irritation is liable, especially in children of a neurotic constitution, to be followed by convulsions. Among the most frequent seats of such irritation is the gastro-intestinal canal. The cause of the trouble may be an overloaded stomach or intestine, or the presence of irritating material within these. Hard, crude, indigestible substances, as green apples, the skin of dried fruit, nuts, etc., are especially likely to produce an attack. Lumbricoid worms undoubtedly play an important part in the causation, notwithstanding Henoch (*op. cit.*) declares that he has not seen a single case of eclampsia traceable certainly to worms.

Dentition has hitherto borne a large share of the blame for these cases. The pressure of the developing tooth upon the gum offered so ready an explanation of the event and so simple a means of relief in the gum-lancet, that there was nothing more firmly settled in the minds of physicians of a past generation than the causal relationship of the one and the therapeutic value of the other. We cannot ignore altogether this clinical experience or this etiological factor. That it produces such milder spasmodic disturbances as obstinate vomiting, diarrhœa, and cough, is a strong ground for believing that it is capable of greater mischief. There is no doubt, however, that its influence has been greatly exaggerated, and that "teething fits" are not nearly so common as was once supposed. According to Henoch (*op. cit.*), only rarely are convulsions observed in teething children who are not rickety.

Severe paroxysms of coughing, as in pertussis, may lead to convulsions, possibly from the venous congestion of the brain to which they give rise. Among other causes reported are foreign bodies in the nose, ear, or skin, and the presence of calculi in the genito-urinary tract. Demme[1] reports a case in which repeated attacks completely disappeared on the removal of a rectal polypus. Attacks have been occasioned by a fall. Violent excitement, especially fright, is a well-known cause. It is alleged that changes in the milk from imprudences in the diet of the mother or nurse may be causative.

Uræmia sets in with violent convulsions. Many acute diseases, especially pneumonia and the exanthemata,—measles, scarlatina, small-pox,—frequently begin in this way. The high temperature and possibly, to some extent, the infectious material are to blame here, but in local inflammatory disorders reflex irritation may also be concerned. It is well to remember, however, that symptomatic fever may be present in cases purely reflex. Henoch (*op. cit.*) saw two cases of simple tonsillitis accompanied by repeated convulsions on the first day; and intense fever in any slight local affection may produce them. Attacks of intermittent fever are apt to be ushered in in the child by a convulsion, which takes the place of the rigor, itself a convulsive phenomenon.

Symptomatology.—The symptoms are nearly identical with those of epilepsy. They begin with sudden unconsciousness and rolling of the eyeballs upward or to one side. The muscles of the face contract spasmodically, the mouth being drawn to one side. The jaws are firmly closed, and chewing movements or grinding of the teeth ensue. The limbs become stiff and frequently twitch. The fingers are flexed into the palm, and the toes towards the sole. The head is drawn backward, and the muscles of respiration contract, producing noisy, hissing inspiration, and often arresting respiration for some seconds. The muscles of the abdomen become rigid, and the urine and fæces are frequently expelled. The interference with respiration occasions some cyanosis. The movements of the tongue and cheeks cause the expulsion of frothy saliva from the mouth, often blood-stained in older children, from biting the tongue. The symptoms, which are very alarming to the friends, usually last only a few minutes, gradually abating, and leaving the child in a stupor. Usually there is a repetition of these attacks several times, unconsciousness continuing from one to the other. They may continue for hours or even days, and may by their force and frequency lead to exhaustion and death. Henoch (*op. cit.*) relates a case of temporary aphasia, drowsiness, etc., occurring without and in place of the convulsion; and in another, where aphasia was the only symptom, complete recovery ensued after vomiting cherries. He also calls attention to paresis or paralysis of a limb occasionally following convulsions and lasting several days, and cautions us to be on the lookout in such cases for possible brain-disease.

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[1] Jahresber. des Berner Kinderspitals, 1879.

Diagnosis.—Whilst frequently indistinguishable from epileptic convulsions, infantile eclampsia may present only certain of the features of the epileptic fit, and may be very slight or partial. It presents a much greater variety in intensity than the epileptic paroxysm; it is also less sudden in its development, less regular in occurrence, and tends to increase in severity and frequency until the cause is removed. We cannot exclude cerebral disease at once, especially if we are unacquainted with the previous history of the child. Prolonged coma succeeding an attack may justly excite a suspicion of meningitis. Henoch (*op. cit.*) reports a case where coma with fever lasted almost three days and was succeeded by temporary aphasia. This attack was due to an overloaded stomach. As a rule, unilateral convulsions indicate a cerebral origin, but exceptionally they are observed in eclampsia, and, on the other hand, organic disease of the brain is sometimes accompanied by bilateral convulsions.[1] It is important to remember that gross disease of the brain in children sometimes continues for months without other symptoms than repeated convulsions until a sudden hemiplegia or coma sets in: this should inculcate the necessity of caution and delay in deciding upon the nature of the case. The importance of directing attention to the condition of the osseous system is evident from what has preceded. The epiphyses, the fontanels, and the forearm should be examined, and in most cases of recurring eclampsia between six months and two and a half years rickets will be found to be more or less well marked. Almost always in these cases laryngeal spasm will be present, either ushering in the convulsion or occurring in alternation with it. The presence of fever should lead to the examination for local inflammations.

Prognosis.—The prognosis of eclampsia as to life is more serious than in epilepsy. It is to be based upon the age and state of health of the child, and the severity, frequency, and duration of the fits. A young and feeble child is quickly exhausted by frequent attacks. If the fits have continued for several months, there is considerable danger of their eventuating in true epilepsy. Long or permanent mental deterioration may result under the same circumstances.

Treatment.—The relief of the convulsion is the first indication to be followed. If the physician be present during the fit, he may resort to chloroform inhalations, so strongly recommended by Henoch,[2] being careful, however, to watch pulse and respiration. Its use becomes improper, of course, if signs of collapse appear. A warm bath is a domestic remedy which is sometimes effective.

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[1] Henoch (*op. cit.*) reports a case of intussusception at eight years, in which unilateral convulsions took place on the day of death.

[2] *Op. cit.* We could hardly go so far with this excellent author as to intrust its administration to the friends. He declares his belief, however, that it is perfectly safe to do so, and that he has never seen any ill consequences therefrom.

As soon as the convulsion is relieved, remedies especially addressed to the cause of the attack, whatever it be, are required. Emetics and purgatives will be found most generally appropriate. Of the former, a teaspoonful of vinum ipecacuanhæ may be given once or twice; of the latter, calomel is most readily administered, being placed dry within the mouth and aided by an enema. In convulsions prolonged for several hours, Henoch (*op. cit.*) recommends (in robust children) the application of cold compresses or an ice-bag to the head, and in exceptional cases even leeches to relieve the venous engorgement. If worms have been passed or be strongly suspected, an anthelmintic may be administered.

In the intervals, sedatives, to relieve the reflex irritability of the nervous centres, especially bromide of potassium and chloral, are called for. The latter may be effectively administered by the rectum. Tonics are also required in most cases, and, remembering the frequent association of rickets, remedies especially addressed to that diathesis are often indicated, as iron, cod-liver oil, phosphorus, the hypophosphites. Should there be evidence that the pressure of an advancing tooth is the cause of the trouble, the gum may be lanced.

TETANY.

Synonymes.—Tetanilla, Arthrogryposis,[1] Intermittent tetanus; French, Contracture essentielle ou idiopathique des extrémités.

This name is given to a rare and peculiar neurosis characterized by tonic spasms of the muscles and affecting especially the extremities. The spasms may occur in paroxysms or may be indefinitely persistent. The affection is most frequent between birth and five years of age: in a table of one hundred and forty-two cases collected by Gowers (*op. cit.*) twenty-nine per cent. occurred during the first decade and fifty-five per cent. during the first two decades of life. It is much more frequent in males, especially in the first years of life. In rare instances several cases have been noted in the same family. The disease is traceable in the majority of cases to persistent diarrhœa, to exposure to cold, especially during acute disease (typhoid fever, rheumatism, pneumonia, etc.), or to debility. It may be due to teething, genito-urinary irritation, etc. In young children the evidences of rickets are rarely absent, and it is often associated then with laryngismus stridulus and eclampsia. Instances are recorded in which it was dependent upon the presence of a tape-worm. It has been known to prevail in an endemic form, as in an outbreak in a girls' school in France in 1876, in which thirty of the pupils were attacked.[2]

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[1] Some authors—as Strümpell, Henoch, Koppe—make a distinction between the paroxysmal and the persistent forms of contracture, describing the latter under the name of idiopathic contractures, or arthrogryposis. There appears no sufficient ground, however, for such a distinction.

[2] Simon, Mattraits, Thèse de Paris, 1877. Strümpell expresses a doubt as to the genuineness of the disease in such cases (*op. cit.*).

The disease sets in with "tingling" or "burning" sensations, followed in a few hours by sudden tonic flexure of first the fingers, then the toes. The fingers are in the attitude of holding a pen, and the feet in that of talipes equinus.[1] The spasm may be limited to these parts, or may extend to the muscles of the neck and the trunk, especially the abdomen. The respiratory muscles may also be involved, producing dyspnœa. The jaws are often spasmodically closed, and the angles of the mouth drawn. There may be strabismus. Consciousness is not lost. The affected muscles are the seat of cramp-like pains, and attempts to extend them produce pain. The spasms are usually paroxysmal, lasting from a few minutes to hours, and then gradually abating, to recur after hours or days. Relief in the intervals is sometimes not complete. In other cases spasms are continuous for days or longer at a time. There is increased irritability in the affected nerves and muscles, and percussion or pressure upon them during the intervals will cause spasmodic contraction. Electrical excitability—both faradic and galvanic—is still more noticeable. The spasm is almost always symmetrical. In young children it is usually continuous and comparatively mild in degree. It sometimes persists during sleep. Moderate fever is sometimes an accompaniment. The duration is variable,—from a few days to several weeks. There is a tendency to recurrence.

Post-mortem examination throws no light upon the character of the disease, as there are no constant and characteristic lesions. Gowers (*op. cit.*) is disposed to regard it as seated in the cord and medulla rather than in the peripheral nerves, basing his opinion on the discovery of slight changes in the motor cells of the cord in severe cases, on slight spinal weakness occasionally following the disease, on its bilateral symmetry, on the peculiar and uniform character of the spasms, and on the muscular atrophy that has been observed to succeed it.

The diagnosis is to be based upon the peculiar form of spasm, its symmetry, its commencement in the extremities, and its limitation to certain groups of muscles.[2] The increased irritability of the nerves will aid in making the diagnosis. The dependence on diarrhœa and "taking cold" should be remembered. In tetanus locked-jaw is the earliest symptom, whereas in this malady it is the latest. Disease of the brain is excluded by the absence of brain-symptoms and paralysis and by the bilateral character of the spasm.

The prognosis is favorable. The affection rarely involves any danger to life, and then through interference with the respiratory function or through associated conditions.

The treatment requires the removal of the cause, if one be discovered. The child is to be carefully protected from exposure, and the bowels must be regulated if at fault. Warm baths and diaphoretics are recommended.

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[1] Strümpell, op. cit.

[2] Henoch (*op. cit.*) regards the affection ("idiopathic contractures") as an abortive form of eclampsia.

Tonics are required, especially cod-liver oil and iron, on account of the associated rickets. Sedatives—the bromides, Calabar bean, chloroform—may be tried, but little reliance is to be placed on them. Suppositories of gummi asafœtidæ will be found useful.

NODDING SPASM (SPASMUS NUTANS; SALAAM CONVULSIONS).—A number of cases have been described by Henoch, Ebert, Demme, and others, in which infants, and sometimes older children, were affected with clonic spasm of the sterno-mastoid and adjacent muscles, giving rise to nodding and rotatory movements of the head. The rotation was almost always towards the same side. Very frequently nystagmus accompanied, but rarely strabismus. The movements are, as a rule, continuous; much less often they occur in paroxysms. The affection is a reflex neurosis, usually due to teething. It is to be distinguished from the swaying movements connected with masturbation, and also from a fatal form sometimes noted in connection with epileptic attacks. The treatment consists mainly in the removal of the source of irritation.

SALTATORY SPASM (STATIC REFLEX SPASM).—A few cases of saltatory spasm have been reported in children of ten years and upward. It consists in clonic spasms of the muscles of the legs, causing the patient to leap or jump or run whenever he attempts to stand. It occurs in neurotic subjects, and comes on suddenly, usually after some depressing influence. It generally continues some months and then ceases gradually. It is associated with increased reflex irritability of the cord. The tendon-reflexes are correspondingly increased. Strümpell (*op. cit.*) regards many cases as hysterical. Nervine tonics offer the best prospect of benefit from treatment, especially arsenic in small doses, and asafetida in combination with cannabis indica, preferably given by suppository.

HABIT SPASM (HABIT CHOREA; MIMIC OR HISTRIONIC SPASM; CLONIC FACIAL SPASM; CONVULSIVE TIC).—Under the name habit spasm, etc., certain involuntary and usually unilateral spasmodic movements, chiefly of the head, face, and shoulders, have been described, which are very commonly met in children from four to fourteen years of age. They consist of winking, twitching of the mouth, jerking the head, and similar movements. The affection occurs especially in nervous and excitable children, and is usually preceded by depressing influences, such as bad health, overstudy, fright, mental excitement, etc. It may be referable to local irritation in the part affected, as when in the lids from conjunctivitis, and very severe cases are sometimes met with as the result of masturbation. It is often associated with hysteria, and often arises by irritation,—not so much directly, perhaps, as by suggestion. In this way Gowers (*op. cit.*) would explain the cases of apparent heredity. The movements recur every few minutes, but are not always identical. Their most common form is spasm of the orbicularis palpebrarum,—blepharospasm or nictitating spasm,—which may extend to the eyebrows and the facial muscles, and even to the occipito-frontalis. Another frequent form is contraction of the zygomatic muscles, drawing the mouth to one side. In other cases the head is drawn backward or forward or to one side, or there is a jerking of the arm or shoulder or a slight movement of the hand. Sometimes half the body is affected. The leg is rarely affected. The muscles of respiration may be involved, giving rise to a sudden inspiration accompanied by a peculiar sound. A cough is not uncommon: Strümpell (*op. cit.*) reports the case of a boy, aged ten, who had a peculiar reflex, hollow, barking cough, occurring spontaneously or after pinching any part of his skin; it lasted some weeks, and then disappeared suddenly. In other cases there is yawning, sneezing, weeping, or laughing. Henoch (*op. cit.*) reports three cases of the latter in very young infants, originating in intestinal irritation. Blachez records a case in which there was a sudden loud cry. There is no anæsthesia or pain in these cases. The affection usually ceases after an indefinite period (months or years), but occasionally persists to adult life.

The spasms are increased by observation: hence the friends should be cautioned to take no notice of them. They are seldom under control, and fear of punishment is likely to aggravate them; but, according to Gowers (*op. cit.*), the promise of a reward at the close of each day provided the spasms have not occurred will sometimes cause their gradual disappearance, the strong desire to avoid them effecting that which the will could not alone achieve. Attention must be directed to the general health, and change of scene and surroundings is advisable.

Arsenic is the best drug, and may be given in combination with quinine and strychnine. Weir Mitchell has cured obstinate cases by the hypodermatic injection of arsenic. The bromides may be required to quiet cough and spasm or to relieve mental excitement.

It has been found that there are certain points along the course of the trifacial (and of other nerves when they are involved), and also over the cervical portion of the spine, pressure upon which inhibits the spasm. They are known as "pressure-points." The application of the galvanic current has the same effect, and may even prove curative. Hence these points should always be carefully sought for, with a view to obtaining this temporary or permanent relief.

TORTICOLLIS (WRY-NECK).—Torticollis may depend upon several different causes, as caries of the vertebræ, rheumatic inflammation of the muscles of the neck, and simple spasm of the sterno-cleido-mastoid and adjacent muscles. For want of space it will be impossible to describe these several forms. In any case, however, it is highly necessary to investigate the cause of the condition and at once institute suitable measures for its relief or cure. In many cases this will be found very difficult, and in commencing caries of the cervical vertebræ it can be treated only by mechanical means to prevent inflammation and resulting ankylosis of the vertebræ. Pain and stiffness often accompany the onset, which is very gradual, extending over months. Exceptionally the onset is brief, the affection becoming fully developed within a few days. The sterno-mastoid is almost always involved, but rarely alone. The trapezius, splenius, scaleni, and platysma myoides are involved with a frequency corresponding to the order of their names as here given. Various movements may be impressed upon the head, according to the muscles involved. The most frequent one is that in which the head is turned upward and towards the opposite side, the mastoid process being drawn downward and forward by the sterno-mastoid towards the sternum. The head may be drawn backward by the muscles at the nape of the neck. Sometimes the spasmodic movements extend to neighboring muscles, as of the face and arm. The constant contraction of muscles produces more or less fatigue and soreness in them.

The course of the disease varies: after a certain increase, it may become permanently stationary, or it may slowly decline, and may in exceptional cases even cease altogether. The pathology of the affection, like that of the other neuroses, is unknown. The diagnosis involves no difficulty. The prognosis as to recovery is bad, especially in severe cases.

In the treatment nervous sedatives often exert a strikingly palliative effect, especially asafetida, bromide of potassium, cannabis indica, chloral, conium. It has been suggested in the spasmodic form to treat the contracted muscles by injections of atropine, with a view to relaxing the tense muscular fibres: this treatment however, has not yielded any permanent results. Electricity, in both forms, is of use in the treatment of functional or spasmodic wry-neck, but if the condition be due to caries of the cervical vertebræ this agent will be worse than useless. Operations upon the nerves and muscles only aggravate the case in the spasmodic form, but in the fixed congenital form the division of the contracted tendon produces a rapid and permanent cure. Henoch[1] reports a case of the intermittent form occurring daily with great regularity, and rapidly cured by quinine.

TREMOR.—Henoch (*op. cit.*) reports a case of general tremor in a child of fifteen months, which ended favorably. This symptom, so common in advanced life, is extremely rare in childhood, and, when met with, is usually connected with serious infectious or brain disease. In the case in question the trembling was continuous, and involved the hands, head, and feet. The child cried a great deal, as if in pain, and the cry was quavering in character. The neurosis, for such it appears to have been, disappeared under chloral hydrate, after lasting twenty-nine days. Demme[2] reports a similar case, but of longer duration, lasting from the fourth to the eleventh month.

PERIPHERAL PARALYSIS.

This may be met with as a rare sequence of the acute infectious diseases, especially typhoid fever and the exanthemata, and, whilst it is then often due to central lesions, it may also be referable to peripheral neuritis, single or multiple. The most frequent and important of these infectious paralyses is the diphtheritic, which will require special consideration. Paralysis also accompanies the "toxic neuritis" produced by lead, arsenic, mercury, copper, etc., which has been previously mentioned. Next to the diphtheritic form, the most common of these affections in the child, as in the adult, is facial paralysis.

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[1] Op. cit. Similar cases are reported by Folliet and Simon, Rev. Mens., February, 1883; Lancet, 1879, i. 26.

[2] Neunzehnten Jahresbericht des Jenner'schen Kinderspitals, Bern, 1882.

FACIAL PARALYSIS (MIMETIC PARALYSIS; BELL'S PARALYSIS; PARALYSIS OF THE MOTOR PORTION OF THE SEVENTH CRANIAL NERVE).—This affection frequently occurs at birth or shortly after, usually as the result of injury inflicted upon the nerve in the neck by the forceps. It is also not infrequent after perfectly normal labor, if prolonged or difficult, and in normal pelves.[1] Under the latter circumstances it has been variously attributed to pressure exerted by the promontory of the sacrum or by the ischiatic spines.[2] Depaul saw two cases due to narrowing of the pelvis by tumors.[3] Under these various circumstances the paralysis may be bilateral.[4] It may also be associated with paralysis of the corresponding arm from injury to the brachial plexus in the neck, as will be described farther on. Lesions have been found in autopsies of such cases, either in or around the nerve, especially at its emergence from the stylo-mastoid foramen, and including fatty degeneration of the nerve itself.[5] The appearances of the affected parts are similar to those seen in the adult,—immobility of one side of the face and lids, the eye wide open, etc.,—but as distinguished from the adult these changes are much less marked in the young child, owing probably to the relatively greater quantity of fluid and adipose tissue in the latter and the smaller development of the muscular tissue. It may be scarcely noticeable when at rest, or a slight drooping of the angle of the mouth may be all that is observable, but on crying or laughing it becomes at once obvious. The paralysis may also be limited to a certain part of the face, as the lips or the eyelids. The orbicularis palpebrarum is not always affected. The tongue and uvula are usually intact, and hence sucking is not, as a rule, interfered with. Electro-contractility of muscles is sometimes preserved, sometimes lost.[6] A favorable prognosis may usually be given under these circumstances, as the paralysis will almost certainly disappear in a few days or weeks, as the effect of compression wears off. A few cases, however, are on record in which the injury was so great as to lead to degenerative changes in the nerve and permanent loss of function. The treatment consists in protecting the eye from air and light, in seeing that the infant receives a due amount of nourishment, and in the application of mild currents of electricity.

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[1] Paralysies obstétricales des Nouveau-nés, Paris Thesis of P. A. H. Nadaud, No. 282, 1872. Also Dublin Quarterly Journal of Medical Science, 1859, vol. xxvii. p. 155, cases of Dr. George H. Kidd. Also, Henoch, op. cit.

[2] Evory Kennedy, Dublin Journal of Medical Science, 1836–37, vol. x. p. 430.

[3] Nadaud, op. cit.

[4] Gowers, op. cit., p. 647.

[5] Parrot et Troisier, Note sur l'Anatomie pathologique de la Paralysie faciale, etc., Paris, 1876, vol. iii. p. 449 *et seq.*

[6] Guéniot, Paralysie consécutive à la Compression des Nerfs, Paris Thesis, No. 134, 1872.

In later childhood the causes and symptoms resemble those of adult life.[1] Besides cold draughts blowing upon the side of the face and producing the inflammatory or so-called "rheumatic" form, from which recovery almost invariably takes place in a few weeks, a common cause is the pressure of an enlarged gland or abscess upon the nerve-trunk at its emergence from the stylo-mastoid foramen. The commonest cause, however, in children, as we might infer, is caries of the petrous portion of the temporal bone, by which the nerve is injured or destroyed as it traverses the Fallopian canal.[2] Otorrhœa is always present in this form, and the discharge will often contain fragments of bone, and sometimes the ossicles from the tympanum will escape. Sometimes there is a tender swelling over the temporal bone behind the ear, and fistulæ may form communicating with the carious cavity. Pieces of dead bone may occasionally be extracted from the meatus or from the mastoid process. Prolonged paralysis leads to muscular atrophy and reaction of degeneration; the muscular tissue may entirely disappear. Autopsy in such cases reveals extensive caries of the petrous bone and frequently localized meningitis; loose pieces of dead bone are sometimes found. The prognosis as to recovery is bad in these cases; they are often connected with tubercular disease, which may extend to the brain, or cause general tuberculosis, or prove fatal by producing thrombosis of the cerebral sinuses. Henoch (*op. cit.*) has known the affection to commence as early as the third month. A frequent cause of it is a neglected otitis media, especially with scarlet fever; and this fact should inculcate the necessity of paying particular attention to the otorrhœa which so frequently accompanies or follows that disease.[3]

In the treatment of facial paralysis we must be guided by the cause when that can be ascertained. If due to the existence of ear-trouble, that must be removed; if to cold or exposure, measures with special reference to the rheumatic origin should be instituted. But to whatever cause it may be due, unless the trouble be central, electricity will be found of great service. The use of both currents is to be recommended, the galvanic to revivify the nerves, and the faradic to improve the condition of the paralyzed muscles. Both currents are best applied by placing one electrode over the exit of the nerve and the other indifferently over the various muscles. A very mild current can be applied in this way to the orbicularis palpebrarum. A most important point in the treatment of facial paralysis is to overcome the contraction and overaction of healthy muscles, because their distortion prolongs the attack and counteracts the good effects of other treatment. This is best done by the application of the rubber band and hook described by me,[1] which creates an artificial muscle taking the place of the muscles useless from disease. By this simple apparatus the distortion which so impedes recovery is overcome, the nutrition and return to functional activity of the muscular fibres are promoted and hastened, and the duration of the disease is proportionately shortened. In addition to this, massage or shampooing of the affected side is of great value in preventing atrophy and producing a more natural condition of the circulation. The application of a blister behind the ear is of benefit in many cases. For the treatment of the caries and otorrhœa I refer the reader to the articles in this work dealing with these subjects.

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[1] Gowers (*op. cit.*) reports an interesting case in which complete and permanent paralysis was caused by a school-master's striking a boy with the corner of a book just below the ear.

[2] Bezold, Labyrinth-Necrose, 1886, calculates that it accompanies necrosis of bone in one per cent., an estimate which Gowers (*op. cit.*) thinks too low. Of eighty cases of facial paralysis (not including cases due to ear-disease and syphilis), it occurred but twice under ten years (Gowers).

[3] Gowers (*op. cit.*) calls attention to the furrow that is seen in the paralyzed cheek in children as the result of secondary contracture as voluntary power returns. It is quite as disfiguring as the previous condition, and in severe cases may be permanent.

Allied to the congenital paralysis of the face above referred to is a similar affection of the arm, due to pressure upon the brachial plexus at birth. It is most frequently referable to the use of the forceps compressing the plexus at the side of the neck just above the clavicle, but it may also occur in labors where no forceps have been used, especially if protracted or difficult. Instrumental face presentations are particularly amenable to this accident, which may affect both arms simultaneously,[2] or may involve only one or more groups of muscles, as the deltoid and extensors, or may extend to other parts, as the face. Depaul[3] attributed it to too deep an introduction of the forceps into the pelvis; Jacquemeyer,[4] to prolonged pressure of the humerus against the axillary plexus. Forcible dragging upon the arm or shoulder or dislocation of the shoulder during delivery is sometimes accountable for it. The paralysis usually passes off in a few days, but if the damage be severe and irreparable it will be permanent. Death may ensue quickly from other injuries received. Anæsthesia sometimes accompanies the motor paralysis. In severe cases, with irreparable lesions, atrophy of the limb soon sets in, with loss of electrical reaction, and the limb becomes in time shrivelled and shortened. The treatment of most value is electricity, and it should be applied at the earliest period, before muscular and nervous degeneration have taken place. The galvanic current is to be preferred. In late childhood violent stretching of the brachial plexus, as by a sudden wrench of the arm, may produce a paralysis continuing for weeks or months.[5] This last condition is closely allied to the paralysis often observed later in life, in which the extensor group of muscles is paralyzed by pressure on the musculo-spiral nerve. A paralysis of the extensors of the arm is also one of the diagnostic marks of toxic paralysis from lead; and if any of these causes should produce this form of paralysis in children, it will demand the same treatment as in adult life. Besides the ordinary treatment of electricity, frictions with lotions containing tincture of nux vomica or strychnine, and careful massage, an artificial extensor muscle can be applied, as advised by me in a former article on this subject.[1] The use of this apparatus has in my experience materially shortened the duration of the paralysis, which is very difficult to overcome on account of the persistent distortion of the hand from the overaction of the flexor muscles.

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[1] Medical News, vol. xlvii. p. 597.

[2] Midwifery, W. Smellie, London, 1758, vol. ii. p. 506.

[3] Guéniot, Bulletin de la Société de Chirurgie, Paris, 1867, vol. viii. p. 34.

[4] Manuel des Accouchements, 1846, vol. ii. p. 785.

[5] Henoch, op. cit. A violent wrench in putting on a child's jacket produced paralysis of the deltoid, which disappeared only after several weeks' application of frictions and electricity

Möbius[2] has described a paralysis of the ocular muscles which, from its recurrence from time to time, he calls "periodical oculo-motor paralysis." It is usually unilateral, often dates from infancy or childhood,[3] and recurs at variable intervals, usually months or years, until middle life. As the child grows up, the intervals diminish in length, whilst the attacks become more and more severe. They commence with severe pain in the eye, often accompanied by headache and vomiting, lasting two or three days, and followed by paralysis of the third and sometimes of the sixth nerve, which usually passes off gradually after a few days or weeks. The nature of this affection is entirely unknown.

Reflex Paralysis.—Peripheral paralysis, like peripheral spasm, is sometimes set up by local irritation, of which a well-marked example is the paraplegia due to phimosis, to which attention was first prominently directed by Sayre in 1870.[4] Sayre's cases occurred in boys of from three to fifteen years, and the symptoms were paresis of the lower limbs of years' duration, constant and painful erections, amounting almost to priapism in some cases, great mental irritability, inability to articulate properly, insomnia, etc. Relief, usually complete, was afforded by circumcision, the adherent prepuce being torn off from the glans, and the hardened secretion being removed from behind the corona.[5] Similar relief was afforded in the case of a girl by clipping the clitoris. Dr. Sayre also saw similar symptoms produced by the irritation of other nerves. Further investigation showed that the irritation may exist in any part of the body. Thorowgood,[1] for instance, reports the case of a girl aged ten, suffering from paraplegia, who, on the second day after the expulsion of many ascarides by an aloetic purgative, was able to run and walk, and a few days later was perfectly well. Henoch (*op. cit.*) also reports the case of a boy of seven who had practised masturbation for two years, and suffered in consequence with nocturnal incontinence of urine and inability to walk or even stand or sit without support, and when supported his gait was distinctly ataxic like that of locomotor ataxia. The ataxia was markedly increased on closing the eyes. Plantar reflex was weak and slow. The fæces sometimes passed involuntarily. Under treatment by lukewarm baths for ten minutes daily, with cold shower over head and back, and strict watching, he entirely recovered in five weeks. Other forms of paralysis have also been noted from phimosis, as hemiplegia,[2] strabismus,[3] etc. Several explanations have been proposed for this reflex paralysis. Brown-Séquard suggested a reflex spasm of the arteries of the cord, producing anæmia, defective nutrition, and consequent loss of muscular power. Leyden and others tried to explain them by supposing a neuritis arising at the point of irritation and extending thence to the cord. Inhibition of the cord-centres from protracted reflex irritation (or, in other words, exhaustion from repeated and excessive stimulation) is, however, the most rational theory of the pathology of these cases.

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[1] New York Medical Journal, May, 1874.

[2] Neurolog. Centralblatt, 1884.

[3] Snell reports a case commencing at eleven months, Transactions of the Ophthalmological Society of England, vol. v.

[4] Articles by Sayre on the subject were published in the following: Transactions of the American Medical Association, 1870; Medical and Surgical Reporter, 1876, vol. xxxv.; Virginia Medical Monthly, 1877, vol. iv.; Philadelphia Medical Times, 1882–83, vol. xiii.; Transactions of the Ninth International Medical Congress, vol. iii.

[5] The following case, which I take from my note-book, is typical of this condition and shows the results of operation. H. B., three years old, a well-developed boy, walked badly, tripping and falling constantly. Had good muscular development, except in muscles of thigh; gluteal muscles flat and flabby. Frequent priapism, genitalia very sensitive, and prepuce long and narrow. Operation of circumcision was performed April 9, 1885. Large quantity of smegma turned out. No after-treatment. The child improved at once, and gradually recovered muscular development in the gluteal region. In three months after operation the thighs were as well developed as the rest of the body.

DIPHTHERITIC PARALYSIS.

One of the most characteristic of the phenomena of diphtheria is the paralysis which frequently follows its attacks. Although the contrary has been maintained, it may be assumed that when a paralysis of certain muscles succeeds a sore throat, the latter was a true diphtheria, since ordinary cases of sore throat never produce this sequela. Gowers (*op. cit.*) estimates that on an average one-fourth of those who do not die from the primary disease suffer from subsequent paralysis. Out of sixty-eight cases seen by him, fifteen were under ten years of age. According to the statistics of Bailly,[4] post-diphtheritic paralysis occurs in about one of eleven cases. Of ninety cases of paralysis due to diphtheria collected by Maingault,[5] twenty-nine occurred in children. According to the statistics of Landouzy,[6] the tendency to it increases the older the patient, infants being comparatively insusceptible to it. It may follow diphtheria located anywhere,[1] and is as frequent after mild as after severe cases.[2] It sets in most often during convalescence from the throat or other primary affection, but sometimes comes on during the active course of the disease. According to Squire,[3] there are two kinds of paralysis, one coming on during, or directly after, the attack, and of a gravity proportioned to the general disease, the other coming on later, not before the second or third week, and not corresponding to the severity of the local lesion or general disease.

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[1] Lancet, July 28, 1883.

[2] Otis, American Journal of Obstetrics, 1874–75, vol. vii. p. 478.

[3] Eggleston, Journal of the American Medical Association, 1886, vol. vi. p. 511. In this case the strabismus was associated with paraplegia.

[4] Ulysse Bailly, Paris Thesis, 1872, No. 19, Paralysies consécutives à quelques Maladies aiguës.

[5] Paralysie diphthéritique, Paris, 1860.

[6] Des Paralysies dans les Maladies aiguës, Paris, 1880, quoted by Gowers, op. cit.

Pathology.—The principal changes are found in the nerves going to the paralyzed parts, and are inflammatory and degenerative, the so-called "parenchymatous neuritis," or "multiple degenerative neuritis."[4] They may in severe cases extend back along the *anterior* roots to the cord. They consist in segmentation and breaking up of the white substance of the nerve-fibres, with a multiplication of the nuclei of the nerve-sheath, an accumulation of the granulation-corpuscles among the remains of the fibres, and sometimes even a disappearance of the axis-cylinders.[5]

Oertel[6] found hemorrhages in the nerve-sheaths of the peripheral nerves. As a rule, there is no inflammation in the interstitial connective tissue. The palatine nerves offer an exception to this, which is probably to be referred to their contiguity to the inflammation of the primary disease and to the direct extension of this process to their sheaths. There are often evidences of fatty and granular degeneration of the motor nerve-cells in the anterior horns of the cord[7] and in the muscles of the heart, palate, and extremities. In consequence of these changes, there is loss of faradic irritability. Gowers (*op. cit.*) regards the degeneration of the nerve-fibres and the muscular changes as due to the prior alteration in the nerve-cells.

Symptomatology.—The palate scarcely ever escapes, and is usually the first part to be affected, the symptoms setting in, as a rule, shortly after the disappearance of the membrane. It may be the only part affected. In consequence of its involvement, when the patient attempts to swallow, fluids regurgitate through the nose, and the voice becomes indistinct and nasal, owing to the nasal cavity not being shut off from the pharynx in phonation. On examination of the pharynx, the palate is found to be stationary on inspiration and phonation. Vision for near objects—as in reading or writing (near-sightedness)—is next impaired, in consequence of loss of power of accommodation (paralysis of ciliary muscle). The recti muscles may also be involved, producing strabismus, most frequently external strabismus. Later, in a large proportion of cases, there are weakness and ataxia of the limbs, sometimes accompanied by slight sensory disturbances. In some cases there is loss of power of the respiratory muscles, the laryngeal muscles, the muscles of the neck, the bladder or rectum, or other parts. Hemiplegia and general paralysis are rarely-met forms. The onset and progress of the affection are gradual, never sudden. Owing to the degenerative changes in severe cases, after two or three weeks there is reaction of degeneration,[1] and the knee-jerk is invariably lost. A remarkable fact with reference to the latter, discovered by Bernhardt,[2] is that this loss occurs in two-thirds of the cases in which there has been no paralysis whatever. It was usually noted in the second month. Dysphagia may appear if the pharyngeal muscles be involved. If the larynx be affected, the voice becomes hoarse or disappears, and food often enters the glottis in consequence of the non-closure of the epiglottis (superior laryngeal nerve). The tongue and face in rare cases are involved. A tendency to heart-failure is indicated by frequency, feebleness, or irregularity of the pulse, and fatal syncope is liable then to occur. There is sometimes incontinence of urine or retention. Albuminuria is a very common accompaniment of these paralyses. As the extent of the paralysis varies, so do its severity and duration. Recovery ensues in from two weeks to several months.

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[1] Yet Henoch (*op. cit.*) has never seen it after any form except laryngeal diphtheria.

[2] According to Henoch (*op. cit.*), it is even more frequent after mild than after severe cases.

[3] Paralysis after Diphtheria, Transactions of the International Medical Congress, Seventh Session, 1881, London, vol. iv.

[4] Déjérine, Lésions du Système nerveux dans la Paralysie diphthéritique, 1878; D'Espine and Gombault, as quoted by Sanné, Diphthérie, Paris, 1877.

[5] Gowers, op. cit.; Déjérine, Jahrb. f. Kinderheilkunde, 1878, xiii.; also Ludwig Buhl. Einiges über Diphtherie, Zeitschr. f. Biologie, Munich, 1867.

[6] Deutsches Archiv f. Klin. Med., viii. 1871.

[7] Oertel, op. cit.

Diagnosis.—This is usually simple, in view of the previous attack of diphtheria and the peculiarities of the paralysis. Difficulty may arise from the former having been overlooked. Usually in such cases a history of some sore throat, or of an offensive discharge from the nose, can be elicited. When the throat- and eye-symptoms have been slight, the paraplegia may simulate locomotor ataxia, which it resembles in certain features, as ataxia and absent patellar reflex ; here the previous history, the relative rapidity of onset, the absence of lightning pains and gastric crises, and the spread of the paralysis from one part to another, are to be relied on for the discrimination. Locomotor ataxia also is extremely rare under twelve years of age. The same features are to be depended on for the discrimination from other spinal affections. In simple paraplegia the knee-jerk is usually excessive. Paralysis of the palate does not occur in hysteria, but the latter may be associated with or succeed diphtheritic paralysis.

Prognosis.—This is, as a rule, good. Death may occur from paralysis of the heart or of the muscles of respiration, or from exhaustion due to dysphagia or to inability to get the little patient to take food. In view of the liability to the first of these, in cases even otherwise doing well, a cautious prognosis is advisable. Cardiac paralysis is almost inevitably fatal.

Treatment.—Every effort should be made to keep up the child's strength by food and stimulants. Solid food is generally better swallowed than liquid, on account of the involvement of the palate. When swallowing is impossible or dangerous from paralysis of the pharynx or superior larynx, rectal nourishment must be resorted to, or food (as milk, yelk of egg, beef tea) introduced by means of an œsophageal tube. According to Gowers (*op. cit.*), it is absolutely essential to the patient's safety that he should be fed under these circumstances every twelve hours at least. If heart-failure be threatened, absolute recumbency must be insisted on. I have found good results from the stimulant effect of carbonate of ammonium, in a mucilaginous solution, which renders the dose easier to swallow. Frictions with stimulating lotions or with oil help to strengthen the patient. As soon as possible the use of electricity should be commenced; both currents may be used, the galvanic being applied from the nape of the neck to the group of muscles involved, and the faradic used only to the local groups of paralyzed muscles. Vigorous massage and shampooing must be resorted to frequently during the day, and sponging with warm whiskey at night will be found of service. Under this treatment I have had very gratifying success in two cases of diphtheritic paraplegia. The Germans recommend strychnine very highly, particularly by hypodermatic administration. Henoch (*op. cit.*) gives from one-sixty-fifth to one-sixteenth of a grain daily. Iron in some form is very important, and general tonics should be also used, to build up the system as rapidly as possible.

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[1] Henoch (*op. cit.*) did not observe any diminution of electrical excitability in his cases.

[2] Virchow's Archiv, 1885, Bd. xcix. p. 293.

PRIMARY MUSCULAR ATROPHIES.

By JAMES STEWART, M.D.

Synonymes.—Idiopathic muscular atrophies; Myopathic atrophies; Progressive muscular dystrophies.

Under the head of primary muscular atrophies are included certain different clinical types, all of which are, however, characterized by a primary progressive wasting of some or nearly all of the voluntary muscles of the body.

In the present state of our knowledge, it is impossible to say whether we have to do with one disease presenting in different cases different anatomical features, or with a distinct series of primary muscular affections. It is contended by many that there is essentially only one form of primary muscular atrophy, and that the various clinical differences are simply accidental; while, on the other hand, it is maintained by some that there is a fundamental pathological difference between at least a certain number of the different types which are described. At the present time the weight of opinion is with the former hypothesis.

The classification of the primary muscular atrophies at present adopted, even taking the view that we have several distinct pathological forms to deal with, is far from satisfactory. In order to present the subject in its fullest clinical aspect, it is necessary to follow it. The dissimilarities and likenesses of the various types can in this way be made clearer. It is to be distinctly understood that this classification is purely tentative.

The Various Types of Myopathic Atrophies.—I. Pseudo-hypertrophic muscular paralysis. II. The juvenile form of muscular atrophy (Erb). III. The facio-scapulo-humeral form (Landouzy-Déjérine type). It is universally admitted that the above types are purely primary muscular affections.

A type of muscular atrophy now commonly described as the "peroneal type" has been variously described by different authorities, a few contending that it is myelopathic, others that it is neuropathic, while still others rank it among the myopathies.

Leyden and Möbius describe what is called the "hereditary type,"—an apparently unnecessary subdivision, as heredity is common to all myopathies.

Transitional forms of the different types are not uncommon. Cases in different members of the same family have been described where two and even three different types have been seen.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

Synonymes.—Muscular pseudo-hypertrophy; Lipomatous muscular atrophy; Lipomatosis luxurians muscularis progressiva (Heller).

Pseudo-hypertrophic muscular paralysis is a disease of childhood. Cases of what we now know to be examples of this disease were described as early as 1830, and a marked example, as it affects families, was described by Meryon in 1852. The first true clinical description was given by Duchenne. So complete was this description that little or nothing has been added to the symptomatology since that period.

Etiology.—We have no precise knowledge as to what are the essential causes of this disease. We know that it begins usually between the ages of two and eight, and that it is more commonly observed in males than in females.

It is probable that in exceptional cases signs of the disease may be noticed even as early as the first year, if looked for. Very rarely does it appear so late as at the age of puberty. It is asserted that the disease runs a much milder course, and that it is later in making its appearance, in females than in males.

In a majority of the cases the influence of heredity is apparent, often to a very remarkable extent. Meryon relates an instance where eight brothers died of the disease. In this family all the daughters escaped. In some families, on the other hand, the disease appears to affect only the females. Again, numerous family cases have been observed where both sexes have suffered. The morbid inheritance is always through the mother. The disease is transmitted by her without her being affected. The malady may be congenital and not hereditary.

Symptoms.—Some form of motor weakness is generally the first symptom which attracts the parents' attention. In numbers of cases the first difficulty noticed was inability of the child to climb stairs without pulling himself up by holding on to the balusters. Simultaneously with the motor weakness, or more frequently some time after its appearance, there is noticed an hypertrophy of certain muscular groups.

The calf-muscles undergo this change more frequently and to a greater extent than any other set of muscles. The measurement of the calves of children under ten years often exceeds that of the calves of well-developed adults.

The spinati are probably next to the calf-muscles those which are most frequently found hypertrophied.

The deltoids are often found in an hypertrophied condition. A few cases have been recorded where all the muscles of the shoulder-joint were hypertrophied.

The biceps, triceps, latissimus dorsi, and sterno-mastoids have been found hypertrophied in different cases. The temporals, the masseters, and the muscles of the tongue have been found to have undergone the same change.

With the exception of the pectorals, all the voluntary muscles of the body were found by Duchenne in one case to be hypertrophied.

The muscular hypertrophy eventually disappears, and is succeeded by an atrophic condition of the muscles. This change occurs earlier in the hypertrophied muscles of the upper extremities than in those of the lower. The muscles of the calves are usually the last to undergo atrophy.

The atrophy, however, is frequently the primary pathological change. This is especially noticeable in the muscles of the trunk and upper extremities. The pectorals are generally the first to show signs of wasting, and, as a rule, it attains a greater degree in this group than in any other.

Next in order in degree of wasting are the latissimus dorsi, the trapezii, the serratus magnus, the extensors of the back, and the muscles of the thigh. All the voluntary muscles may become atrophied. As a rule, however, the small muscles of the hand escape.

The contrast between pseudo-hypertrophic paralysis and progressive muscular atrophy (myelopathic) is usually very marked. In the former the small muscles of the hand escape, while in the latter the disease usually begins in these muscles.

The posture and gait of patients with pronounced pseudo-hypertrophic paralysis are very characteristic. When standing, the abdomen projects forward, and the hollow of the lumbar vertebræ is greatly exaggerated. The buttocks are slightly thrown back, while the shoulders greatly project backward, forming the secondary compensatory curve for the lordosis. A plummet let fall from the spines of the upper dorsal vertebræ will fall considerably behind the sacrum. The patient stands with the feet planted widely apart. If an attempt is made to bring the feet together or to straighten the trunk, the child at once falls forward.

The cause of the lordosis is the weakness of the extensors of the hip. When the patient sits, his lordosis disappears, and in those cases where he is no longer able even to walk or stand, the spine becomes convex, from weakness of the extensors of the back.

The gait in pseudo-hypertrophic paralysis is of a peculiar swaying character. The body is thrown to the side of the active limb, so as to bring the centre of gravity over each foot. The passive leg is thus enabled more easily to be swung around. The cause of this waddling gait is the weakness of the extensors of the hip.

A very characteristic symptom of this disease, first pointed out by Gowers, is the way in which the patient raises himself from the horizontal to the vertical position. Owing to the weakness of the extensors of the knee, the patient has to place his hands on the lower part of the thighs and gradually raise himself upward by this assistance. If the degree of weakness is only slight, he may be able by a single effort to straighten his trunk. If the weakness is considerable, he has gradually to raise his hands placed on the thighs one above the other until they reach the upper part of the thigh, and then by a final effort he assumes the erect position. Where there is advanced weakness of the extensors of the knee, even with this manœuvre he cannot raise himself from the floor. The writer has had a case under observation where the patient was unable to raise himself with his hands, but by placing his chin on the edge of the chair he effected his purpose through the action of the muscles of the neck. Cases of this disease are met with where this characteristic way of rising from the floor is not seen. The extensors of the thigh not having suffered, the patient is able to rise from the recumbent position in the usual way.

When the disease is advanced, certain deformities set in, as the result of muscular contraction. In the upper extremities it is common to see the biceps so contracted as to prevent the extension of the arm. The knee-joints become stiff also from contraction of the flexors.

Contraction of the muscles of the calf is frequent. This contraction is so marked in certain cases that the patient is unable to put his heels to the ground.

Electrical Reactions, etc.—Even in the early stages of the disease there is usually a distinct lowering of the muscular irritability to both faradism and galvanism. In the advanced stages this is still more marked. Greatly hypertrophied muscles respond but feebly to both currents.

Cases have been reported where little change has been noticed in the electrical reactions through the whole course of the disease. The reaction of degeneration is never present.

The mechanical irritability is usually much decreased. Fibrillary contractions do not occur.

Gerhardt[1] describes a case where there was constant tremor in the extremities.

The knee-jerk usually disappears entirely as the disease advances. It is common for patients to complain of coldness, especially in the lower extremities, but usually measurements of temperature are negative. Sensation is always normal. There is seldom any vesical or rectal failure.

Complications.—Exceptionally symptoms of mental weakness are seen. Westphal reports a case complicated with paranoia. In many cases it has been noticed that the patients have been late in learning to speak. Difficulty of speech (pseudo-aphasia) is also an occasional defect.

Epilepsy and irregularities in the size and shape of the skull have been described. Vigouroux and Buss have described a case where there were also present symptoms of Thomsen's disease.

Cases of pseudo-hypertrophy of muscles with optic neuritis and atrophy of the disk have been noticed.

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[1] Quoted by A. Pick in Eulenburg's Real-Encyklopädie, Band xvi. p. 99.

Cardiac hypertrophy without valvular lesion has been said to occur. Diabetes insipidus has been observed in a number of cases.

Pathological Anatomy.—The pathological changes in the muscles in pseudo-hypertrophic paralysis are very marked. We find (1) changes in the muscular fibres, (2) changes in the muscular connective tissue, and (3) changes in the amount of fat-deposition in the muscles.

Before death the pseudo-hypertrophic condition has generally almost entirely disappeared, so that it is rare to find any muscle actually increased in volume. The muscles present a pale yellowish color, and are with difficulty distinguishable from masses of fat. It is only on careful microscopic examination that we can be absolutely certain of the presence of muscular fibres. The muscular fibres present great diversity in their breadth, some fibres being found which are extremely narrow, while others are seen to have about the normal diameter. All degrees of variation between these two extremes are to be made out, even in one muscle. Hypertrophied fibres are never seen post mortem. The smallest fibres are found in those mostly replaced by fat. Single muscular fibres vary frequently in their breadth. The transverse striation may be apparent, but it is frequently lost through granular and hyaline changes.

Increase in the nuclei of the affected muscles has been noticed in some cases. Increase in the connective tissue is invariably found. The amount of fat between the muscular bundles and connective-tissue fibres varies considerably. Usually it is very great, but occasionally it is only slight. In the former case, if we examine a muscle which is in an advanced stage of the disease, we shall find numerous large fat-cells, while in other muscles, only beginning to suffer, the fat-cells will be few in number.

Nervous System.—In all the trustworthy recent examinations of the spinal cord no changes of any significance have been found. The remaining portions of the central and peripheral nervous system have been likewise found normal.

The pathological changes in pseudo-hypertrophic muscular paralysis may, then, be summarized as follows: 1. Changes in the breadth and contour of the muscular fibres. 2. Increase in the intermuscular connective tissue. 3. Fatty infiltration of the muscles.

Pathology.—It follows from the post-mortem changes that pseudo-hypertrophic paralysis is essentially a muscular disease,—a true myopathic atrophy. There are differences in opinion as to how this atrophy of the muscular fibres is brought about. Some contend that the first change is a primary myositis, and that the increased connective-tissue formation and consequent atrophy of fibres constitute a later manifestation.

The well-known congenital nature of the disease points, however, very strongly to its being primarily due to the connective-tissue changes. The atrophy of the muscular fibres is, according to this view, the direct result of the increase of interstitial tissue. The formation of fat, to which the present name of the disease (pseudo-hypertrophy) really owes its origin, is merely an evidence of the lowered state of vitality of the tissues concerned, and forms no essential part of the disease.

There is an error in development, and that error consists in a tendency to excessive growth of the muscular connective tissue.

Why the disease should affect mainly the male members of a family remains unexplained, as do many other phenomena in connection with inheritance.

THE JUVENILE TYPE OF PRIMARY MUSCULAR ATROPHY.

This type of myopathic atrophy is now commonly known as "Erb's juvenile form." There is some difference of opinion as to whether it should be described as a distinct form or not. Gowers[1] considers that this is not necessary. Hitzig[2] and Sachs[3] in two excellent papers advance strong reasons for its separate consideration. Hitzig's reasons are based on pathological grounds, while those of Sachs are from the clinical side.

As this form has not as yet been described in this country, except by the writer, I will here give a brief account of a case which formed the subject of a clinical lecture delivered by me in June, 1884:[4]

The patient at the time the following history was obtained was twenty-one years of age. His occupation was that of a farm-laborer, and his complaints were of weakness in the back and legs of three years' standing. He had never suffered from any serious illness previous to the onset of this weakness. He attributed his trouble to a fall which he received six months anterior to the above complaints. It was, however, afterwards ascertained that for some time previous to this he disliked going up-stairs, because he found it both difficult and tiresome. No history of hereditary atrophy.

In the upper extremities there was a marked contrast between the well-developed muscles of the forearms and the atrophied ones of the upper arms, the circumference of the thickest part of the upper arms being an inch less than that of the corresponding part of the forearms. At the thickest part of the thighs the circumference was an inch less than at the thickest part of the legs. In the upper part of the body the following muscles were greatly atrophied: the pectorals, especially in their costo-sternal parts, the lower half of each trapezius, the rhomboids, the latissimi and the whole group of spinal extensors, and the triceps and the brachialis anticus of each side. The biceps of each arm was atrophied and contracted.

The coraco-brachialis, the supra- and infra-spinati, the deltoids, as well as all the forearm and hand muscles, were found well developed. In the lower limbs the glutæi and ilio-psoas were in a state of more or less marked atrophy. The quadriceps of each thigh was found more extensively atrophied than any other muscular group in the lower extremities. The right peroneals were slightly affected, while the left were normal. There was slight hypertrophy of the calf muscles.

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[1] Diseases of the Nervous System, Amer. ed., p. 395.

[2] Berlin. Klin. Wochenschr., 1888, Nos. 25, 34, 35.

[3] New York Medical Journal, December 8 and 15, 1888.

[4] A full description of this case will be found in the Canada Lancet for September, 1884.

All the atrophied muscles were firm. No fibrillary twitchings. Knee-jerks absent. Response to induced current hardly perceptible in the markedly wasted muscles. No reaction of degeneration.

Unfortunately, the subsequent history of this important case is unknown.

The prominent clinical features of this form of atrophy, which are well illustrated in the above case, are: (1) The time of life at which it begins, which is usually before the fifteenth or twentieth year. In this particular it differs from pseudo-hypertrophy, which is practically a congenital affection. (2) The atrophy is limited to a particular group of muscles,—in the upper extremities, to the shoulder and upper-arm muscles principally; in the lower extremities, to the gluteal and thigh muscles. The muscles of the forearm, of the hand, and of the legs remain normal, or may even be slightly hypertrophied. Hypertrophy is, however, not the rule. Lipomatosis may occur. There is an absence or a diminution of the knee-jerks. This loss is more marked when the quadriceps is the seat of wasting. There are no fibrillary twitchings. There are only quantitative changes in the electrical reactions. The disease may be hereditary.

Clinically, the difference between pseudo-hypertrophy and Erb's juvenile form may be said to be as follows: (1) Pseudo-hypertrophy occurs earlier. (2) The localization of the atrophy is different. Such differences are surely not sufficient grounds for making two distinct clinical entities. If the results obtained by Hitzig (*op. cit.*) prove to be true of all cases, we shall, however, have sufficient reason to consider these forms as distinct muscular atrophies. Hitzig, from examination of the muscles in three undoubted cases of juvenile atrophy, comes to the conclusion that the primary change is an increase in the muscular fibres from parenchymatous inflammation. He further maintains that there is no hyperplasia whatever of the interstitial tissue.

Lipomatosis occurs, he says, after the disappearance of fibres, through their mutual compression, and is never primary.

THE FACIO-SCAPULO-HUMERAL TYPE OF PRIMARY MUSCULAR ATROPHY: LANDOUZY-DÉJÉRINE TYPE.

Clinically, this form may be said to be similar to Erb's juvenile variety, with the addition of atrophy of the muscles of the face.

In the cases of this type the atrophy usually begins in the muscles of the face and at a very early age. Commonly both sides of the face are affected, it being exceptional to find unilateral atrophy. One side may suffer a considerable time before the other. The wasting of the muscles of the face gives rise to a peculiar, and, it is said by Landouzy and Déjérine, a characteristic, expression. To it they apply the term "myopathic face."

The orbicularis oris is constantly affected, and in consequence the lips are separated, and when thickened, as they often are, we see the "tapir mouth" of the authors just quoted.

There is obliteration of the naso-labial furrow, from atrophy of the zygomatici. The orbicularis palpebrarum of both sides, the frontalis, and the buccinators suffer also. The levators of the angles of the mouth usually escape.

The atrophy of the shoulder and upper-arm muscles corresponds in every particular with that found in Erb's juvenile form, and hence does not require any special description. Atrophy of the muscles of the lower extremities is also frequently present. In both the upper and the lower extremities contractures are frequent, occasionally to an extreme degree.

Landouzy and Déjérine[1] describe ac ase of myopathic atrophy of this type where the tongue also was affected. The atrophy appeared first in the arms and shoulders, later in the tongue, and still later in the face.

Fibrillary twitchings are absent. The muscles react to both the faradic and the galvanic current in proportion usually to the amount of atrophy. There is no reaction of degeneration. Landouzy and Déjérine report an autopsy on a case of this variety. The appearances were those of a primary degeneration of the muscles. There was very slight increase in the amount of connective tissue and fat.

This type of atrophy is very rare. The only case up to the present time reported in America is by Osler.[2]

THE PERONEAL TYPE.

The disease generally shows itself at an early age. Charcot, however, describes two cases in females where the first symptoms were noticed at the ages of fourteen and fifteen. A very exceptional case is reported by Osler,[3] where the disease made its appearance at the age of forty-seven. According to Charcot, the following is the course of the atrophy. It shows itself first in the peripheral muscles of the lower extremities, usually in the extensors of the great toe, and afterwards in the common extensor of the toe and peroneal group. The above is the order usually noticed by the parents of the child, but it is not unlikely that the small muscles of the foot are the first to undergo degeneration. The calf muscles are later in being involved. The muscles of the thigh suffer still later, and the first of these to exhibit signs of atrophy is usually the vastus internus. The adductors generally escape. From two to five years after the beginning of the atrophy in the feet and legs, the hands show signs of a similar degeneration. The first muscles to be affected are those of the thenar and hypothenar eminences and the interossei. After the intrinsic muscles of the hand, the extensors of the forearm suffer. The pronators and supinators are affected later. In Charcot and Marie's[1] cases, five in number, the supinator longus escaped, and also the muscles of the upper arm, shoulders, neck, body, and face.

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[1] Revue de Médecine, 1886, p. 1004.

[2] American Journal of the Medical Sciences, September, 1889.

[3] On Heredity in Progressive Muscular Atrophy, as illustrated in the Farr Family of Vermont. Archives of Medicine, 1880.

J. Hoffman[2] reports a case where the extension upward of the atrophy was much greater than in any cases heretofore reported. The pronators and supinators were atrophic, as well as the muscles of the shoulder. A second case by the same author showed traces of atrophy in the muscles of the shoulder and the face. It follows from these observations that, as in ordinary myopathies, the entire voluntary muscular system may become in some cases eventually involved.

Hypertrophy has never been observed.

The atrophy and consequent muscular weakness progress together.

Fibrillary contractions are common, especially in the muscles of the hand, but are never so marked as in the ordinary form of muscular atrophy of spinal origin.

The idio-muscular contraction to percussion is diminished even in muscles which do not present any atrophy or weakness.

A very noticeable feature in nearly all the cases of this type hitherto described is the presence of the reaction of degeneration. Charcot and Marie, Ormerod,[3] Schultze,[4] Müller,[5] Tooth,[6] and Sachs,[7] all report cases where there was the reaction of degeneration. Shooting pains in the region of the affected muscles have been observed in a few cases. Cramps of the muscles of the thighs are described by Charcot and Marie. Anæsthesia and hyperæsthesia are also exceptional symptoms.

A remarkable series of cases affecting many members of a family is described by Herringham.[8] In all these cases the affection was in the daughter's sons. In the first case of this series, which is the only one fully described, the atrophy first showed itself in the peroneals, and later affected the thenar and hypothenar eminences and the interossei. The reaction of degeneration, which was absent at first, made its appearance later, both in the muscles of the arms and in those of the legs. Fibrillary tremors were present in the affected muscles and in a number of those apparently healthy.

As already mentioned, there is a difference of opinion as to the nature of this form of muscular atrophy. Sachs and Hoffman look upon it as due to spinal changes. The former considers it due to degenerative changes in the ganglion-cells of the lumbar cord. Hoffman, of embryological grounds, is inclined to think that these changes are of a molecular character, and that they lead to secondary changes in the nerves, beginning first in the peripheral parts. For it he proposes the name progressive neurotic muscular atrophy. Tooth and Schultze are of the opinion that the disease is one of nerve-degeneration. The only autopsies published—three in number—showed extensive nerve-degeneration, especially in the most peripheral parts, together with an ascending degeneration which involved the columns of Goll.

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[1] Revue de Médecine, sixième année, p. 97.

[2] Archiv für Psychiatrie und Nervenkrankheiten, Band xx. S. 685.

[3] Brain, vol. vii. p. 334.

[4] Ueber den mit Hypertrophie verbundenen progressiven Muskelschwund, etc., 1886.

[5] Zur Einleitung in die Elektrotherapie, Wiesbaden, 1885.

[6] Brain, July, 1887.

[7] New York Medical Journal, December 15, 1888.

[8] Brain, vol. xi. p. 230.

Those who consider it to be of primary myopathic origin are in the minority.

It is highly probable that we may have a peroneal type of muscular atrophy due to spinal, neural, or muscular changes.

THE HEREDITARY FORM OF PROGRESSIVE MUSCULAR ATROPHY (LEYDEN-MÖBIUS TYPE).

Leyden and Möbius describe, under the head of hereditary progressive muscular atrophy, what they consider a distinct clinical variety. It is characterized, they say, besides being always hereditary, by beginning usually in the muscles of the lower extremities and the back and eventually invading the muscles of the upper extremities. The atrophy is associated with hypertrophy of the calf, and, it may be, other muscular groups.

Harrington[1] describes fifteen cases occurring throughout a period of one hundred and fifty years in one family. In all the atrophy was confined to the muscles below the knees and elbows, and made its appearance before the twentieth year.

Sachs and others consider that there is no sufficient reason for the creation of a distinct type on the points laid down by Leyden and Möbius.

As heredity plays an important *rôle* in pseudo-muscular hypertrophy and also in Erb's juvenile form and the peroneal type, it follows that no type can be built on this mere fact; and as to the parts affected, we find undoubted examples of the other varieties commencing in the muscles of the back and legs. Harrington's cases are, no doubt, examples of the peroneal type.

DIAGNOSIS OF MYOPATHIC ATROPHY.

As muscular atrophy occurs from cerebral, spinal, and neural changes, as well as from primary disease of the muscles, it is important to examine into the question whether it is posssible to say in any given case what form of atrophy we have to deal with.

1. *The Distinction between Primary Myopathy and Atrophy from Disease of the Brain.*—It is alleged by some physicians that no pronounced muscular atrophy ever results from purely cerebral lesions. It is certainly

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[1] American Journal of Insanity, quoted in Jahresber. f. ges. Med., XXII. Jahrgang, p. 161.

a very rare condition, if we exclude the slight degrees of wasting that naturally result from the disuse of paralyzed parts. The very existence even of a trophic influence exerted by the brain is denied by some.

Quincke[1] reports the case of a boy, aged fourteen, who was suddenly taken ill with repeated tremors in the left arm and hand. After some time cramps of the left leg were noticed, and after a particularly violent convulsion of the left extremities paralysis followed.

Recovery of the leg was rapid, while the arm recovered slowly and incompletely and presented considerable traces of atrophy. Repeated convulsive attacks followed, and about two months after the onset the child died. The atrophy had in the mean time increased in the left arm and hand, and there were distinct traces in the left lower extremity. Before death the paralyzed parts had almost completely regained their power. At no time was there a trace of contracture.

On section, a glioma was found in the right motor area. There was no pressure exerted by it on the internal capsule or the large ganglia. The spinal cord was carefully examined from the level of the exit of the seventh cervical nerves upward, but no change whatever could be detected in the ganglion-cells of the anterior horns. The anterior and lateral pyramidal columns presented no traces of degeneration.

Two other cases of a similar nature are reported by Quincke, where paralysis of the extremities was attended by marked atrophy, coming on about four weeks after the loss of power. He refers to two additional cases, one reported by Burresi[2] and the other by Gliky,[3] where marked atrophy was found in cerebral tumors.

A careful examination of the cases referred to leaves, I think, little room to doubt that we may have a muscular atrophy due to cerebral causes solely. In all the cases reported a tumor was found implicating the motor area or its immediate neighborhood posteriorly.

The diagnosis between such a condition and primary muscular atrophy cannot present any difficulty.

2. We now come to a more important point,—that is, the diagnosis between a muscular atrophy due to primary disease of the muscles and one due to degeneration of the ganglion-cells of the anterior horns, or *the Diagnosis between Myopathic and Myelopathic Muscular Atrophy.*

The typical form of myelopathic atrophy is the disease commonly known as progressive muscular atrophy (the Aran-Duchenne or thenar type). This disease nearly always begins in the small muscles of the hand; it is for this reason called the thenar type. The cases of myelopathic atrophy which begin elsewhere than in the muscles of the hand are very few in number. Myopathies usually begin in the muscles of the shoulder, face, or back.

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[1] Deutsches Archiv f. Klin. Med., vol. xlii. p. 492.

[2] Virchow-Hirsch, Jahresbericht, 1877, Band ii. S. 124.

[3] Deutsches Archiv f. Klin. Med., Band xvi. S. 463.

In the vast majority of myelopathies the degeneration involves the white matter of the cord as well as the anterior horns. As a result of this extension, we have paresis out of proportion to the muscular wasting and hypertonus added to the symptoms of the atrophy. These symptoms are never observed in true myopathies. Frequently also in myelopathies bulbar symptoms make their appearance, from the extension of the degenerative process to the medulla.

True myelopathic atrophy is attended by the reaction of degeneration. In myopathic atrophy it is exceptional to find any electrical change other than a diminished response to both forms of current. Further, in myelopathic atrophy fibrillary twitchings are invariably present, not only in the atrophied muscles, but also in many that do not as yet show any wasting whatever.

Pain in the neighborhood of the joints in the affected limbs is frequently complained of in spinal atrophy. In my experience it is exceptional to hear of any such complaint in primary muscular atrophy.

Trophic changes are frequent in myelopathies. They do not occur in myopathies.

In both forms the degeneration is essentially progressive, but the downward progress is much slower in the myopathic than in the myelopathic variety.

Hypertrophy of certain muscles is the rule in myopathies. It has never been described in myelopathies.

Myopathic atrophies begin early in life, in the vast majority of cases before the tenth year, while myelopathies nearly always begin after the twentieth year.

Heredity is a marked feature in myopathies, but not in myelopathies.

3. *The Diagnosis between Myopathic and Neuropathic Atrophies.*—The only form of muscular atrophy (the peroneal type excluded) due to changes in the nerves that may be mistaken for myopathic atrophy is that which results from certain cases of chronic multiple neuritis. The mode of onset and course of an acute or subacute multiple neuritis is so characteristic that there is no danger of its being confounded with the disease under consideration. In chronic multiple neuritis, however, there may be a time when the resemblance is marked; but this resemblance cannot be for any length of time so close as to make the diagnosis really difficult. An inquiry into the causes of a given case of chronic multiple neuritis will nearly always elicit a history of alcoholism or a family history of tuberculosis. Chronic multiple neuritis is never hereditary. Clinically it is characterized usually by paralysis out of proportion to the atrophy, and there may be distinct symptoms of ataxia. Usually there are marked symptoms of sensory irritation. It is in those cases where the paresis is slight as compared with atrophy, and where, as now and then happens, there is no distinct involvement of the sensory fibres, that the diagnosis becomes at all difficult.

When, as sometimes happens, degenerative atrophy of the nerves exists in addition to and consequent on a true myopathic muscular atrophy, it will in many cases be difficult or impossible to apportion to each factor its due share in the causation of the wasting.

In this connection it will be appropriate to direct attention to a form of degenerative myositis of a subacute or chronic character recently described by Senator.[1]

Here there is marked atrophy, with paresis, but there are no sensory symptoms except great tenderness on pressing the muscles. This symptom, together with the clinical course of the disease, will be sufficient to make the diagnosis clear.

THE PROGNOSIS IN THE DIFFERENT TYPES OF MYOPATHIC ATROPHY.

The prognosis in all forms of myopathic muscular atrophy is very unfavorable. It is most so in the pseudo-hypertrophic form. If the patient should attain his twentieth year before the disease is very pronounced, there is even then only a slight hope that it may not advance. Gowers says that after the power of standing is lost the patient will not likely live more than seven years.

In the other varieties long periods of arrest of the degenerative process are more common, and in a considerable proportion the patient may attain to an advanced age.

THE TREATMENT OF PRIMARY MUSCULAR ATROPHIES.

In all the forms of myopathic atrophies successful treatment is out of the question. We have no positive knowledge that any drug exerts a beneficial influence. Arsenic and phosphorus are recommended, but in a disease running so prolonged and various a course it is easy to be misled as to the action of such agents. Both Duchenne and Erb report cases of arrest following the employment of electricity. Massage is recommended for patients who are no longer able to walk. In families with a predisposition to atrophy it is important that sufficient exercise be taken. Excessive exercise should be carefully avoided. It is questionable whether the use of splints or plaster bandages does not do more harm than good.

FACIAL HEMIATROPHY.

Facial hemiatrophy is characterized by wasting of one side of the face. The disease was first accurately described by Romberg in 1855, although cases of it had been reported many years previous.

Causes.—The disease is one of childhood, usually beginning from the fifth to the twelfth year. It is very rare for it to begin in adult life. It is not hereditary. As a rule, no exciting cause can be made out. In a certain proportion of cases, however, an apparent exciting cause may be traceable. A case is recorded where it followed a blow on the orbit. The case reported below apparently followed a frost-bite. In Romberg's famous case facial erysipelas followed by neuralgic pains immediately preceded the atrophy. A few cases are on record where it set in after some one of the acute infectious diseases.

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[1] Zeitschrift für Klin. Med., Band xv. S. 61.

Symptoms.—Gradual wasting of one side of the face is usually the first symptom observable. It is not uncommon, however, to obtain a history of the wasted side being at first taken for the normal one, while the healthy side was supposed to be hypertrophied. The wasting may begin in all parts at about the same time, while in other cases it may begin in one particular part, generally on the cheek. It gradually extends, and usually involves the entire lateral half of the face.

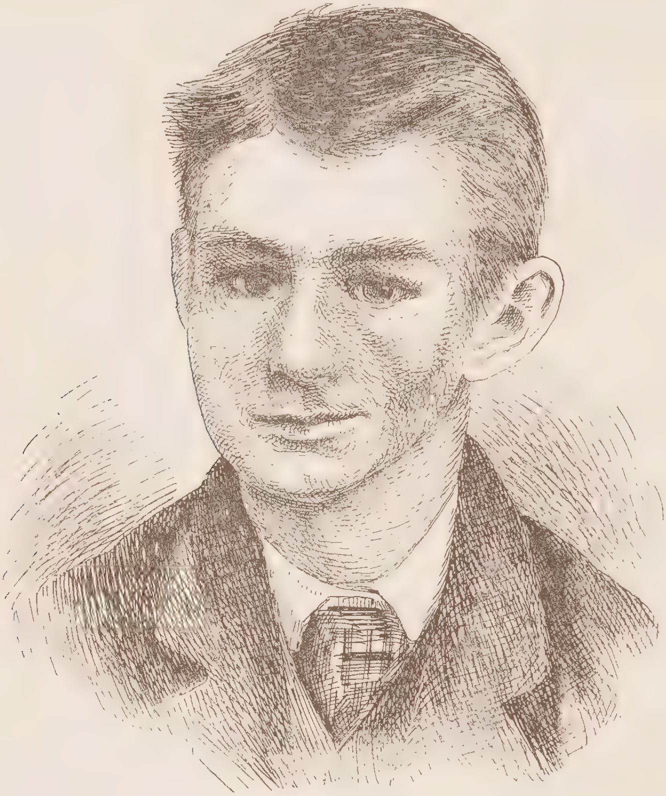
The atrophy affects all the tissues,—skin, subcutaneous tissue, muscle, and bone. The hairs drop out, and, if there be any left, they are thinner and less pigmented than those of the healthy side.

The following case, which has recently been under the writer's observation, may be taken as a fairly typical one of facial hemiatrophy:

A boy, aged fourteen, was noticed about two years ago to have the two sides of the face unequally developed. This asymmetry was considered by the parents to be owing to swelling of the right side of the face. After some time they consulted a physician, who informed them that the difference was owing to wasting of the left side and not to swelling of the right side. Eighteen months previous to the difference being noticed he was severely frost-bitten in the left cheek and ear. No history of heredity.

The striking difference between the two sides of the face is very inadequately represented by the cut on the following page. The atrophy, it will be noticed, affects only those portions of the face which are innervated by the two lower divisions of the fifth. The skin, the subcutaneous tissues, the muscles, and the bones are all atrophied. The muscles have, however, suffered less than any of the other tissues. The skin is not only thinner on the wasted side, but it also has a paler hue. The hairs are finer and smoother than those on the corresponding parts of the opposite side. The lower jaw is not only thinner, but also shorter. The teeth appear to be equally well developed on the two sides. It is difficult to estimate the degree of pure muscular wasting. It is certainly not very marked. The muscles act to voluntary impulses as well on one side as on the other.

The following is the result of repeated electrical examinations. The faradic irritability of the facial nerve is normal, being fully equal to that of the right. The response to faradization of the muscles of the left side is as well marked as it is on the right (normal side). The response to galvanization of the left facial nerve is not different from that of the nerve on the right side. The muscles of the left side show, however, a readier response to galvanization than do those of the right. Contraction is obtained on the left from one and a half milliampères, while it takes three



Left facial hemiatrophy.

milliampères to bring out a similar contraction on the right. There is no change in the normal formula; the K.S.Z. < A.O.Z. and A.S.Z. There is no increase in the mechanical irritability of the muscles or facial nerve on the left side.

Owing to atrophy of the turbinated bones and the nasal cartilage on the left side, the left nostril is wider than the right. There is distinct atrophy of the left half of the tongue, more marked towards its anterior part. The arches of the palate are normally and equally developed. There is no deviation of the uvula.

There is no affection of any of the special senses. He sees, hears, smells, and tastes as well on the left side as he does on the right. There is no disturbance of either superficial or deep sensation on the atrophic side. Repeated measurements with a surface thermometer and a differential calorimeter have failed to show any difference in the temperature of the two sides of the face.

There is no difference in the size of the globes of the eyes. Neither is there any retraction or other change noticeable. The left disk is normal.

The secretion of saliva and of tears is not lessened on the left side. There is no atrophy to be detected elsewhere.

Course.—Facial hemiatrophy progresses usually until it has attained a considerable degree, and then remains stationary for the remainder of the patient's life. It does not interfere with the general health. The term "progressive," which is sometimes used to designate it, is, therefore, not strictly correct.

Pathology.—Many theories have been from time to time advanced as to the nature of facial hemiatrophy, some contending that it is due to disease of the sympathetic, others looking upon it as an affection of the fifth nerve. A few have sought to explain its origin on the hypothesis of an increased and subsequently contracted interstitial tissue.

The only full and satisfactory post-mortem examination ever performed in a case of facial hemiatrophy is that reported[1] by Mendel, of Berlin. The patient was a woman who at the time of her death was fifty-one years of age. The atrophy set in a short time after an attack of facial erysipelas at the twenty-fifth year. The wasting involved the entire face, the anterior part of the left half of the tongue, and the muscles innervated by the left musculo-spinal nerve.

The following were the changes found after death, which was caused by phthisis. Proliferating interstitial neuritis affected the entire left fifth from its origin to its terminations. The changes were more advanced in the second division than in the other branches of the nerve. A very marked difference was found also between the right and left descending roots of the fifth, that on the left side having undergone almost complete degeneration. A similar change was found in the substantia ferruginea, the nucleus of the so-called trophic root of the fifth nerve.

The examination in this case proves conclusively that in at least certain cases of facial hemiatrophy we have to do with a neuritis of the fifth nerve. Whether these changes are the fundamental lesions in all such cases remains to be determined.

Treatment.—No cases have been reported where treatment has been of any benefit. Once the atrophy is established, be its origin nuclear or peripheral, it is useless to expect any beneficial result from electricity.

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[1] Neurolog. Centralblatt, 1889, No. 14.

HEADACHE.

By E. C. SEGUIN, M.D.

HEADACHE is a symptom of such frequency, and one that precedes and accompanies so many diseases, that to treat of it fully would require much space and a critical review of the symptoms of many of the maladies of childhood.

In some cases of non-febrile disease, headache stands out as the chief, or in some cases as the only, symptom; and our study of it will be limited to this category.

The meaning of the symptom headache has been the subject of many too *a priori* essays. Often it is judged by its location, by its nature, by its coincidence with external appearances of the face and head, and with too little regard to the patient's general condition. We believe that the attempt to judge of the significance of headache by its location—frontal, vertical, or occipital—is at the present time a vain one. We are absolutely without knowledge of the conditions which lead to the localization of the pain.

Another theoretical view, based on external appearances, is that headache, when it is the chief symptom, may be due to hyperæmia or anæmia of the brain. This, also, we believe to be a superficial and misleading view, because there is no known or necessary relation between the state of the intracranial circulation and of that of the face and head. For example, Day makes the statement[1] that nosebleed in school-children is an evidence of hyperæmia. Now, it is well known that nosebleed in children may represent local nasal lesions, general anæmia, or passive congestion dependent upon mitral insufficiency. In cases of the so-called angeiospastic form of migraine, the pallid face is, we think, rather a concomitant than a genetic state: the cause of the headache produces external anæmia, yet the essential causal condition may have nothing whatever to do with the circulation: it is far deeper and more obscure.

These considerations lead us to think that classifications of headaches based on their location, and on the apparent state of the blood-supply to the head, are of no practical value. The time may come when the location of headache will be a clue to its pathology, but it is not so now. Indeed, in the present fragmentary state of our knowledge on this point, the localization of a headache sometimes is a misleading guide; as, for example, the apparent relation between frontal pain and optical defects. In most cases, we believe, frontal headache is dependent upon general morbid states, while in many cases of eye-strain the pain is mainly or solely occipital, or even cervical.

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[1] Headaches, p. 391, 4th ed., June, 1888.

It may be best to treat fully of the most easily recognized types of headache, and leave all others for the practitioner to study by the light of the general medical history of the patient and by experimental treatment.

The most clearly defined groups of headache are:

1. Megrim or migraine (" sick-headache," " nervous headache," " neuralgia," " bilious headaches," in popular parlance).
2. Anæmic or exhaustion headache.
3. Asthenopic headache, the result of eye-strain.
4. Headache of organic cerebral disease (including syphilitic pain).

Megrim is by far the most easily recognized and definite variety of headache, though its true pathology is at present unknown.

Though usually considered a disease of adult life, it is, we think, not rare below the age of twelve years, and may affect children as young as six years and even younger. It affects the two sexes about to an equal degree.

Its chief characteristics are (*a*) direct hereditary transmission; (*b*) periodical or quasi-periodical attacks; (*c*) tendency to location in one side of the head (hemicrania); (*d*) occurrence of prodromi; (*e*) similarity of attacks in many succeeding years; (*f*) frequency of nausea and vomiting; (*g*) tendency to spontaneous cessation between the fortieth and fiftieth years of life.

These characteristics of megrim are worthy of further study.

(*a*) *Hereditary Transmission.*—This is so marked that in some families so-called nervous or sick headache may be traced through three or even four generations. Atavism may occur. In a large family several members usually escape, or suffer from only very rare ill-defined paroxysms of headache.

Transmission, in our experience, is more frequent and powerful through the mother into female children. Not infrequently the paroxysms of a child are almost identical with those of its parents, even as regards the occurrence of unusual symptoms, such as hemianopsia, aphasia, etc. As an element in the discussion of the pathology of migraine it may be well to call attention to the similarly strong hereditary transmission of ocular defects, refractive and muscular.

(*b*) *Periodicity of Attacks.*—As distinguished from all other forms of headache (except, of course, malarial neuralgia of the cranial nerves), a tendency to periodical recurrence is very characteristic of megrim. Attacks occur monthly or weekly or two or three times a month. In older female children the question of the influence of the menstrual function often comes up; but a careful review of my cases has led me to consider that event as merely an exciting cause of an attack, just as indigestion or excitement may be, and not as a *causa vera* of the disease. Other neuroses besides megrim are apt to be more pronounced at the menstrual period, simply because the nervous system is then more excitable and is sometimes profoundly disturbed. As a part of the periodicity of megrim must be mentioned its very frequent appearance on first waking or very soon after. This will assist in distinguishing it from anæmic and ordinary school headaches. Exceptions occur, and attacks may set in at any time of day.

(*c*) *Localization of the Pain at the Onset of the Attack.*—This is almost always striking and characteristic. Pain appears in one temple, or in the orbit, or (rarely) in the post-auricular area of the head,—a deep, peculiar pain, which often spreads to the whole side of the head. Rarely is the pain bilateral, and still more rarely does it extend into the face, the nose, or down the neck. In early life this hemicranial form prevails through the whole attack, but in later years, more especially after forty years, the pain may "pass over" to the opposite side of the head, preserving all its characteristics. Thus, a megrim may be a left hemicrania for twenty-four or forty-eight hours, then a right hemicrania for another similar period. It is extremely rare for both sides to be simultaneously involved, as in anæmic and dyscrasic or toxic headaches.

(*d*) *Occurrence of Prodromi.*—All other forms of headache, whether in children or in adults, make their appearance as pain pure and simple, but with megrim it is often different. Various paræsthesiæ and some suspensions of functions often precede the onset of pain. Perhaps the most frequent of these precursory signs (invaluable as guides to the palliative treatment of the attack) is some disorder of vision. Sometimes this consists in a general diffused obscuration of sight, a cloudy vision lasting from a few minutes to half an hour before any pain is noticed. In other cases the ocular prodromi are of a hemianopsic order: sometimes true lateral hemianopsia exists for a time; in other cases photopsia, or colored vision, is described by the patient, colored zigzag or fortification-like lines appearing on one side of the patient, the zigzag lines presenting somewhat of a rainbow or prismatic color. In other cases colored balls appear and move away before the patient, almost always on one side. In still other cases scintillations of light, or a central or lateral scotoma of large size, may appear as preliminary to the advent of pain. A prodromus of much scientific interest, usually combined (coexistent) with lateral hemianopsia or hemiphotopsia, is amnesic aphasia, which may last for half an hour. These ocular phenomena (which have led certain writers into the pathological fault of describing an ophthalmic migraine as an entity) are extremely interesting, especially as regards the theory of the seat of the intimate vascular disorder which lies at the basis of the megrim, but a more elaborate study of them would be out of place in this article. Suffice it to say that, to our mind, these and the sensory prodromi (to be mentioned below) indicate that spasm of one of the posterior cerebral arteries or of its most important branch—the occipital artery—is a fundamental element of megrim.

In some cases numbness and heaviness of one arm or of the neck and arm are described as prodromi. In some very rare cases nausea is a prodromus (or symptom, probably produced by anæmia of the medulla oblongata), which leads the practitioner, already biassed by medical tradition and popular opinion, into the gross error of looking upon megrim as a result of indigestion. Among the rarer prodromi may be mentioned unusual mental activity, and even excitement, on the day and evening preceding the paroxysm.

(*e*) *Similarity of Attacks during many Succeeding Years.*—The attacks, with or without prodromi, are, as it were, stereotyped. So marked is this that if subjects of megrim are at any time affected with toxæmic or anæmic headaches, or with the cranial pain of syphilis or cerebral tumor, they themselves most clearly distinguish the two forms of headache.

(*f*) *Frequency of Nausea and Vomiting.*—The occurrence or absence of this symptom of megrim leads to the popular classification of megrim into "sick" or "bilious" and "nervous" or "neuralgic" headaches,—a classification which we regret to say is largely accepted by the profession, and which we believe to be the cause of much misdirected treatment. These gastric phenomena are, we believe, always of central origin, as much so as the ocular prodromi. The frequent coincidence of indulgence in overeating, or of the eating of supposed indigestible food, with nausea and vomiting during the attack of megrim, apparently makes up a gastric pathology of megrim upon which much useless medication is attempted. In our experience, borne out by that of others, nausea is most frequent in the so-called ophthalmic form of megrim. These gastric symptoms assist in differential diagnosis, as they almost never occur in other forms of headache. Vomiting may occur with the occipito-cervical pain of cerebellar disease, but it is simple vomiting without preceding nausea: the patient empties his stomach once or repeatedly, but he does not have the distressing constant nausea of megrim.

(*g*) *Cessation of Attacks, or Transformation into another Type of Headache, betweeen the Fortieth and Fiftieth Years of Life.*—This singular phenomenon is in some respects alien to the subject in hand, but it is worthy of note as bearing, along with the time of appearance of megrim, and with its frequency of transmission, upon the theory of its production. Between the fortieth and fiftieth years there also occurs a remarkable quasi-normal event,—viz., the rapid failure and even complete loss of the power of accommodation for vision, hence a cessation of unconscious constant strain to correct hypermetropia and astigmatism.

It may be well to add a general description of an attack of megrim. Usually on waking, or soon thereafter, the patient discovers a deep-seated pain in one side of the head, generally in the temporal or in the temporoorbital or in the supraorbital region. This increases in severity, extends more or less to the whole side of the head (hemicrania), and often is accompanied by nausea and vomiting. The pain usually lasts all day, and the patient awakes the next morning free from headache and feeling very well. In other cases the onset occurs during waking hours, and may be preceded by certain peculiar symptoms (prodromi), such as temporary lateral hemianopsia or hemiphotopsia (colored fortification-like lines), colored balls before the eyes, cloudy or indistinct vision, temporary amnesic aphasia, shivering, numbness of one arm, etc. In cases of one-sided prodromi, the headache which follows is usually limited to the same side of the head and then runs the usual course. In cases preceded by general dimness of vision, the headache is apt to be fronto-temporal and bilateral. In rare cases the headache first appears as an occipital pain; this is more common in later years, when various transformations of the affection take place.

A careful study of the above points in semeiology, as applied to a child's headaches, will be sufficient to establish the diagnosis of megrim.

The pathology of megrim is extremely obscure; we are only beginning to escape from popular delusions on the subject. It is certainly not a neuralgia, as its quasi-periodic recurrence, its times of appearance and disappearance (as regards age), and its direct transmission indicate. The pain is not a *nerve pain*,—*i.e.*, one limited to the area of distribution of one or two nerves. The gastric theory is, in our opinion, absolutely untenable, since its periods of recurrence are too regular, and no system of diet suffices for its cure. It is certainly not a menstrual headache, even though female patients are more apt to have an attack at the "period." That malassimilation and lithæmia (the gouty diathesis) have something to do with the disease we are disposed to admit, but would lay much less stress on this etiological factor than we did twelve years ago.[1] In many cases the urine frequently or constantly shows deposits of oxalate of lime, uric acid, and lithates. This condition (lithæmia) is, we may here observe, frequently inherited.

The pathology of megrim to which we now incline (allowing due share to various exciting causes and to lithæmia) is that of eye-strain. A full consideration of this proposition would be impossible in this article. The principal evidences in favor of this theory are: (1) The fact that almost all subjects of megrim have ocular defects, usually hypermetropia or hypermetropic astigmatism. (2) That hereditary transmission is frequent in these ocular defects as well as in megrim. (3) That megrim is apt to make its appearance at the age (from eight to twelve years) when children begin to use their eyes steadily for near work (study). (4) That in the period of life when accommodative power ceases, megrim also disappears. (5) That we now know from experience that the full correction (when possible) of ocular defects benefits megrim more than all other therapeutic measures.

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[1] New York Medical Record, December 8, 1877.

Treatment.—The treatment of megrim resolves itself into two distinct categories.

1. *The Treatment of the Attack.*—In the first place it is best, in our experience, for the patient to give up to the attack, and remain quietly at home, in a dark, quiet room. In cases where nausea is prominent, it is best not to give any food, and to administer broken ice, or weak hot tea.

Several remedies have the power to arrest or "break up" an attack of megrim. They should be given early in the attack,—during the prodromal stage, if possible. Antipyrin holds, in our opinion, the first rank. Five grains given to a child above eight years of age every hour will often wholly arrest the paroxysm. For patients over fifteen years of age doses of from ten to fifteen grains are required. It is our practice to give from three to eight minims of tincture of digitalis with each dose, to counteract the depressing effects of antipyrin upon the heart. Caffeine ranks next in efficacy. Pure caffeine should be given, and not the citrate or "bromo-caffeine." In doses of from one to two grains every quarter of an hour at the beginning of the attack, it often completely relieves the patient. It is well to limit the number of doses to four or six, according to the patient's strength. Paullinia sorbilis, in the shape of fluid extract or of elixir, has some value, but it must be given freely even to children,—from one-half to one teaspoonful every half-hour at the onset of the attack.

When an attack of megrim is fully developed, it is almost impossible to check it. In adults, aconitine, croton chloral, gelsemium, and sulphonal may be given; but we have had no experience in the use of these remedies in children. Morphine, of course, will cut short or mitigate an attack of megrim, especially if given hypodermically; but the objections to this practice are so great that we absolutely refuse to employ it. In our experience, megrim is rendered more frequent and more severe by the use of morphine in adults, and there is no reason why it should not do the same in children. We have notes of several cases in which an occasional megrim was transformed into a daily headache, with fully-developed morphine-habit, by the mistaken kindness of the attending physician. We would strongly urge upon practitioners to abandon the use of morphine in megrim.

A great many mitigating measures may also be employed,—heat or cold to the head, applied by means of the rubber bag, sinapisms on the mastoid processes or on the neck, hot mustard foot-baths, the use of the menthol cone, mild galvanization (anode to temples and brow), emetics, etc.

2. *The Treatment of the Disease: Inter-Paroxysmal Treatment.*—It may be safely said that, owing to our ignorance of the intimate pathology of megrim, no successful treatment has yet been discovered. Until within a few years nothing was attempted in the way of systematic treatment of this disease, beyond regulating the patient's hygiene. In 1872, Dr. Richard Greene, an English physician, published a short paper[1] extolling the merits

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[1] The Practitioner (London), vol. ix. p. 267.

of extract of cannabis indica given in small doses daily for a long time, for the cure of megrim. Soon after, we began using this remedy, and published a report upon the subject in 1877,[1] corroborating Dr. Greene's results. We have continued to employ the drug, with varying success; often obtaining intervals of several months by its continued use. Few extracts of cannabis indica are good, and we are in the habit of prescribing Herring's English extract. The dose should be gradually raised from one-tenth of a grain to one-half grain after each meal; our rule being to give as much as the patient can bear, and keeping up this dose for a year or more, just as we do in the bromidic treatment of epilepsy. Arsenic, quinine, or iron may be combined in the prescription.

It will be found that many subjects of migraine have lithæmia, as shown by the constant or frequent appearance of lithates, uric acid, and oxalate of lime in the urine. This condition may be corrected by a proper diet,—viz., one consisting of a minimum of sweet and starchy foods, a moderate amount of meats, and an abundance of green vegetables, milk, eggs, poultry, and fish. The child should be made to take some regular exercise, in addition to play; to use the cold plunge- or sponge-bath (at least from May to November); and to secure plenty of sleep. An important part of the treatment of lithæmia is the use of an abundance of water, partly taken at meals. A mild alkaline water, such as one of the lithia waters in the market or artificial Vichy water, may be desirable. In the way of drugs, the free exhibition of dilute nitromuriatic acid, in doses of from three to ten drops in a tumblerful of water after meals, is the best. If strong alkalies or lemon-juice are given, they should be administered at the close of digestion,—viz., three or four hours after meals.

In cases where anæmia and debility with low arterial tension are present, the addition of strychnine, in doses of from one one-hundredth to one-fiftieth of a grain, to the dose of nitromuriatic acid is valuable. In some cases presenting the opposite condition, of plethora, high arterial tension, and especially nervous excitability, the continuous use of some form of bromide, given at bedtime two or three times a week, will assist the relief of megrim.

The recently-advocated theory that megrim, as well as some other forms of headache, is due to ocular strain, has opened up a new and apparently more rational treatment of the disease. The ocular theory of megrim is supported by a remarkable series of facts, some of which have already been referred to. Another thing in its favor is that it is extremely rare to meet with normal eyes in victims of megrim: the great majority of subjects have hypermetropia, astigmatism, or the two combined, besides, in some cases, faulty muscular equilibrium. We are so deeply impressed with the importance of ocular strain as a prime factor in the genesis of megrim that we would strongly urge that in every case a thorough examination—*i.e.*, one made while the eyes are under the full influence of atropine—should be made of the refractive and muscular power of the eyes of patients suffering from megrim, and that, too, at the earliest possible age. Many adult cases are relieved or almost cured by the correction of ocular defects, and we believe that if this treatment were applied more extensively to children, even before they begin to have headaches, much suffering might be spared, and many cases of so-called neurasthenia, or nervous exhaustion, supposed to be due to over-study in children and youths, might be prevented. To be successful, the correction of faults must be as perfect as possible, and in some cases no doubt tenotomy is demanded, and may have to be repeated. Unfortunately, in many cases perfect or easy vision cannot be secured with the means at our command. In some cases several sets of glasses have to be tried before suitable ones are found, as occasionally the glasses which theory demands are unbearable in actual use.

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[1] New York Medical Record, December 8, 1877.

As a part of what might be called the prophylaxis of megrim, especially in children of parents having the disease, we would advocate that a thorough examination of the eyes be made before they are sent to school,—*i.e.*, between the ages of eight and ten years. If faults are found, we believe that they should be corrected, even if megrim, school-headache, or simple asthenopic symptoms have not yet shown themselves.

Closely allied to this subject of the prophylaxis of megrim by the correction of ocular defects is that of school hygiene, as far as it relates to the mode of lighting the rooms and desks, and to the important question of relative height of the scholar's seat and desk, in order to obtain a normal working distance between his eyes and the book or paper. These highly important points have been long neglected, and, besides asthenopia, increased myopia, and school-headaches, spinal curvatures may arise from vicious attitudes. In the last few years efforts have been made to remedy both these sources of ill health and "breaking down," but yet the vast majority of school-rooms are defective and harmful.

It may be objected that we have given far too much space to this one form of headache; but we are impressed with its pathological importance, as being the source of great—often extreme—suffering throughout life, and as leading, through the causes which produce it, to other and more serious neuroses. How many persons are disabled for work and pleasure for days and weeks by megrim, how many form the morphine-habit by learning that the drug will cut short an attack of headache, and how many drift into neurasthenia and hypochondriasis, and are forced to curtail their education, and to restrict or wholly give up active occupation,—all because in early youth they were allowed to struggle on, unaided, against defective vision, lithæmia produced by injudicious diet, etc.; conditions which are all more or less—much more as our experience grows—preventable and remediable by medical art.

2. School-Headache.—Under this head are included simple asthenopic headaches, which appear almost daily, and are at once arrested by relief from study. The pain in these cases is usually frontal, sometimes temporo-

frontal, and may be accompanied by a sense of fatigue or of irritation about the eyes, and in some cases by injection of the eyeballs. In other cases the pain is occipital, though this is far less frequent than in adults, who very often have occipital pain as an immediate result of eye-strain (usually from insufficiency of the recti interni). The child is not anæmic, is well in most respects, but shortly after beginning his school-work the headache appears. In some children this form of headache is so frequent and severe that study must be given up, or pursued irregularly.

The differential diagnosis of this headache from the headache of malnutrition and cerebral anæmia will be considered in the succeeding section. From malingering the diagnosis is often difficult. By imitation or through evil suggestion a lazy child will claim to have headache whenever he attends school. The diagnosis is established upon the following data.

In the malingerer the facies does not indicate the suffering which is loudly complained of; the pain is said to come on at once after beginning school-work; the child can read pleasant stories, or use its eyes in games requiring fixation and accommodative effort, without pain; the ophthalmic examination is negative or nearly so, and attempted correction with weak glasses is not pleasantly received, or is flatly rejected.

The treatment of school-headache is by correction of ocular defects, whether refractive or muscular, and by better arrangement of light, seats, etc., in the school-room.

3. Headaches of Anæmia and Malnutrition.—The diagnosis of these headaches is arrived at by a careful study of the patient's general condition and by the exclusion of megrim, of asthenopic headache, and of signs of organic cerebral disease. The child is usually frail, ill nourished, and pale, eats little or no breakfast, and often has deposits of urates, uric acid, and oxalate of lime in his urine. The headache appears in the forenoon, at school or at home (independently of eye-strain), and is to a marked degree relieved by food, or by some stimulating drink, such as coffee or tea. The seat of pain is usually frontal and vertical, but the whole head may be involved. It is never a distinct hemicrania starting from one spot. There may be an appearance of flushing about the ears and face, which often leads to a mistaken view of the case and to the administration of such remedies as bromides.

The treatment of these cases consists in all measures intended to improve the child's nutrition: more sleep; a forced breakfast of eggs or meat, with tea or coffee (which we believe are not injurious to children above six years of age); cold sponging, followed by thorough rubbing of the body, and a brisk calisthenic exercise. If the home hours of meals cause an interval of more than four hours between breakfast and the mid-day meal, a portion of liquid food (milk or some form of beef-peptonoid) should be given three hours after breakfast. The mid-day meal should be generous and hot for growing children, and parents or teachers should see that this meal is eaten slowly and carefully: certainly not less than half an hour at table should be insisted on. In our opinion, growing children, during school-years, require three full meals a day, each meal to embrace some form of animal food, in order to provide material for cerebral activity as well as for body-growth. In evidently lithæmic cases, and where children tend to accumulate fat without corresponding increase in the quality of the blood and in muscular strength, sweet foods should be excluded, and starchy foods reduced to a minimum. As a matter of detail we would state it as our opinion that the prevalent American custom of eating oatmeal or other farinaceous food at the beginning of breakfast is very pernicious. This starchy food is the least useful for the child, and, besides, it satisfies his appetite, so that when the nitrogenous food (eggs or meat) is brought on the table it is refused. The animal food should, we believe, be eaten first; then if any appetite remains a small portion of farinaceous food is allowable.

The medicinal treatment of these headaches may be conducted as follows. The attacks themselves may be cut short, usually, by a stimulating food, the best of which is hot coffee or tea. Milk or a solid luncheon will often succeed. Caffeine in doses of one or two grains every half-hour may be given, or fluid extract of Paullinia sorbilis in doses of one-half or one teaspoonful every half-hour. A sinapism on the back of the neck, or the application of heat to the head (hot-water bag or hot dry cloths), may succeed. In some cases simply rest in the horizontal posture, with food, will stop the pain. A general tonic treatment by the administration of arsenic, iron, Calisaya bark, strychnine in small doses (gr. $\frac{1}{100}$ to $\frac{1}{50}$ *ter die*), cod-liver oil, and in some cases phosphorus (the best form being Thompson's tincture so diluted with glycerin as to give from gr. $\frac{1}{200}$ to $\frac{1}{50}$ *ter die*), will complete the cure. In the cases where lithæmia is present the use of dilute nitromuriatic acid in doses of from two to ten minims is indicated, and an alkaline water (one of the lithia waters or Vichy) may be given some three hours after meals. A very advantageous combination consists in the addition of from six to ten drops of Fowler's solution to a quart of the alkaline water, the whole to be consumed in twenty-four hours. In some rebellious cases of uric-acid formation a strict milk diet continued for two or three months will succeed; but care must be taken that enough milk be used,—from two to three quarts a day for a child under fifteen years. As regards iron, it will be found that it often increases headache, and it must be given in extremely small doses, or in the shape of chalybeate waters.

In addition to the above more or less well defined forms of headaches, children suffer from diffused or fronto-vertical headaches associated with various pathological conditions, which must be determined by a thorough examination of the patient. Chief among these pathological factors are:

(*a*) Passive hyperæmia due to mitral insufficiency, or to impending menstruation.

(*b*) Transitory or accidental dyspepsia.

(*c*) Genital irritation. (One of the worst cases of occipital headache we ever saw in a child was cured by circumcision.)

(*d*) Aural disease, in which case the headache is localized about the mastoid or the temporal region near the affected ear.

(*e*) Incipient organic cerebral disease: this is usually a sharp pain, often nocturnal and causing the child to cry out. In other cases it is occipital, accompanied by more or less rigidity of the neck and by vomiting unattended by much nausea. In some cases of cerebral tumor the pain is fixed in one part of the cranium; but no reliance should be placed on this as an indication of the seat of the lesion. When the lesion is syphilitic (which is rare in children), the occurrence of evening exacerbation will often serve as a guide to diagnosis.

(*f*) Simple febrile states, or the prodromal stage of exanthemata; in which the headache is diffused and throbbing, and is accompanied by the symptoms of fever.

(*g*) Malarial headache manifests itself as a diffused pain, usually accompanied by febrile symptoms. In other cases the pain is strictly in the area of distribution of one of the cranial nerves (usually the supra-orbital), and constitutes a true neuralgia, which sometimes presents strict periodicity.

(*h*) Uræmia is a fertile source of headache of great severity. It is often occipital.

The thorough study of a case of headache in a child (or an adult) is, in our opinion, one of the most difficult problems of medicine. Usually this is investigated in a superficial and unscientific manner, and the physician often allows himself to be biassed by the patient's opening statement that he has "neuralgia." The intimate relation of certain forms of headache with states of suboxidation or chemical dyspepsia is also often ignored, and the symptom is treated instead of the fundamental pathological condition. In our opinion, each case of headache (except, of course, the accidental headaches of the febrile state) should be carefully and specially studied. The family history should be investigated, the urine carefully examined (and by this we mean the examination of a series of specimens), and the state of the eyes, heart, and blood ascertained. In anomalous cases, which do not readily fall into one of the recognized categories, all possible sources of peripheral irritation should be sought for, and malingering borne in mind. Some cases require the co-operative skill of several specialists (neurologists, ophthalmologists, and gynæcologists), for in a case of chronic or habitual headache every clue should be investigated in the most scientific manner possible.

The examination of the eyes in a case of recurrent or nearly constant headache is a matter of such great importance that a word as to its manner and scope may not be out of place. In the first place, the examination should extend to the external muscular apparatus, and the balance of power of the various recti muscles at twenty feet and in accommodation should be accurately determined. In the second place, the refraction should be tested while the eye is under the full influence of atropine. We have notes of several cases in which before the use of atropine vision was normal or even above normal (at twenty feet), and yet in which after the action of atropine marked hypermetropia was found. In such cases the good vision was obtained by an unconscious accommodative effort, involving undue expenditure of nerve-force,—or, in other words, *strain*. A third point is that under some conditions even a slight fault is worthy of correction, at least temporarily. By this we mean that in certain conditions of anæmia, neurasthenia, or convalescence from acute disease, the accommodative power is enfeebled, and an habitual slight strain, which was well borne while the patient was in good health, becomes a potent secondary cause of new symptoms of the neurasthenic order.

As regards therapeusis by drugs, it should be as rational as possible at the present time. Several of the best palliative remedies (antipyrin, antifebrin, paullinia) act in a manner which we do not understand; but other remedies, such as caffeine, croton chloral, aconitine, atropine, the mineral acids, alkaline or ferruginous waters, dietetic and hygienic measures, are employed upon tolerably clear indications, and we have a fair knowledge of their mode of action. The remedies to be used most sparingly are the bromides and morphine. The former tend to depress the nervous power, and to weaken the strength of the heart and the arteries, which effects are certainly most undesirable in cases of headache of whatever sort. The latter is most dangerous. It affords a sudden complete relief to pain, yet reduces the ability to bear subsequent attacks, and is a most fertile source of the morphine-habit. From our experience we have no hesitation in stating it as our belief that hundreds of persons are rendered morphiomaniacs by the thoughtless administration of morphine for headaches. Of course there are cases in which extraordinary violence of pain or the coexistence of uræmia or of organic cerebral disease justifies the use of morphine; yet we hold to our belief that, as a rule, the physician should stand firm in his refusal to give morphine or any opiate in cases of headache.

CHOREA.

By B. SACHS, M.D.

By chorea we designate a neurosis which is characterized by irregular involuntary movements of the muscles, chiefly of the arms, legs, face, and tongue; these movements begin somewhat suddenly, are very frequent while the disease lasts, but, as a rule, cease during sleep.

Synonymes.—This neurosis is known, more especially among the Germans, as chorea minor, in contradistinction to chorea magna, a neurosis of an hysterical or a psychic character. With the latter we are not directly concerned, and the trouble under discussion will be designated as *chorea*, pure and simple. St. Vitus's dance, Scelotyrbe, Melancholia saltans, Chorea of Sydenham, are other names which are occasionally given to this disorder: the first and the last of these designations are still in common use; the other terms are obsolete.

History.—Choreiform troubles have been well described by medical authors of many centuries; but they have generally referred to the dancing manias of a religious character, so common in the Middle Ages. An outbreak of a severe form in Strasburg in the early part of the fifteenth century has given one of the names in common use at the present day, for during this outbreak the chief magistrate of Strasburg ordered those afflicted with dancing mania (*χορεία*) to repair to the chapel of Saint Vitus in Zabern, a small village in Alsace, not far from Strasburg. The name, St. Vitus's dance, is the only point of affinity between the dancing mania of old and the idiopathic chorea of the present day. Idiopathic chorea was placed on a firm footing by the descriptions of Sydenham, and it is a mere matter of justice to remember his name in connection with this important neurosis.[1]

Etiology.—Chorea is distinctly a neurosis of early life. With the exception of two cases reported by Sinkler[2] in persons over eighty years, the vast majority begin in early youth, and first attacks of this disease almost invariably occur thus early. Dr. Stephen Mackenzie has given the statistics of four hundred and thirty-nine cases tabulated for the British

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[1] The history of chorea has been well written by Soltmann in Gerhardt's Manual, vol. v.

[2] Journal of Nervous and Mental Diseases, July, 1881.

Medical Association Collective Investigation Committee.[1] Of these four hundred and thirty-nine cases, thirty-four per cent. occurred between the ages of five and ten years, forty-three per cent. between ten and fifteen years, and sixteen per cent. between fifteen and twenty years. The largest number of attacks occur in the thirteenth year of life. But some cases occur even much earlier than these tables would indicate. I have seen one case in a child a little under one year of age, and several in children between two and three years of age.

Sinkler[2] refers to a case of congenital chorea due to fright of the mother during pregnancy, and similar cases have been recorded by others. Hereditary predisposition must be taken into account. Chorea is evidently one of several neuroses which may be developed in the children of parents afflicted with epilepsy, migraine, or chorea. I have on various occasions had under treatment a father for epilepsy and his child for chorea.

Reference must be made to a special form of chorea, the so-called hereditary chorea of Huntington.[3] This form appears late in life, generally about the age of forty or later; it occurs in members of the same family, and is distinguished from the ordinary chorea by the association with it of marked psychic disturbance amounting to dementia.

Peretti[4] gives the history of four families descended from a choreic woman; two families were free, but in the other two families twelve persons were affected with chorea. Additional cases have been reported by McLeod,[5] Zacher,[6] Hoffmann,[7] and Sinkler,[8] the last-named author showing that the disease was recognized by Dr. Waters in 1841.

In June, 1890, Weir Mitchell[9] made an interesting report upon several cases of "spinal chorea," possibly of hereditary form, occurring in adult individuals and resembling dog-chorea.

Sex.—All authors are agreed as to the greater liability of the female sex. Sinkler has collected three hundred and twenty-eight cases, of which two hundred and thirty-two were females and ninety-six males. Gowers,[10] who has combined the statistics of several other writers with his own, found that of one thousand cases only three hundred and sixty-five were boys. My own experience is in entire accord with these statistics: of seventy cases, twenty-one were males and forty-nine were females.[11]

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[1] British Medical Journal, February, 1887.

[2] Pepper's System of Medicine, vol. v. p. 441.

[3] Medical and Surgical Reporter, 1872.

[4] Berlin. Klin. Wochenschr., 1885.

[5] Journal of Mental Sciences, 1881.

[6] Neurolog. Centralblatt, 1888.

[7] Virchow's Archiv, vol. xci.

[8] Journal of Nervous and Mental Diseases, February, 1889.

[9] At the meeting of the American Neurological Association. See Journal of Nervous and Mental Diseases, 1890, p. 427.

[10] Diseases of the Nervous System, p. 957.

[11] This table includes only cases seen within the last year and a half.

Race.—Statistics with regard to the influence of race upon chorea are insufficient. Weir Mitchell[1] thinks that the negro race is almost exempt from chorea, but my own experience is at variance with this. Among the very limited number of negroes who seek treatment at the New York Polyclinic, there have been a number of children suffering from chorea, and, curiously enough, all of these have been boys. Competition at school seemed to me to have been the exciting cause of these cases.

Climate has no distinct bearing, as far as can be made out, either upon the development of the disease proper or upon the causation of the individual attack, but there seems to be a distinct *seasonal* influence upon the occurrence of attacks. Dr. Morris Lewis,[2] of Philadelphia, has found that the fewest attacks occurred in October (4.1 per cent.) and the greatest number in March (15.3 per cent.).[3] On the whole, my own experience is in agreement with this; but I have never been able to overlook the fact that many of the attacks occurring in early autumn follow upon the opening of school, and that in March the strain of winter duties is greatest, and, as applied to the individual case, particularly in private practice, I have always been able to find some other than seasonal and a more plausible influence. Dr. Morris Lewis has paid much attention to this subject, and his conclusions deserve careful consideration. It is my duty, therefore, to refer to these additional facts: no correspondence has been found between the occurrence of chorea and variations in temperature, humidity, or barometric pressure; there was a correspondence, however, between the number of attacks of chorea and the number of rainy and cloudy days and the number of storm-centres that passed over Philadelphia. I repeat that all such statements must be taken with great reserve; they will gain in value if other observers in different stations reach the same or similar results.

As regards the causation of the disease itself, and not of the attacks, other factors play a much more important *rôle.* Above all, we have to mention fright, the acute diseases, particularly acute rheumatism and scarlatina, cardiac disease, and pregnancy.

Fright and strong emotional excitement are so frequently the cause of chorea that it is unnecessary to give statistics on this point. It is the direct cause of choreic attacks in fully one-fourth of the reported cases, and in my own cases it was found in more than half.

The first attack as well as recurrent attacks may be due to this cause; a first attack, as a rule, follows either immediately or within a few days after the fright. Thus, I had under my observation for a long time a child that lived near the Brooklyn Theatre at the time of the great fire. The child was startled by the sight of the flames, and within a few hours began to twitch, and soon developed severe chorea. In one case the sight of a street-brawl, in another the sight of a dead body, and in still another the news of the death of the child's father, was the direct cause of the choreic attack. In children who have had chorea trivial occurrences are apt to bring on an attack. Slight overstrain at school, the unexpected report of a pistol, a severe thunder-storm, or a severe scolding by a parent (as occurred in a private case of mine) may be sufficient to bring on an attack.

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[1] Lectures on Nervous Diseases.

[2] The Polyclinic, January, 1887: cf. also Dr. Mitchell's views.

[3] Wicke's (1844) and Gerhardt's statistics are in general agreement with this.

Acute Rheumatism.—Different interpretations have been put upon the fact, but the fact is undisputed that in a very large number of cases of chorea we also obtain a history of rheumatism; but a causal relation can be proved in those cases only in which the rheumatism distinctly preceded the attack. This occurred in about one-fourth of the cases reported by Sée,[1] Roger,[2] the British Medical Association Collective Investigation Committee, and Gowers. Eichhorst and Strümpell think the relationship between rheumatism and chorea an occasional one; Brieger[3] insists on the existence of an alternation between these conditions.

Steiner[4] states that in but four of two hundred and fifty-two cases did chorea develop during the course of acute articular rheumatism. Sturgis found rheumatism (preceding?) in twenty per cent. of two hundred and nineteen choreas, but does not think there is any causal relation, because fifteen per cent. of all children are rheumatic. Allan McLane Hamilton[5] found twenty per cent. of all school-children in New York City choreic, or affected with some similar disorder. These statistics are startling, and need verification: if correct, the coincidence of the two diseases would be more probable than a causal relation between the two.

I could obtain a satisfactory history of rheumatism in only eight of seventy cases of chorea. Romberg and Niemeyer denied any relationship between the two affections, but their objections were urged before the more careful statistics of recent years had been recorded. Sinkler found a history of rheumatism in thirty-seven of two hundred and seventy-nine cases. Hirt,[6] one of the latest writers on this subject, thinks that there is a common toxic agent, which if it affect the cortex will produce choreic movements, if it affect the joints chiefly will give rise to acute rheumatism. This is jumping at conclusions. That toxic agents may occasionally produce rheumatism and chorea I am willing to allow, but the large number of cases due to fright cannot be accounted for in this way. Here, again, the germ-theory obscures our vision. An infectious or toxic disease may produce chorea, but if it does so it acts simply as a debilitating factor, just as chorea may be due to any exhausting disease, to a profound anæmia, and the like. Among recent writers, Herringham,[7] Cheadle,[8] Mackenzie,

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[1] Mém. de l'Acad., xv. 1850.

[2] Arch. gén. de Méd., 1886, xii.

[3] Berlin. Klin. Wochenschr., 1886, No. 10.

[4] Ziemssen's Cyclopædia, vol. xiv. p. 427.

[5] American Psychological Journal, February, 1876.

[6] Pathologie und Therapie der Nervenkrankheiten, p. 397.

[7] Lancet, January 12, 1889.

[8] Ibid., May 4, 1889.

Bouchaud,[1] believe that an intimate relation exists between chorea and rheumatism.

Heart-disease precedes chorea in one-quarter to one-half of the cases.[2] I have found organic heart-disease in twelve cases out of seventy. There is no need in this instance of assuming any infectious agent as the cause of both the myo- and the endo-carditis on the one hand, and of chorea on the other: if any explanation must be given of the relationship between these two conditions, it seems much more rational to ascribe the chorea to the slight derangements and irregularities of vascular supply of the highest nerve-elements, resulting from the cardiac lesion, although there may be no gross signs of faulty compensation.

Pregnancy is so frequent a cause of this disorder that chorea gravidarum is a well-known affection. As a rule, it occurs in women who have had attacks of chorea in earlier life. A first attack during pregnancy is rare. Women under twenty-five are most apt to be affected, and the largest number of attacks occur during the third month,[3] and more particularly during a first pregnancy. Not long since I had occasion to see in consultation a young lady who had had chorea in childhood, and who had slight choreic movements of the tongue during her period of engagement. These disappeared before marriage. Shortly after marriage she became pregnant, and in the second or third month of pregnancy she developed severe chorea. Although this lasted a few months, pregnancy was not interfered with, and at full term she gave birth to a strong, healthy child.

I have known cases to develop after childbirth, and in a case seen about six months ago distinct chorea was associated with acute puerperal mania. In this case there was undoubted chorea, and the movements were very different from the ordinary restless movements of acute mania. The association of these two conditions is not surprising, if we regard both of them (the acute mania and the chorea) as symptoms of exhaustion of the entire central nervous system.[4]

In the absence of pregnancy, marriage alone, with the attendant excitements, is sufficient to produce a recurrence of chorea. This was well illustrated by a case under my observation at the Polyclinic, of a young Russian woman aged eighteen years, who had married a few months previously, who was markedly hysterical and could be easily hypnotized, and who developed chorea almost immediately after marriage, but was not pregnant when last seen, six months after marriage. The choreic movements, it is worth adding, disappeared entirely during the hypnotic state, as they do in ordinary sleep.

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[1] Rev. mens. de l'Enfance, 1889.

[2] Statistics of the British Medical Association Collective Investigation Committee and of Gowers.

[3] This statement is based upon numerous statistics.

[4] Ruheman (Berlin Dissertation, 1889) and Handfield Jones (British Medical Journal, 1889) are the recent writers on the chorea of pregnancy.

It seemed proper to introduce these statements regarding chorea in the adult, for it is almost invariably the result of chorea in early life.

Reflex chorea is frequently mentioned. The reflex origin of all functional disorders of the nervous system is urged by many writers; but, if carefully sifted, it will be found that there are very few truly reflex choreas. The only cases I have seen which were of indubitable reflex origin were due to intestinal parasites. I have in mind two cases, and only two, out of a very considerable number, in which the presence of a tape-worm seemed to account for the chorea; at all events, the latter disappeared very soon (within a week) after the removal of the worm. I am in doubt as to reflex choreas due to nasal or ocular trouble; some of these are more properly habit choreas, or, according to Gowers, habit spasms, but of those cases of true, generally facial, chorea which I have seen, which had been under the treatment of rhinologists and oculists, and were referred by them to me, all were relieved by general choreic treatment, and not by local treatment of the nose or eyes. Allowing for the fact that these cases were the very ones which did not do well under local treatment, and that those that do well do not reach the neurologist, yet, from my own experience with a large number of cases which consulted me in the first instance, and from conversation with many specialists, I feel warranted in saying that there are very few cases of true reflex chorea.

The relation between epilepsy and chorea is worth mentioning. Gowers refers to a few cases in which epilepsy dated from chorea, and I have now under observation at the Montefiore Home a woman about forty years of age who, in addition to carcinoma uteri, presents the most marked chorea of the face-muscles and of the tongue; so extreme are the choreic movements that speech is absolutely unintelligible, and the attempt to use the lips results in the most curious grimaces. In this case the chorea began after the cessation of epileptic attacks a few years ago.

Pathology; Pathological Anatomy.—The morbid changes underlying chorea are still a great mystery. Not that autopsies are wanting, for there are many such on record, in spite of the fact that chorea is rarely a fatal disease; but there have been so many different post-mortem findings described that each writer may be said to have his own peculiar view of the pathology of this disease. Those who may be interested in the older theories concerning chorea will find them without number in Soltmann's article on chorea in Gerhardt's large manual. Anything and everything, from blood-diathesis to a tumor in any part of the brain, has at one time or another been held to be the cause of chorea. Sée collected eighty-four cases of chorea on which a post-mortem examination had been made. In sixteen no changes were found in the central nervous system. In thirty-two there were lesions in the brain and nervous centres (softening and tuberculosis), and in the remainder there was congestion of the serous membranes. Ogle[1] also speaks equally vaguely of congestion of the nervous centres; Pye-Smith[1] mentions hyperæmia of the brain and cord,—statements which every one will be able to take at their true value. In 1868 Steiner[2] reported the careful examination of three cases of chorea. He found a cerebro-spinal anæmia and some connective-tissue proliferation in the upper part of the spinal cord; consequently, he looked upon chorea as a sort of "spinal irritation." This view is not shared by any of the more recent writers, although several of them have found changes in the spinal cord as well as in the brain.

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[1] British and Foreign Medico-Chirurgical Review, 1868.

Meynert[3] and Ellischer[4] have found hyaline degeneration in the nerve-cells of the central ganglia. The last-named author also found changes in the vessels of the central ganglia, as well as extravasation of blood into the connective tissue of the brain, and numerous emboli in the smallest vessels of the cortex. Flechsig has found hyaline changes in the anterior divisions of the lenticular nucleus.

Dickinson,[5] whose studies were made with unusual care, has analyzed a large number of post-mortem reports, and finds that hyperæmia of the brain and cord and numerous hemorrhages into those regions of the brain supplied by the middle cerebral artery constitute the most frequent morbid condition of chorea, but he objects to the embolic theory.

The embolic origin of chorea has been maintained by many authors, chief among them being Hughlings Jackson, who holds, furthermore, that the frequent involvement of the face-muscles proves the cerebral origin of chorea. The association of endocarditis with chorea (in seventeen of twenty-two fatal cases collected by Dickinson) lends considerable force to the arguments of those who advance the embolic theory of the origin of chorea. This explanation is unsatisfactory in that large number of cases in which there is no endocarditis, and, furthermore, a most careful examination by Gowers and others has failed to reveal emboli in the brains of persons who have died with chorea. The most direct proof of the relation between chorea and capillary embolism of the brain was given by Angel Money, who by injections of a fluid into the carotids of animals produced movements closely resembling those of chorea,[6] and this condition was associated with capillary embolism of the brain and cord.

To complete the list of post-mortem findings, we add that Golgi found calcification of the cells of Purkinje in the cerebellum (quoted by Gowers). Ellischer[7] and Lockhart Clarke found changes in the nerve-elements and connective tissue in the spinal cord, and Ellischer even describes a hyaline degeneration of the axis-cylinders of peripheral nerves. Dana[1] found an intense cerebral and spinal hyperæmia; Handford[2] found numerous small hemorrhages, thromboses, and a general dilatation of the blood-vessels in the medulla, pons, and spinal cord. Garrod[3] attributes chorea to an overgrowth of connective tissue in the nerve-centres, while Grosse[4] proclaims himself an adherent of the theory of the embolic origin of chorea.

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[1] Guy's Hospital Reports, 1874.

[2] Jahrb. f. Kinderheilkunde.

[3] Allg. Wien. Med. Zeitung, 1868.

[4] Ziemssen's Cyclopædia, vol. xiv.

[5] Medico-Chirurgical Transactions, 1876, vol. xli.

[6] Lancet, 1885.

[7] Virchow's Archiv, vol. lxi.

From the preceding account of post-mortem findings but one inference can be made: the true pathology of chorea is still unknown. Such facts as we have obtained point to a wide-spread nutritive and functional disturbance of the higher nerve-elements throughout the central nervous system. This disturbance need not be due in every case to one and the same cause. The ganglion-cells and higher nerve-elements are very sensitive to the slightest changes of blood-supply. In one case a general hyperæmia may be sufficient to disturb their normal blood-supply, and in another case capillary emboli may produce the same result. As there is good reason to suppose that irritation of gray matter anywhere in the central nervous system may give rise to epileptic convulsions (how else can we explain the initial convulsions of anterior poliomyelitis?), so a change in the nutritive condition of the ganglion-cells and higher nerve-elements in any part of the gray matter of the central nervous system may give rise to choreic movements. That chorea is in most instances a *cerebral* disease may be inferred from the very frequent association of the severer forms with psychic changes, from the involvement of the face, as Jackson pointed out, and from the association with epilepsy, as proved by cases of Gowers, and by my own case, recorded on p. 845.

The occurrence, or rather the persistence, of chorea in dogs after section of the spinal cord, and the very remarkable cases in man, reported by Weir Mitchell, and resembling dog-chorea, seem to me to point to the possibility of unusual forms of chorea being due to involvement of the spinal cord.

In conclusion, let me remind the reader that the post-hemiplegic chorea of children is quite as apt to occur after cortical as after intra-cerebral lesions. Inasmuch as I have allowed that gray matter anywhere in the brain or the spinal cord may be the seat of the lesion of chorea, I have no reason specially to defend or combat the older view of the thalamic origin of chorea. In some cases the lesion may be in the thalamus, but it may also be in the lenticular nucleus, as in the case of Flechsig, or in any other ganglionic mass.

Symptomatology.—Involuntary and irregular twitching movements of any muscle or groups of muscles of the body constitute the cardinal symptom of chorea. The muscles of the hands and fingers and of the face and tongue are most often affected, but it is not rare to find the leg-

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[1] Medical Record, October 19, 1889.

[2] Brain, July, 1889.

[3] Lancet, November 23, 1889.

[4] Berlin. Klin. Wochenschr., August 19, 1889.

and trunk-muscles also involved. These twitching movements are apt to be worse on voluntary effort, but they also appear at shorter or longer intervals when no muscular effort is attempted; they may become most marked if the patient makes a determined effort to keep the part absolutely quiet.

In addition to these twitching movements there is a general restlessness of the body; while under examination the patient changes his position every second,—rests first upon one foot, then upon another, keeping up a constant change. If the physician examine the hands, the twitching may be inhibited in them for one moment, but some other part, the face or the leg, will twitch all the more for the time-being.

In a large number of patients but one-half of the body is involved, and in that case we speak of hemichorea. As far as can be gathered from extensive statistics, right and left hemichorea are equally frequent. This is fully confirmed by my own cases.

As a rule, the twitching movements are so distinct from the start that no special examination is needed for purposes of diagnosis; in some instances, however, the disease, or a choreic attack, begins with a general restlessness, and the choreic movements can be discovered only upon close inspection of the hand or tongue. If the nature of the disease is in doubt, I ask the little patient to place its hand quietly upon my own, or between my two hands: in this way the irregular choreic movements can be easily seen or felt. The true nature of many a trouble which appears to be nothing more than a "little nervousness" may thus be detected. The very greatest importance, and more than is generally allowed, should be attached to the tongue. *In doubtful cases choreic movements of the tongue will often prove the nature of the disease.* I have found this to be true in a number of instances of supposed habit chorea or habit spasm of the face-muscles.

If the tongue, when protruded, shows the characteristic choreic movements, it is well to regard the disease as true chorea. The exact nature of these tongue-movements is difficult to describe; they are slow, coarse, and rhythmical, and when the tongue is protruded the mouth is opened unnecessarily widely, and the tongue is soon caught between the teeth. These movements give rise to what I should be inclined to call the "facies" of chorea.

All the movements of a choreic patient (except in the milder cases) are characterized by extreme awkwardness. This is well shown if the patient fastens or unfastens his clothes, in raising a glass of water to the lips, in attempting to hold the pen in writing; in short, in whatever muscular effort the patient may make. This awkwardness and the constant jerking of the head and body are the source of greatest annoyance to the patient. In fully nine-tenths of the cases the patient, while annoyed, does not appear to be fatigued by the jerking movements of the muscles; but in the remaining tenth the movements are so severe that they lead to great exhaustion. It is fortunate for these patients, as for all other choreic patients, that the movements cease during sleep. Two years ago I had under treatment a little girl, six years of age, the child of healthy and intelligent parents. I had carried the child successfully through its first attack of chorea; in the second attack, which came on after a fright, the movements were so extreme and sleep was so poor that within a few weeks the child died of exhaustion; the heart had been involved in the first attack; it was not involved in the second attack, except that the almost continuous and extreme movements increased the work which it had to perform.

This is the only fatal case of chorea I have seen among a large number of severe cases. In the severer forms the movements are often so excessive that the bed must be well padded, or the patient be watched constantly, to prevent injury to the body.

Some weakness of the muscles is regularly associated with the choreic movements. Gowers has proposed the term "paralytic chorea" for those cases in which there is marked paralysis, but I can find no sufficient excuse for the introduction of this term. The difference is one of degree, not of kind.

Speech is frequently involved. The choreic movements of the tongue and laryngeal muscles may make speech altogether unintelligible. In the milder cases there is a little awkwardness of articulation, a tendency to break off words, to begin sentences again and again, but nothing more.

Deglutition may be difficult; the tongue is frequently bitten, and, from the awkwardness in the use of knife and fork and in passing food to the mouth, the patient is an ungainly sight while at his meals.

Mention should also be made of *laryngeal* chorea, a special form of chorea in which the laryngeal muscles are chiefly affected, the result being a peculiar bark, which is repeated at short intervals. These cases are sometimes mistaken for cases of hysterical bark, but the general restlessness and the age of the patient, and the choreic movements of the tongue, make the diagnosis an easy one. A few years ago I saw a typical case of the kind at the Polyclinic. The patient was a girl ten years of age, who began to "bark" after a sudden fright. The child presented other symptoms of chorea, and recovered under the usual treatment for chorea. There was no symptom of hysteria in the case. From my own experience I infer that this is the rarest form of chorea.

The *electrical reactions* are sometimes slightly altered in cases of chorea. Rosenthal, Benedikt,[1] and Gowers found an increased response to the faradic and galvanic currents on the part of the muscles and nerves of the affected side. This could be determined only in cases of hemichorea. Some have asserted that the reaction of degeneration with qualitative galvanic changes (A.C.C. $>$ K.C.C., etc.) occurs in some instances. I am inclined to question the truth of this statement. I have made a large number of electrical examinations of choreic patients, and have never found a change of the normal galvanic formula. If the reaction of degeneration be present, some complicating disease should be looked for.

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[1] Quoted by Gowers.

Mental disturbance is not unknown in chorea, though it is an exceptional occurrence. As a matter of fact the mental calibre of choreic patients, and of choreic children in particular, is rather above than below par. Children who by means of a better mental development stand head of the class, who work for prizes and win them, children who are under constant mental strain, and about whom parents and teachers make much ado because they are bright,—these are the very ones most apt to be attacked by chorea. In some exceptional cases there is a "chorea of the brain," as well as of the body, and in those patients a maniacal condition is associated with chorea. It is a rather rare occurrence to find cases which were at the beginning distinctly choreic, and which became maniacal later on. The reverse is much more frequent. In many cases of acute mania, particularly in young women, I have noticed that the movements of the extremities and of the tongue were typically choreic.

A majority of the patients with severe chorea show at the start a marked irritability of temper; some exhibit distinct apathy amounting almost to stupor. In a very few cases, particularly if the chorea becomes chronic, a condition of dementia may ensue. But I repeat that the mental disturbances are generally slight and transient.

Complications.—Whatever our views of the causal relation may be (see p. 843), the fact remains that rheumatism and cardiac disease are the most frequent complications of chorea. Acute articular rheumatism is a frequent and immediate forerunner of chorea; it may also appear unexpectedly in the course of an attack of chorea. Meynet,[1] Barlow and Warner,[2] and Hirschsprung[3] describe small subcutaneous nodules as evidence of the rheumatic diathesis in cases of chorea. In the course of an attack of chorea with acute articular rheumatism, endocarditis may appear. In such cases the endocarditis is to be attributed to the rheumatism rather than to the chorea.

The condition of the heart should be frequently examined. The frequency of anæmia in cases of chorea makes it incumbent upon the physician to allow for hæmic murmurs and for a slight dilatation of the heart, which may be due to profound anæmia. But in a very large number of patients the murmurs and other diagnostic signs will point to the existence of organic heart-disease. Mitral regurgitation is by far the most frequent form. In the statistics of the British Medical Association Collective Investigation Committee there were one hundred and sixteen cases of mitral disease and only six of aortic disease. Gowers, who criticises these statistics justly enough, states that he found only two instances of aortic regurgitation among two hundred and fifty cases of chorea. Sinkler found cardiac murmur in eighty-two out of two hundred and seventy-nine cases, but he does not decide how many were due to organic cardiac disease. It may at times be difficult to determine this question, but if a patient whose heart was normal develops a cardiac murmur while under observation, the probability of an organic lesion is very great. As before stated, organic disease of the heart was positively present in twelve of seventy cases examined for this special purpose. An excess of urea and phosphates has been found in the urine of choreic patients, but very little importance is to be attached to this, as also to the exceptional occurrence of optic neuritis.

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[1] Lyon Médical, 1875, No. 49.

[2] Transactions of the International Medical Congress, 1881, vol. iv. p. 116.

[3] Jahrb. f. Kinderheilkunde, March, 1881.

Convulsive attacks are referred to by several authors. I doubt whether this occurred in cases of true chorea: while there is no reason why chorea and epilepsy may not occur together, I have not seen a single case in which such an association existed. The cases of this description which have been reported have been, as a rule, cases of hemichorea; and it is possible that these convulsive attacks, as well as the hemichorea, were of the post-hemiplegic order, and due, therefore, to organic disease of the brain.

Duration.—The duration of the disease varies greatly. As the disease is very apt to relapse, it is better to speak of the duration of attacks. A single attack may last from three weeks to six months, and even longer. The average duration is conceded on all sides to be ten weeks. The British Medical Association Collective Investigation Report, as amended by Gowers, shows that fifty-seven per cent. of all cases last between one and three months. With the cessation of a single attack the disease can scarcely be said to disappear, for the slightest cause, a fright, an acute disease, overstrain, or mere climatic and seasonal changes, may bring on another attack. Statistics are not available on this point, but it is fair to assume that a majority of the cases of chorea suffer at least one relapse. Of seventy cases which I have tabulated with the assistance of Dr. Peterson, we observed the first attack in thirty-five, the second in eighteen, the third in eleven, the fourth in four, the fifth in one case, and the sixth in one.

This is in agreement with the general experience that two and three relapses are very common. In a few cases the disease becomes continuous and chronic, as in the patient of Meldner,[1] who developed chorea in early life and remained choreic until his death at the age of sixty-six years.

The interval between the relapses varies from a few weeks to one or two years, except in cases of chorea of pregnancy, in which an interval of ten years or more may have passed between successive attacks. The female sex, for evident reasons, is more prone to relapses than the male sex.

Successive attacks are very apt to imitate the first attack in every respect. If the first attack be one of hemichorea, later attacks are apt to be of the same order. It is generally supposed that later attacks are apt to be milder than a first attack. If this be the rule, there are very marked exceptions to it; in my own experience, a second or third attack may be even more serious than the first. Death in a second or third attack is supposed never to occur, yet the little patient referred to above died in a second attack of chorea. All such sweeping statements should, therefore, be accepted with great reserve.

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[1] Wochenbl. der Gesellsch. der Wiener Aerzte, 1869.

Diagnosis.—The movements of chorea are so distinctive that it is not easy to mistake them for anything else. Cases of imitative or possibly hysterical chorea may at first sight so closely resemble true chorea as to lead to a mistake in diagnosis; but in hysterical cases, the perfect rhythm of the movements, the atypical onset, the prolonged duration of the disease, and the general bearing of the patient, will give a clue to the hysterical character of the trouble.

Post-hemiplegic choreic movements are apt to be mistaken for true chorea, or rather hemichorea.

In an article by the author and Dr. Peterson it was shown that choreic movements persisted in six out of one hundred and five cases of infantile hemiplegia, and in one out of twenty-four cases of diplegia.

About four years ago I was asked by a colleague to see a case of persistent chorea, which, he said, would not yield to the ordinary treatment. The doctor had noticed the chorea, but had overlooked the hemiplegia, the result of an apoplectic attack several months before I had seen the child. It will be easy to avoid an error of this kind if the physician will inquire into the previous history of the patient, and if he will in every case examine the child for possible hemiparesis, and will also remember that in cases of post-hemiplegic chorea he will find, as a rule, some rigidities and marked increase of the tendon reflexes on the paretic side or in the paretic limb (the leg may recover; a paretic or contractured arm or hand may be the only sign of a preceding hemiplegia).[1] Moreover, in these cases of hemiplegia the choreic movements persist, and do not occur in attacks, nor do they yield to ordinary treatment.

A confusion with epileptiform convulsions is scarcely conceivable, for the attacks of the latter are less frequent, and there is generally some momentary loss of consciousness, besides other symptoms pointing to epilepsy. But only very recently I was asked by a colleague to see his three-year-old daughter, whose trouble puzzled me for a time. The child would give sudden and very quick twitchings of one arm and of a leg; if these twitchings occurred while the child was walking or running across the room it would suddenly stand still, evidently surprised by these jerking movements. It was natural to think of petit mal, but it was only by the frequency of the movements, by close inspection of the character of these twitchings, and by the general choreic behavior of the child that petit mal could be excluded and the disease recognized as true chorea. The case yielded also to the ordinary anti-choreic treatment.

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[1] See Sachs and Peterson, Journal of Nervous and Mental Diseases, May, 1890.

Prognosis.—On the whole, the prognosis of chorea is extremely favorable. Death occurred in only two per cent. of the cases collected by the British Medical Association Committee. Sinkler states that in Philadelphia in seventy-four years there have been but sixty-four deaths from chorea. If a patient survive the first attack, the prognosis of a succeeding attack becomes still more favorable, with the rare exception of such a case as I have referred to. It is well, however, to be on one's guard in stating the length of time which an attack is apt to last. So much depends upon the kind of care a patient receives, that it is a safe procedure to say that, if all directions are carried out strictly, the child may recover from a first attack in from six to ten weeks, unless complications should arise. Some cases get well much more quickly, but these are, after all, the exceptions. The more severe the movements are at the start, the more prolonged the course of the disease is apt to be, though it is certain that severe cases under proper treatment will yield much more quickly than milder cases that are improperly handled. The danger of relapses under unusual strain or excitement should be borne in mind.

Treatment.—There is every reason to believe that some of the milder cases of chorea would get well without any treatment whatever; but no disease, not even epilepsy, calls for more active treatment than chorea does in its severe forms.

Rest, absolute rest, is the *sine qua non* of all treatment. I am so thoroughly convinced of this that I should be willing to abandon all medicinal treatment rather than allow a patient with a moderately severe form of chorea to walk about for the first few weeks of treatment. It is my habit to refuse to undertake treatment unless parents promise to carry out this order strictly. In the milder forms, a few hours' rest in the morning and afternoon may be sufficient to control the movements, but in all other cases the patient, at whatever age he or she may be, should be put to bed at once. Often it will be necessary to keep the mother or a nurse sitting at the bed for a few days to keep the child quiet and to prevent any harm coming to the child from the excessive violence of movements. After a few days of enforced rest a decided improvement is noticeable. In moderately severe cases, absolute rest of two weeks should be insisted upon. After the expiration of this term, the patient is allowed to remain out of bed for a few hours at a time, and this is continued, unless the choreic movements should increase, in which case rest in bed must again be insisted upon. For some weeks the patient must not be allowed to indulge in any violent exercise; riding, dancing, bicycling, should be prohibited altogether. Such exercises should also be forbidden in those mildest cases which do not require absolute rest in bed.

This rest-treatment can be carried out in dispensary practice as well as in private practice. It is our custom at the Polyclinic to give directions for one or more weeks, and to have patients call at long intervals, while parents or relatives are expected to report regularly. While in bed, children can be pleasantly entertained by others, and, if the movements are not excessive, may be allowed to play or read in bed; but the reading-matter should be carefully selected so as to keep the patient's mind free from all excitement. If these details are carefully attended to, the result of treatment will be all the more satisfactory.

Next in importance to rest is a nutritious and easily digestible diet. I am in the habit of saying in my lectures on the subject that milk and rest will cure most cases of chorea. Inasmuch as a large number of choreic patients are profoundly anæmic, a diet of milk and eggs, of koumys, or of matzoon[1] is the very best.

The monotony of daily life can be varied to advantage by the use of lukewarm baths. The immersion in a cold bath or the wet-pack with subsequent friction is not to be recommended. Whatever good either may accomplish is counterbalanced by the shock of the cold water. It is a better plan to bathe the patient in lukewarm water, to add cold water to the bath while the patient is in the tub, and by squeezing out a sponge held over the neck and back of the patient to get the effect of dripping water upon the skin. After the bath (which should not be given until the first signs of improvement are visible) the patient should be kept absolutely quiet and wrapped up warmly. In spite of the excellent effects of such methods, we are compelled in nearly every case to resort to

Medicinal Treatment.—Among drugs arsenic is still *facile princeps.* Dr. Seguin, in a paper[2] which deserves to be read by every practitioner, places arsenic first and rest second in the treatment of chorea. While I think that this order should be reversed, it is due Dr. Seguin to state that in his opinion "physicians, almost without exception, give nearly useless doses of arsenic." Dr. Seguin regards eighteen to twenty-five, or even twenty-seven, drops after each meal as the "really efficacious dose." Very free dilution in some alkaline water is necessary, and the whole quantity may be taken in divided drinks within an hour after a meal. Having full confidence in Seguin's therapeutic suggestions, I deem his method worthy of trial; but I believe that so much more can be accomplished by rest than by arsenic that these excessive doses will not often be necessary. In every case it will be wiser to begin with only four or five drops of Fowler's solution *ter die,* and to increase gradually. In case of excessive restlessness I am in the habit of prescribing the arsenic together with the elixir of the bromide of potassium, or in case sleep is slightly disturbed I give the evening dose only in this way, for it is a fact to which all are agreed that large doses of bromide do not act favorably in chorea. Bastian[3] and Schrötter,[4] among recent writers, make free use of chloral and bromides.

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[1] Very few children will, in my experience, continue taking either koumys or matzoon for any length of time.

[2] New York Medical Journal, March 21–May 31, 1890.

[3] Brain, 1889.

[4] Wiener Med. Wochenschr., 1889.

The only other drug which I can conscientiously recommend is the tincture of cimicifuga. From fifteen to thirty drops three times daily may be administered in cases in which arsenic[1] is not tolerated by the stomach.

I have never been compelled to resort to the hypodermic injections of arsenic, as recommended by Eulenburg and Hammond.

Among the innumerable drugs which have been recommended I have made a trial of the oxide and sulphate of zinc, of physostigmine, of the salicylates (Weir Mitchell), and of antipyrin (Simon and Legroux), without feeling that any one of these drugs is superior or even equal in efficiency to arsenic.

In all but the mildest cases narcotics and sedatives will occasionally be necessary.

In the earlier stages of chorea the patient must obtain sleep at every cost. Chloral (fifteen or twenty grains per rectum) is to be preferred. Chloralamid (ten grains) and sulphonal (fifteen, twenty, or thirty grains, according to the age of the patient) may be substituted for chloral. In a few cases of extreme restlessness and insomnia I have obtained good results with hyoscyamia (tablets of the one-hundredth of a grain), which can be administered in the afternoon and evening. As the drug is tasteless, active, and readily absorbed, I can see no sufficient excuse for hypodermic injections. Hyoscyamia is specially valuable in cases complicated with acute mania. Here the effect of the drug is at times miraculous. Morphine should never be given. I regret to see its use recommended by a recent author (Hirt). Other drugs will act more promptly without having any of the disagreeable effects of morphine or the opiates.

Very few cases of chorea get well without at some time requiring a heart tonic. Digitalis in drop doses of the fluid extract, or from three to five drops of the tincture of strophanthus three times daily, should be given in case of heart weakness or feeble pulse. And, lastly, we are often compelled to resort to the preparations of iron (tincture of the chloride of iron, fifteen to thirty drops *ter die;* Blaud's pills, four to six daily; and various elixirs and peptonates of iron). Many a case will do better on rest and iron than on rest and arsenic; but rest above all things is the mainstay of treatment.

Electricity has been advised by Erb and others. The generally sedative action of the galvanic current may be employed to good advantage, particularly in the evening, when it will help to bring on sleep. A moderate stabile current (fifteen to twenty cells, not above ten milliampères) applied to the spine will suffice. I am opposed to the use of the galvanic current to the head.

Massage may be given in selected cases for the purpose of improving the general nutrition and possibly of lessening the choreic movements.

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[1] Arsenic, like the iodides, is frequently not well borne at first; after a few days' intermission we can go on increasing the dosage without incurring further trouble.

Much can be done to avoid relapses, if patients are kept from school until every trace of chorea has disappeared, and if they are not allowed for a year or more after an attack of chorea to undergo any severe mental strain.

Periods of examination and of prize competition are fraught with the greatest danger for such patients. Girls who have had chorea are in danger of relapses at the age of puberty; moderate healthful exercise and a general attention to all principles of hygiene are of importance.

The danger of chorea during pregnancy should be kept in mind, but no one would for this reason object to a girl's marriage, unless she have chorea at the time of the proposed marriage; for of those who have had chorea in early childhood, very few, after all, develop chorea during pregnancy.

CONVULSIONS IN INFANCY AND CHILDHOOD.

By MORRIS J. LEWIS, M.D.

A convulsion should be looked upon as a symptom, and not as a well-defined disease; but as in children this symptom may be the only one of prominence in many cases, and as we, unfortunately, have very much to learn in regard to the etiology and pathology of the various conditions of the nervous system which give rise to this symptom, it may not be inappropriate—and it is certainly more convenient—to treat it as a separate affection, always bearing in mind that it is merely a symptom.

DEFINITION.

A convulsion (from *convello*, "I rend, overturn, or tear in pieces") means any violent perversion of the animal movements (Dunglison); and under this head would be included chorea, cramp, and tetanus; but in the sense intended in the present article it may be defined as a violent, irregular, involuntary contraction of the muscles, occurring in paroxysms, and often accompanied by insensibility. These movements may be general or strictly confined to one side of the body, may be tonic or clonic, may affect only a limited group of muscles or a larger number, and one form may merge into another. More or less complete loss of consciousness is a usual accompaniment.

The term *eclampsia* (either from ἔκλαμψις, "brilliancy," "flashes of light before the eyes" (from λάμπω, "I shine"); or from ἐκ, and λαμβάνω, "I seize hold of") has been applied by many authors to the convulsions accompanying pregnancy, uræmia, etc., as well as to those of infancy; and it is apt to mislead unless, as suggested by Nothnagel, it is applied solely to the convulsions of infancy and childhood that are to be considered independent of gross lesions: therefore, from this point of view, eclampsia has been defined by Ross to be "an acute affection arising without structural lesion of the nervous system, and characterized by partial or general convulsions accompanied by a more or less complete loss of consciousness."

ETIOLOGY.

Convulsions in children depend upon abnormal conditions of the central nervous system, concerning the nature of which we still need much information; they are, in all probability, due to an exaltation of the lower nerve-centres, or, more frequently, to a suspension of the inhibitory power of the higher cerebral centres, which latter condition is most probably due to an altered state of the cerebral circulation, whereby there is a suspension of the normal supply of nourishment and an interference with the removal of waste material by the blood. Both of these conditions may exist at the same time.

A convulsion may be caused by the irritation of foci of cerebral disease; by reflex irritation, of whatever nature, proceeding from the central or peripheral nervous system; by abnormal states of the blood as found in the acute specific and other fevers, in diseases of the kidneys, and in acute poisoning, possibly also by auto-infection by ptomaines; by an anæmic state of the brain, such as follows severe hemorrhages and profuse diarrhœas; by the interference with the proper oxygenation and decarbonization of the blood in asphyxia, causing vaso-motor spasm; or by any profound interference with the circulatory and respiratory apparatus.

Acute intracranial pressure may cause convulsions by producing a capillary anæmia, and it is possible that acute cerebral hyperæmia may also act in the same manner. High temperature of the body is in itself a fertile source of convulsions.

All recent investigations tend to prove that the action of the spinal cord proper—and by this is meant the portion below the medulla oblongata—in these attacks is that of a conveyer and not that of an originator of nervous force. In convulsions due to cortical lesions Horsley and Gotch,[1] by a series of brilliant experiments, have proved that both the tonic and the clonic convulsions are entirely of cerebral origin, the spinal cord merely conveying the impulses to the muscles. The same is probably true of many of the convulsions in childhood besides those due to cortical lesions.

Many conditions predispose to this affection, particularly infancy, with its anatomical and physiological peculiarities and diseases. Many authors, for the sake of convenience, divide the convulsions of infancy into three classes: first, "*essential*," those which are reflex in origin and not due to any tangible organic alteration; second, "*sympathetic*," those which exist in various systemic diseases not accompanied by any gross organic lesion; third, those which are "*symptomatic*" of some well-recognized organic affection. This division is purely arbitrary, and as our knowledge advances many of the cases now called essential and sympathetic will be considered symptomatic of some other condition.

Many interesting experiments have been undertaken to discover what changes in the central nervous system are necessary in order to produce convulsions, but it is not within the scope of this article to discuss fully this portion of the subject: a volume in itself would be required to do justice to it. A few facts, however, should be referred to. Kussmaul and Tenner[1] were able to produce convulsions in rabbits by producing a profound anæmia of the brain by hemorrhage, by tying the cervical arteries, and on one occasion by electric excitement of the vaso-motor nerves of the head after partially cutting off the cerebral blood-supply by ligation. They found that arterial congestion of the brain was not capable of producing epileptic convulsions, and conclude that "all theories are false which assert the epileptic attacks to be derived from a sudden determination of blood, whether active, passive, or mixed," and that the medulla oblongata seems frequently to be the spot from which eclamptic and epileptic attacks proceed; but, in contradistinction to this view, Nothnagel[2] considers it beyond question that typical epileptiform convulsions may arise from cerebral hyperæmia.

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[1] Remarks before the Congress of American Physicians and Surgeons, Philadelphia, Medical News, September 22, 1888.

The former observers consider that convulsions may arise from sudden arrest of the nutrition of the brain in three ways: 1st, rapid losses of blood; 2d, sudden stoppage of the flow of arterial blood to the brain; and, 3d, rapid transformation of the red blood, by which it is rendered incapable of nourishing the brain.

Brown-Séquard[3] considers convulsions produced by hemorrhage to be identical with those caused by asphyxia, and that they are due to the irritation of the nervous centres by the carbonic acid which then accumulates in the blood that remains in the system, and not to any alteration in nutrition, which he believes cannot take place in so short a time.

This author's well-known experiments on guinea-pigs show that after injuries to various parts of the nervous system an "epileptic condition" was developed in the animals, so that a trivial irritation of the skin in special areas was sufficient to develop a well-pronounced epileptic convulsion. Nothnagel[4] places the "convulsion centre" in the pons, and has found that reflex irritation of this part, particularly by irritating a certain portion of the floor of the fourth ventricle, suffices to throw the whole body of voluntary muscles into tonic and clonic spasm.

Gowers[5] considers that restraint is the highest function of nerve-cells, and that self-control and the capacity for being controlled are higher functions than liberation of energy; and explains the convulsions following hemorrhage on the theory that the first effect of failing function may be the liberation of energy.

He arrives at the conclusion that the theory of vaso-motor spasm as the immediate cause of the fit is uncalled for and unproved; the imperfect nutrition of the gray matter of the cerebrum, or perhaps of the medulla, being the cause of the lowering of the functions of the same, with the consequent liberation of energy, which thus becomes a sign of failing function.

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[1] New Sydenham Society's Translations, 1859, vol. v.

[2] Ziemssen's Cyclopædia, Amer. ed., vol. xii.

[3] Lectures on the Diagnosis and Treatment of Functional Nervous Disorders, 1868, Part I. p. 37.

[4] Quoted in Ziemssen's Cyclopædia, vol. xiv. p. 198 and vol. xi. p. 281; also Virchow's Archiv, vol. xliv.

[5] Diseases of the Brain, London, 1885, p. 47.

May not the vaso-motor spasm be the starting-point of the anæmia which causes a change in the nutrition of the gray matter?

It, however, remains to be said that we are still very much in the dark as to the immediate processes producing convulsions; but of the predisposing causes we are not so ignorant.

Dickson[1] states that every advancing step tends to the confirmation of the statement that the proximate cause of the attack is cerebral anæmia and arrest of the brain's nutrition, and that the muscular manifestation is the result of the loss of cerebral control.

Marshall Hall considered such seizures due to an impeded ebbing of the venous blood from the brain, and Solly brought forward the theory that the attack was "a determination of blood to the head" causing an increased generation of nerve-force.

Many writers still appear to believe that this is the main cause of convulsions, and much of the treatment for infantile convulsions now in vogue is based upon this theory.

J. Hughlings Jackson[2] conceives of the central nervous system as grouped into three tiers of sensory-motor centres,—the lowest, limited above by the pons; the middle, the so-called sensory and motor cortical centres; and the highest, the brain in front of the latter centres,—all parts of the body being represented in each tier. The two upper tiers control the lowest, which latter is the first to be differentiated, and about the only part of the brain at all developed in newly-born children, and, on account of the want of controlling influence of the as yet undeveloped higher tiers, is apt to discharge nervous force on slight provocation. Convulsions may occur from discharges of nerve-force from each of these tiers.

Fischer[3] states that many facts go to show that the hemispheres bear the brunt of the shutting off of the arterial blood-supply, while the medulla suffers much less or is even stimulated. It would appear from this that in this condition the controlling influence of the higher centres over the lower not only is withdrawn, or at least diminished, but at the same time the latter is stimulated; there is on this theory a double reason given why the various reflex influences acting upon the cerebral circulation in childhood and infancy through the vaso-motor system should give rise to convulsions.

In the present article the greatest stress will be placed upon the so-called "essential" convulsions of infancy and childhood, and, although it will be necessary to discuss the various structural and other causes that are known to give rise to convulsions, the reader is referred to the appropriate articles to be found elsewhere in this work for a fuller consideration of the diseases in question.

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[1] Guy's Hospital Reports, 1873, 3d ser., p. 18.

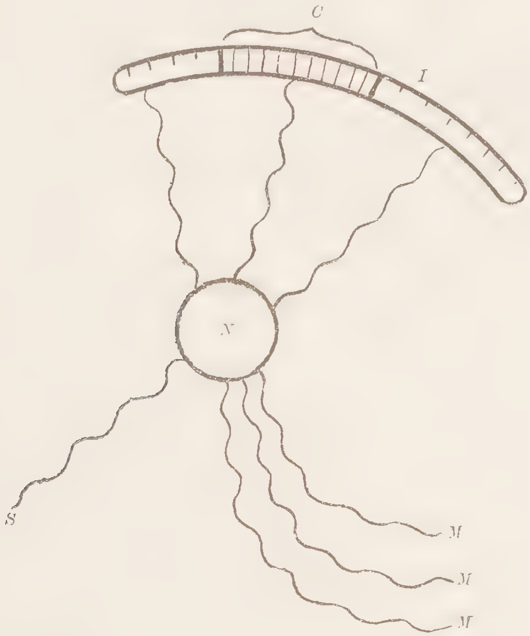
[2] Brain, April, 1886.

[3] Quoted by Huguenin, Ziemssen's Cyclopædia, vol. xii. p. 661.

The causes of infantile convulsions are legion; but, generally speaking, any disturbance of the nervous system, whether central or peripheral, organic or functional, may cause them. The loss of consciousness has been ascribed to the cerebral anæmia, but Brown-Séquard considers that this occurs too rapidly and is too complete to be due only or chiefly to a contraction of the blood-vessels of the cerebral lobes, but that it is due to an "inhibition of the activity of the cerebral nerve-cells."

Fig. 1 gives a diagrammatic representation of the method in which convulsions, irrespective of their exciting cause, may be produced.

FIG. 1.



DIAGRAMMATIC ILLUSTRATION OF CONVULSIONS. (Slightly modified from Dana, article "Convulsions," *Reference Hand-Book of the Medical Sciences.*)—*I*, inhibitory or controlling centres, including *C*; *C*, cortical sensory-motor centres; *N*, lower tier of cerebral centres; *S*, excito-reflex nerve; *M*, *M*, *M*, groups of muscles.

A convulsion may be caused by an over-excitation of *I*, *C*, *N*, or *S*, or by a withdrawal of the inhibitory action of *I*, including *C*. A functional *depression* of *C* may cause a *liberation* of the energy of its cells.

But little aid has been given to the elucidation of this portion of the subject by the ophthalmoscope, mainly because the examination is, during a paroxysm, a very difficult one, and opportunities rarely present themselves. In young children the difficulty is greatly increased, and records of the condition found are wanting.

The observations which have been recorded of the condition seen in epilepsy are at variance: theoretically, we should see an arterial anæmia of the retina during the initial stages; but one or two isolated examinations are not sufficient to base an opinion upon, as too many unrecorded elements enter into the varying condition of the retinal circulation to make them of value.

Allbutt[1] has observed pallor of the disks during a fit, and De Wecker[1] has described a sensible diminution in the size of the artery during the stage of pallor; but Kostl[1] and Niemetschek[1] thought that they recognized in one case a dilatation of the arteries during the attack. Gowers,[2] in a case of convulsions from meningeal hemorrhage, in which there was no pallor of the face, was able to keep a retinal artery and vein under view during the whole of a severe fit; the artery throughout presented not the slightest change in size, but the vein became large and dark during the stage of lividity.

François-Franck[3] states that, in epileptic attacks in animals produced by electrical excitation of the cerebral cortex, the ophthalmoscope showed an active congestion in place of an anæmia of the disks; at the same time the cerebral vessels, as seen by exposing the brain, became a lively red, and were never violaceous.

AGE.—Age must certainly be considered as by far the most important factor in the etiology of convulsions, it being a well-known fact that children under the age of two years are more frequently attacked than those beyond this age.

In the health reports of the city of Philadelphia for the ten years 1876–1885 inclusive, seven thousand five hundred and eight deaths among minors are stated to have been due to "convulsions" and "laryngismus stridulus." The following table of these cases shows the percentage of deaths that occur at various periods of life up to twenty years of age:

	UNDER 1.	1 TO 2.	2 TO 5.	5 TO 10.	10 TO 15.	15 TO 20.
Convulsions	4993	1335	893	178	38	26
Laryngismus stridulus . .	24	7	9	5	. .	. .
Totals	5017	1342	902	183	38	26

This shows conclusively that convulsions, whatever be their cause, are much more frequently seen during infancy than later in life, more than twice as many (66.8 per cent.) occurring during the first year as during the later period up to twenty years.

Tanner[4] gives the following figures on this point. In 1866, in England, deaths from convulsions were registered as 27,431, of which number 26,847 occurred in children under five years of age, 24,577 of these, or 89.5 per cent. of the whole number, being infants under one year of age.

Mackenzie[5] gives the following table of the deaths from laryngismus stridulus, taken from the Registrar-General's Reports, for children under ten years of age:

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[1] Quoted by W. R. Gowers, Medical Ophthalmoscopy, 1879, pp. 158 and 159.

[2] Loc. cit., and London Lancet, 1875, vol. ii. p. 655.

[3] Leçons sur les Fonctions motrices du Cerveau et sur l'Épilepsie cérébrale, Paris, 1887, p. 175.

[4] Practice of Medicine, 1872.

[5] Diseases of the Throat and Nose, London, 1880, vol. i. p. 480.

	UNDER 1 YEAR.	1 TO 2.	2 TO 3.	3 TO 4.	4 TO 5.	5 TO 10.	TOTALS.
Females	1487	691	152	94	60	63	2547
Males	2915	1395	213	97	63	88	4771
Totals	4402	2086	365	191	123	151	7318

The percentage of deaths for the different years in this table agrees very closely with the Philadelphia mortality from convulsions and laryngismus stridulus, given above.

Considerable allowance must be made for carelessness in making out the death-certificate, the convulsion being merely one symptom among many others, which frequently is present towards the close of a young child's illness, no matter what the disease may be; this symptom, and not the exciting cause, being returned as the cause of death. It is often to be looked upon "only as a mode of dying, and one incidental to the time of life," and as having nothing to do with the cause of death. Coutts[1] considers this the cause of the wide-spread opinion of the danger of infantile convulsions.

In many cases where the child dies in convulsions the original cause and not this symptom is returned on the death-certificate. The total number of children dying in convulsions, no matter what the exciting cause may be, is impossible to obtain.

Jamieson[2] gives the following table of three hundred and sixty-five deaths from convulsions:

Under one month	104	From one to two years	73
From one to three months	33	From two to three years	32
From three to six months	28	From three to four years	13
From six to twelve months	71	From four to five years	11

From this it appears that in his experience nearly half of the deaths during the first year of life from this cause occur during the *first month*. The majority of observers differ from Jamieson, and coincide in saying that convulsions are rare in the first month or two of life, except those occurring just after birth, which are frequently due to direct injury to the brain during labor. With the above exception, no one gives statistics as to the deaths during the *various months of the first year* of life. The Philadelphia Health Reports do not enable me to verify or disprove this statement.

From Jamieson's table there would appear to be two periods of frequency,—viz., the first month of life, and the period of dentition (six months to two years).

An explanation of the prevalence of convulsions during the earlier years of life must be looked for in the physiological differences which exist between the nervous systems of the infant, on the one hand, and of older children and adults, on the other.

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[1] Ninth International Medical Congress, Washington, 1887, vol. viii. p. 540.

[2] Australian Medical Journal, 1878.

Grancher[1] quotes the experiments of Simonoff, and of Fritsch and Hitzig, the former as proving that the anterior lobes of the cerebrum possess a controlling power over the lower centres, and the two latter as proving that the same lobes possess an excito-motor influence over them, so that the same portion of the brain is controlling or inhibitory, or excito-motor, according to circumstances.

In infancy the higher brain is imperfectly formed, while, on the contrary, the lower cerebral centres, including the medulla oblongata, are much more fully developed and in the ascendency, the higher cerebral system having not yet attained the controlling power which comes with later years, and, as Hughlings Jackson (*loc. cit.*) says, "the younger the infant, the more of a mere ordinary reflex mechanism will be what there is of its nervous system, and the less check there will be of one part by another." As a consequence, a comparatively trivial disturbance will interfere seriously with the unstable equilibrium existing between these two systems, and a convulsion or an irregular and uncontrolled outbreak of nervous energy is the result.

Every wave of nervous influence is not strictly confined to definite paths, but spreads by radiation, so to speak, upon the neighboring ganglia and overflows into neighboring channels, thus calling into action a larger area than otherwise would be the case. This has been brought forward as an explanation of the influence upon the knee-jerk that is produced by every sensation or voluntary movement, in the recent investigations by Mitchell and myself.[2] These experiments were repeated with identical results, but in a much more thorough manner, by Lombard.[3]

The explanation most generally received is that the afferent impression so occupies the higher centres that their controlling power over the lower centres in the brain and spinal cord is partially removed, allowing a greater response to irritation. In adult life this wave of nervous influence arising from reflex irritation also probably passes into the hemispheres,[4] the resulting movement being less on account of this drain; but in infant life the lower tier of centres is the only one that is functionally active, so that a greater movement results from a given irritation,—first, because there is less diversion of the overflowing force, and, secondly, because the controlling power over the lower centres has not been developed by the as yet undeveloped higher centres. On this theory there is an additional reason for the convulsive tendency seen in early childhood.

The nervous system of the child during the first two years of life is undergoing the most rapid development, and the centres are therefore much more impressionable to reflex action. Causes which in an adult will merely produce a chill may be sufficient to bring on a convulsion in an infant. Another condition which should be looked upon as a predisposing cause peculiar to infancy is the yielding nature of the brain-case, which naturally allows much greater changes to take place in the cerebral circulation, whether of anæmia or hyperæmia, than could possibly exist in older children and adults.

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[1] Gazette Médicale de Paris, January 21, 1888.

[2] Philadelphia Medical News, February 13 and 20, 1886.

[3] American Journal of Psychology, October, 1887.

[4] Wernicke, quoted by Nothnagel, Ziemssen's Cyclopædia, vol. xiv.

Jacobi[1] states that the peripheral nerves are relatively large and but little excitable during the first days of life, and that their excitability increases very fast towards the end of the first year, out of all proportion to the slower development of the inhibitory centres. This fact explains the great preponderance of convulsions during the first year of life.

SEX.—The majority of observers coincide in stating that the male sex is more predisposed to convulsions than the female. Gee[2] reports that in 48 cases of laryngismus, due to rickets, seen by him, 34 were males and 14 females, and that in 56 cases of convulsions in rickety children, 38 were males and 18 females; taking these cases together gives a percentage of 69.2 for the male and 30.7 for the female sex.

Mackenzie's mortality table for laryngismus stridulus (see p. 863) gives 4771 boys (65.1 per cent.) to 2547 girls (34.8 per cent.), a proportion of 100 to 53.39, which certainly seems to prove this point; but it would be interesting to know what proportion of births during that period belonged to the two sexes; a greater preponderance of male births would naturally increase the number of male deaths from whatever cause. Of the 7508 deaths occurring in Philadelphia from these two affections during 1876–1885, 3974 were males and 3534 females. During this period the male births numbered 103,413, and the female 94,048; it is therefore seen that the proportion of male to female children both in the mortality by convulsions (100 males to 88.92 females) and in the number of births (100 males to 90.94 females) is nearly the same, more male children dying convulsed, apparently because there are more male than female children to die: this conclusion would seem natural, as during the early years of life there is but little if any physiological difference between the sexes, so that much difference in the tendency to disease should not be expected.

Meigs and Pepper[3] in 92 cases found a proportion of 47 boys to 45 girls, but Steffen[4] reports 544 cases with a proportion of 386 boys to 158 girls.

HEREDITARY INFLUENCES.—A child cannot inherit convulsions, or, in other words, a symptom, but it may inherit such a condition of the nervous system as to predispose to them. It is a well-known fact that in the children of some families the least exciting cause will be sufficient to induce convulsions, while in others the same source of irritation, although carried to a much greater degree, will not suffice to produce them. This can be explained only upon the supposition that the children are born with a *convulsive tendency.* Occasionally only certain children of a family will show this tendency, which may even be confined to those of one sex.

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[1] A. Jacobi, Introductory to Keating's Cyclopædia of the Diseases of Children, vol. i. p. 3; also Intestinal Diseases of Infancy and Childhood.

[2] St. Bartholomew's Hospital Reports, 1867, vol. iii.

[3] Diseases of Children, Philadelphia, 1874.

[4] Ziemssen's Cyclopædia, vol. vii. p. 995.

Harris[1] reports 38 cases of eclampsia, 37 of which occurred in thirteen families in which collectively there were 55 children; these persons were the descendants of the first, second, and third generations of two pairs of ancestors; the second generation consists of 31 members, 20 of whom have had convulsions.

Coutts (*loc. cit.*) has found in 100 cases of convulsions in infants 29 in which there was a history of neuroses in parents or near relatives, and, including the convulsions occurring in brothers and sisters, the family neurotic percentage rose to 63.

T. Lauder Brunton[2] considers that the children of drunkards are liable to have convulsions in their earlier days, and quotes Lancereaux as stating that drunkards transmit to their offspring, among other morbid conditions, an abnormal reflex excitability and a tendency to convulsions.

Wood[3] is of the opinion that the tendency to convulsions in the child is closely connected with the epileptic diathesis, and that in those cases in which the epileptic tendency exists injudicious hygienic treatment or trivial causes may be sufficient to produce convulsions.

Some authors believe that puerperal convulsions in the mother predispose the child to convulsions.

It has been doubted whether a healthy child can have convulsions solely from reflex irritation, but there are some children in whom the most careful investigation fails to discover any cause in the nervous system or elsewhere for the attacks, and we are forced to fall back upon the supposition of a "convulsive tendency," possibly inherited, as an explanation. Gee (*loc. cit.*) found 56 cases of convulsions in children, out of a total of 102, in which he could not account for the seizures except upon the supposition that they were due to the state of health of the children at the time of the attack.

RACHITIS.—It is generally admitted that rachitis renders a child liable to suffer from convulsions, especially that form called laryngismus stridulus ("inward convulsions," or "holding-breath spells"). The hereditary influences tending in this direction must also be looked upon as positive predisposing causes. Out of 50 cases of laryngismus stridulus seen by Gee (*loc. cit.*) 48 were rickety, and of these 19 had general convulsions; and out of

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[1] American Journal of Obstetrics, 1869–70, vol. ii.

[2] The Book of Health, edited by Malcolm Morris, London, Paris, and New York, 1883, p. 234.

[3] Nervous Diseases and their Diagnosis, 1888.

102 children in whom general convulsions occurred 46 were rickety, or 45 per cent.

Rickets is a much more prevalent affection than is usually considered, in the higher as well as in the lower walks of life, although more frequently seen in the latter.

Gee found that 30.3 per cent. of sick children under the age of two years were rickety, and quotes Ritter as having had a like experience at the Medical Policlinic at Prague,—viz., 31 per cent. of children under five years of age. Parry[1] found in Philadelphia 28 per cent. of all children under five years of age showing signs of rachitis. In Berlin at the University Policlinic[2] the percentage under this age has been found to be 13. Jamieson (*loc. cit.*) states that in his experience this connection between rickets and convulsions has not been observed; the former being said to be a rare affection at Melbourne, Australia, and vicinity, while the latter are far from uncommon.

My own experience at the dispensary of the Children's Hospital leads me to place the percentage higher than any of the figures quoted, rachitis being diagnosticated when the children presented beading of the ribs, markedly-delayed dentition, profuse sweating of the head, abnormally-open fontanels, or enlargement of the radial and tibial epiphyses, the relative frequency of these symptoms occurring about in the order named, the beading of the ribs being decidedly most often encountered. No case older than five years was included in the study. The figures are as follows:

Total number of cases five years old and under, 360; number of cases two years old and under, 263; number of males, 191; of females, 169.

Rachitic cases five years old and under, 137,—38.05 per cent.; rachitic cases two years old and under, 110,—41.82 per cent.

A history as to the presence or absence of former convulsions was obtained in 236, and of these 90 were rachitic,—38.13 per cent.; 172 of these 236 were two years old and under, and 72 of these were rachitic,—44.44 per cent.

Convulsions occurred in 20 of the 236 cases,—8.47 per cent. One of these was an epileptic, and three died in convulsions occurring towards the close of serious illnesses, the others being the so-called dentition convulsions and those due to errors in diet.

Of the 20 cases in which convulsions had occurred, 8 were rachitic—40 per cent.—and 12 non-rachitic,—60 per cent. Of the 90 rachitic cases, 8 had had convulsions,—8.88 per cent.

It must be borne in mind that all these statistics refer to *sick* children of the humbler classes, and therefore do not accurately represent the prevalence of rachitis among the children of the community at large.

Rickets usually appears from the sixth to the thirteenth month, although it may appear at any time and may even be present at birth, so that this condition cannot be considered as the principal predisposing cause of convulsions occurring in the first few months of life. Changes in the cerebral vascularization occur in rachitis, with the consequent nutritive changes in the brain; and this, together with the presence of deformity of the ribs, causing interference with the respiration and deficient oxygenation of the blood, and the craniotabes and the softness of the bones of the head (possibly allowing pressure to be made upon the cranial contents when the child is recumbent), must be looked upon as the explanation of the association of these two conditions. Jacobi[1] considers that every case of laryngismus stridulus, without exception, that occurs during the first eight or nine months of life is due to the presence of craniotabes. This statement does not meet with universal acceptance, as this symptom frequently occurs in rachitic children where craniotabes cannot be detected. "Out of ninety-six cases of laryngismus examined by Lederer[2] there was craniotabes in ninety-two." Pressure with the fingers upon these spots has been reported to cause an attack, an experience which has recently happened to the writer.

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[1] American Journal of the Medical Sciences, January and April, 1872.

[2] Senator, Ziemssen's Cyclopædia, vol. xvi.

Elsässer[3] was the first to promulgate this theory of the attack being due to pressure upon the softened occiput, but Mackenzie[4] and Steffen[5] consider the theory entirely exploded. The latter, while admitting that when craniotabes is present spasm of the glottis may be expected, is of the opinion that this in no way depends upon it and does not necessarily follow, as the child is attacked not only while lying down, but also when sitting up. Mackenzie believes the connection between rachitis and laryngismus to be due to the malnutrition which is present. Senator[6] states that "the laryngeal spasm is often observed without there being any marked softening of the occipital bone; indeed, it is a far more frequent complication of rickets than craniotabes itself." In this connection it may be mentioned that some authors hold that craniotabes is due to a syphilitic taint and not to rickets: thus, Lees and Barlow[7] consider that the existence of marked craniotabes must be regarded as strong evidence for suspecting syphilis, forty-seven per cent. of the total number affected with craniotabes being, in their opinion, almost certainly syphilitic. Eustace Smith[8] also believes this condition is most common in cases where there is a distinct syphilitic taint. Hughlings Jackson (*loc. cit.*) states that the changes in the chest-walls which obtain in rachitis, and the consequent imperfect respiration, cause a deficient aeration of the blood, and that this, acting on the respiratory centre, gives rise to an attack of laryngeal spasm.

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[1] Pepper's System of Medicine, vol. ii.

[2] Quoted by Mackenzie, Reynolds's System of Medicine, vol. iii. p. 449.

[3] Der weiche Hinterkopf, 1843, p. 161.

[4] Manual of the Diseases of the Throat and Nose, vol. i.

[5] Ziemssen's Cyclopædia, Amer. ed., vol. vii. p. 1000.

[6] Ibid., vol. xvi. p. 199.

[7] "Relationship of Craniotabes to Rickets and Congenital Syphilis," Transactions of the Pathological Society of London, 1881, vol. xxxii. p. 327 *et seq.*

[8] A Practical Treatise on Disease in Children, London, 1884, p. 208.

These attacks usually occur at night, when the circulation of the brain is at its minimum, so that the influence of the condition just mentioned has a greater effect. Imperfect respiration is thought by this authority to be the ordinary ultimate cause of convulsions in infancy.

Enlarged bronchial, cervical, and tracheal glands frequently occur in rachitis and in scrofula, and the pressure exerted by these upon the trachea has been thought by some to be the cause of laryngismus; others have believed that the same condition produces pressure on the nerves and thus excites spasm of the glottis. Morrill[1] reports a case undoubtedly due to this cause in a scrofulous child, and Barèty[2] reports twenty cases due in his opinion to enlargement of the mediastinal glands. These may be considered as possible causes if it is true, as Semon and Horsley[3] state, that the abductors of the vocal cords die before the adductors, and that the former are more prone to disease; constant pressure on the recurrent laryngeal nerves by acting in this manner might, it seems, cause a spasm of the glottis by giving that structure over to the adductors. Hooper[4] strongly opposes this theory, while Donaldson[5] believes the abductors to be the more irritable; constant pressure upon the nerves causing them to fail in the performance of their function before the adductors. Lucas[6] considers that laryngismus stridulus occurring in rickets is due to irritation of this nerve from pressure by enlargement of the extremities of the tracheal rings, or to irritation by pressure as the nerve passes under the corner of the thyroid cartilage; but this theory lacks confirmation. Kopp's theory (thymic asthma) that the affection is caused by the pressure on the trachea or nerves by an enlarged thymus gland, may be mentioned in this connection. This theory has been entirely exploded, for this condition is rarely present, and, if it is, does not necessarily cause pressure as alleged.

DISORDERS OF THE DIGESTIVE SYSTEM, AND THE EFFECT OF WEATHER.—**Dentition.**—A fact that seems to be firmly established in the mind of the public, as well as in the minds of many of the medical profession, is that dentition plays the most important *rôle* in the etiology of convulsions. If the child happens to be cutting teeth at the time of the attack, no further cause of trouble is looked for, and if there is no visible irritation of the gums it is still the advancing tooth that is thought to be the cause of the attack, the true exciting cause being thus frequently overlooked. I do not wish to be understood to say that painful dentition may not cause convulsions,—in fact, I believe that such is occasionally the case, particularly when the child has a predisposition in that direction,—but the delayed and irregular dentition is more often but another sign of the rachitical tendency, previously discussed, and this, and not the dentition, is more likely to be the predisposing cause, although an inflamed and tender gum may precipitate a paroxysm. An otitis may be caused by the inflamed condition of the gum and be the starting-point of an attack.

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[1] Boston Medical and Surgical Journal, February 24, 1881.

[2] Gaz. Hebdom. de Méd. et Chirurg., 1881, pp. 35 and 85.

[3] British Medical Journal, August 29, 1886.

[4] New York Medical Journal, July 4, 1885, p. 2.

[5] American Journal of the Medical Sciences, July, 1886, p. 93 *et seq.*

[6] Discussion on Rickets, Transactions of the Pathological Society of London, 1881, vol. xxxii. p. 358.

The time of dentition, which begins about the eighth month, is a period of great functional activity, for, besides the eruption of the teeth, the follicular apparatus of the intestines is undergoing active development in order to prepare the alimentary system for a radical change of diet. As a consequence, gastro-intestinal disorders and nervous symptoms are frequent at this time of life. It is difficult to conceive how a purely physiological process, such as dentition is, can in a healthy child be the sole cause of convulsions and death, but the irritation and pain which accompany the process in some cases may be sufficient to turn the scale in favor of a nervous outbreak.

The period of dentition thus resembles that of puberty in being a period of frequent nervous manifestations.

Sejournet[1] considers the *rôle* played by dentition as of great importance in the production of an attack, and in his opinion stout children are more liable to dentition convulsions than others. Soltmann,[2] basing his conclusion on experiments made on young dogs, considers by inference that the excitability of the sensitive nerves in new-born children is much less than in adults, that there is a period extending from the fifth to the eleventh month, or about the time of dentition, in which there is an increased excitability of these nerves, and concludes that "teething" has a direct influence in causing reflex spasms and convulsions. Jacobi's opinion as regards the excitability of the peripheral nerves in young children, as mentioned on page 865, is of interest in this connection.

More than double the number of deaths from convulsions in children occur during the first year of life, but according to Jamieson's tables (p. 863) over two-thirds of the convulsions during the first year occur during the first six months, so that dentition cannot of necessity be taken as a cause of convulsions in these cases.

Improper Feeding and Indigestion.—The presence of undigested and undigestible food in the stomach and intestines is a very fertile cause—perhaps the most fertile of any one class of causes—of convulsions. Over-feeding even with proper food becomes thus a very important factor; and this statement gains strength when at autopsies a very full stomach is frequently all that is found to account for the death in convulsions of an otherwise apparently healthy infant. Constipation, particularly where large quantities of scybala are impacted in the intestines, is also an exciting cause that should always be borne in mind. Simon[1] considers that eighty per cent. of convulsions in children arise from digestive troubles.

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[1] Revue Mensuelle des Maladies de l'Enfance, iii., Paris, 1885.

[2] Jahrbuch f. Kinderheilkunde, B. xiv. H. 4.

This condition seems capable of producing an attack in an otherwise healthy child, although some authors consider this unlikely. If the "convulsive tendency" exists, the effect is much more pronounced. Temporary indigestion becomes thus an exciting cause, and it is rather curious that there need be no marked evidence of flatulency or pain previous to the nervous outbreak.

A fit of anger or any other violent emotion on the part of the mother or nurse may so alter the composition of her milk as to cause convulsions in a predisposed nursling, probably by rendering it a source of irritation to the child's stomach.

Gastro-Intestinal Disorders.—These disorders, so frequently met with in the hot weather, also produce a profound impression on the child's nervous system, and cause convulsions in two ways: first, by the sudden diarrhœa and vomiting which frequently ensue, and, secondly, by rendering the gastro-intestinal tract unsuited to receive the food which before the attack may have been properly proportioned to the child's age, but which now acts as a direct irritant to the stomach, and is ejected, or passed into the bowels unprepared for intestinal digestion. The diarrhœa and vomiting, by increasing the specific gravity of the blood, may act by rendering the latter too dense to circulate properly, thus starving the brain, and may also, as mentioned by Novi,[2] by depriving the gray matter of the brain of much of its moisture, seriously lower its vitality, convulsions ensuing as a consequence. Intussusception is an occasional cause of convulsions in young children.

In order to discover what relation existed between the number of deaths from convulsions and those from disorders of the alimentary tract, I collected from the health reports of Philadelphia for the years 1875–1886 inclusive all the deaths among minors from "teething," "sore mouth," "cholera morbus," "cholera infantum," "diarrhœa," "dysentery," and "ulceration of the stomach and bowels," and, taking these together, arranged them by months and compared them with the deaths from "convulsions" and "laryngismus stridulus," similarly arranged.

The hot months of summer, as every one knows, cause the greatest mortality among children; 10,631 deaths occurring from the former class of diseases during the months of June, July, August, and September, out of a total of 13,115 for the ten years in question, July with the highest average temperature having the greatest number of deaths,—viz., 5133; this month also showing the greatest mortality from "convulsions,"—viz., 988 out of a total of 7508. Arranging these figures in the form of a table, comparisons are much more easily made.

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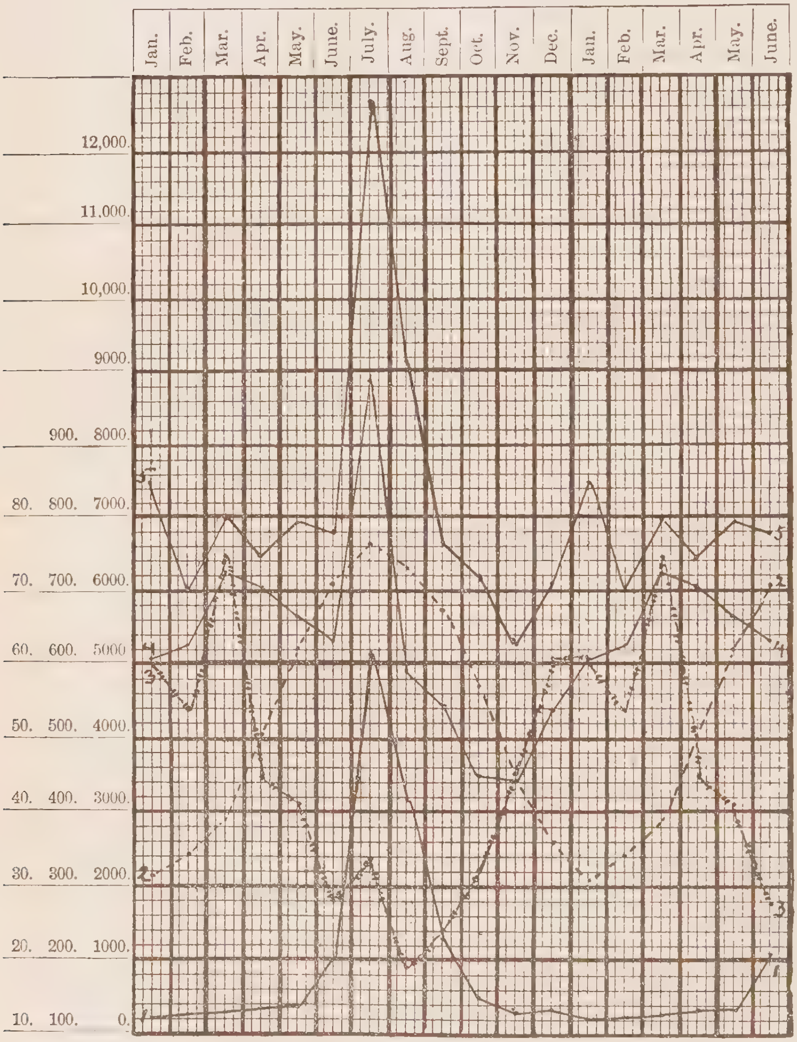
[1] L'Union Médicale du Canada, August, 1887.

[2] Lo Sperimentale, quoted by New York Dietetic Gazette, July, 1888.

Tracing No. 1 represents the deaths from gastro-intestinal disorders, tracing No. 4 the mortality from convulsions, and tracing No. 2 the mean temperature, per month. It will be seen at a glance that an average monthly temperature of 66° F. or over causes a fearful increase in the mor-

Fig. 2.

1876–1885.



Tracing 1.—13,115 deaths from gastro-intestinal diseases. Read by using column of figures 0 to 12,000.

Tracing 2.—Mean temperature. Read by using column of figures 10 to 80.

Tracing 3.—"Storm-centres" passing within 400 miles of Philadelphia. Read by using column of figures 10 to 80.

Tracing 4.—7508 deaths from convulsions and laryngismus stridulus. Read by using column of figures 100 to 900.

Tracing 5.—Total deaths from all causes in children under 10 years of age,—viz., 86,769. Read by using column of figures 0 to 12,000.

tality from gastro-intestinal affections, and that the highest mean temperature coincides with the greatest number of deaths from convulsions. Several causes may act together to produce this July rise in the latter tracing: 1st, convulsions must in many of these cases be looked upon merely as a mode of dying, and not necessarily as a cause of death *per se;* 2d, the large number of gastro-intestinal disorders occurring in that month would of necessity increase the number of deaths from convulsions, both in consequence of the intestinal irritation and on account of the great drain on the fluids of the body causing deficient cerebral blood-supply; 3d, high temperature is in itself an important factor in the causation of convulsions.

The convulsion tracing is at its lowest point in November, and from this month there is a gradual rise until March is reached, then there is a fall until June, and then a sudden rise in July to the highest point, after which there is a steady fall until the low point in November is returned to; this cannot be entirely explained by either of the three factors above mentioned, although the general mortality tracing (No. 5) does bear a general resemblance to it, as would be expected on account of the first reason given above. The gastro-intestinal tracing is almost at its lowest point, and an equal or even higher average temperature in August, September, and October is not accompanied by a corresponding rise in the convulsion tracing. Before attempting an explanation an apparent digression will be made. Gee[1] calls attention to the influence of the season upon the number of attacks of laryngismus stridulus, and states that these are apt to occur more frequently when the children are confined to the house and obliged in consequence to breathe impure air, which has the effect of producing an "erethism of the nervous system which shows itself in turn as a spasmodic diathesis," which, however, in his opinion, does not produce the excessive irritability until it has been prolonged for some time, so that the spring months contain the greatest proportion of the cases; out of 63 attacks seen by him, occurring in the years 1866, 1867, and 1868, 47 occurred in the months of February, March, April, and May. Barlow,[2] on the contrary, states that convulsions, and particularly laryngismus stridulus, occur most frequently in cold weather, 70 out of the 114 cases investigated by him in 1885 occurring during the months of October, November, December, January, and February (61.40 per cent.), and being quite evenly distributed among them, February having the greatest number (16) and December the least (13); the months of May, June, and July show but 10 cases (8.77 per cent.), March 7, and the remaining months 9 each. The results obtained by the study of these two observers, the former in London and the latter in Manchester, although these places are comparatively near each other, differ widely, which should not be the case had the true cause been found.

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[1] St. Bartholomew's Hospital Reports, vol. ii.

[2] British Medical Journal, June 18, 1887.

Henoch[1] has found the greatest number of cases of laryngismus stridulus, in hospital as well as private practice, during the months of January to May, inclusive, and in this he agrees with Gee.

J. Crichton Browne[2] states that convulsions, from which so many infants and children perish, have their minimum death-rate in September and October, their maximum in February and March.

The effect of meteorological changes on nervous diseases has been recognized for some time; this is particularly noticeable in neuralgia[3] and in chorea,[4] the former reported by Mitchell and the latter by the author,—the perturbing influence appearing to be the complex meteorological disturbance known as a "storm." Tracing No. 3 represents the number of storm-centres passing within a circle of four hundred miles' radius drawn around Philadelphia for the ten years in question, this radius being chosen as it was found that the storms passing within that distance produced the greatest effect on chorea, in former studies. The greatest number of storms occurs in March (see table), which coincides with the spring rise in the convulsion tracing: so that it seems possible that the weather may exert more than a passing influence.

The dissimilarity between the two tracings 3 and 4 in midsummer is produced by the heat, the great number of gastro-intestinal cases increasing the deaths from convulsions, as previously stated. No other meteorological factor,—viz., temperature, barometer, mean relative humidity, range of thermometer, or number of cloudy days,—either alone or in combination, seems to account for the March rise of the convulsion tracing. The influence exerted by storms may be indirect and not direct in its action, by causing an increase of other affections in which convulsions close the scene. This study should be applied to the number of attacks, or cases, of convulsions, and not to the number of deaths, to ascertain its true value. As tending to uphold the probability of this theory, it is worth mentioning that in the studies just mentioned, the greatest amount of pain and the highest number of attacks of chorea occur in March, corresponding to the spring rise of the convulsion tracing, and the lowest number of attacks of chorea in October and November, corresponding to the lowest point reached by the convulsion tracing, and, while these months do not show the least amount of neuralgia (June), they are at least months of less pain than those of winter and spring.

Intestinal Parasites.—The effect produced by the presence of worms in the intestinal canal is as much exaggerated by the public generally as is the effect of dentition. Worms are present in many children, particularly those of the poorer classes, and frequently cause no symptoms whatever, and are not suspected until they are seen in the evacuations. When, however, worms are present in large quantities they undoubtedly may act very injuriously, and may even be the starting-point of nervous symptoms or even of convulsions. Most of the symptoms are due to the condition of the gastro-intestinal canal, that favors the development of the parasites. Worms are not so frequently found in the intestines of infants as in those of children about two years of age and over. The tape-worm is very rarely seen in young children. Convulsions produced by these causes are purely reflex.

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[1] Lectures on Children's Diseases, New Sydenham Society, London, 1889, vol. i. p. 185.

[2] The Book of Health, edited by Malcolm Morris, London, Paris, and New York, 1883.

[3] Captain Catlin's Investigations, American Journal of the Medical Sciences, April, 1887, and Transactions of the College of Physicians of Philadelphia, vol. vi. Two papers.

[4] Seasonal Relations of Chorea and Rheumatism, Medical News, Philadelphia, November 13, 1886.

ACUTE INFECTIOUS FEVERS.—In the acute infectious diseases convulsions are frequently met with, usually either at the onset of the attack or towards the close of the disease. In the former case they take the place of the rigor seen at the onset of such diseases in later life; they are to be considered as toxæmic convulsions due to the impregnation of the blood with the specific poison, which acts by producing an inhibition of the controlling action of the higher cerebral centres, possibly acting through the medulla by causing a spasm of the arterioles. The suddenness of its onset is to be explained in the same way that is employed to explain a chill or a sudden fever,—viz., a stimulation or depression, as the case may be, of the inhibitory chemical centre by the specific poison.[1] According to Radcliffe,[2] these convulsions occur in the cold stage of the fever, when the circulation is greatly wanting in power, and they are confined to this stage except when there happen to be brain- or kidney-complications. This last statement cannot be accepted without reservation, as convulsions are known to occur in these diseases when the fever is high.[3] Holmes considers the convulsion seen in the beginning of these diseases as entirely due to the high temperature, and that a temperature of 103° F. in a child is very liable to excite an attack, which attack is most frequently seen in the first few hours of the pyrexial seizure. Maclagan[4] is of the opinion that anæmia of the brain is the cause of the headache which is most frequent in the early stage of acute fevers in adults, and of the convulsions seen in a similar period in the fevers of childhood, the anæmia resulting from the contraction of the minute arteries of the brain.

Welch[5] considers that the temperature regulation is more labile in infants than in adults, and that human beings may tolerate temperatures of 107° F. or even higher for a considerable time. He also states that temperatures ranked as high do not in themselves, independently of other factors, necessarily exert any such injurious influences as have been usually attributed to them,—that unless the temperature is suddenly raised no disturbance of the cerebral functions is noticed until the temperature reaches a certain point, beyond which death ensues, preceded by convulsions and coma; the disturbances of the sensorium depend in a higher degree upon infection or intoxication than upon heightened temperature.

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[1] H. C. Wood, Toner Lecture, No. 4, 1875.

[2] The Dynamics of Nerve and Muscle, London, 1871.

[3] Transactions of the International Medical Congress, Philadelphia, 1876, p. 796.

[4] Fever: a Clinical Study, by T. J. Maclagan, London, 1888, p. 965.

[5] The Cartwright Lectures, On the General Pathology of Fever, Philadelphia Medical News, April 7 and 14, and May 19 and 26, 1888.

Wood[1] has shown that heating the brain of a mammal produces sudden insensibility, with or without convulsions, at a temperature of about 108° F., and that this effect is due to the direct action of the heat.

When convulsions occur in the later stages of the diseases in question, they are to be explained by the presence of serious cerebral, respiratory, or urinary complications.

INTERMITTENT FEVER.—In children convulsions are known to occupy the place of the chill met with in later life in intermittent fever. A true chill is rarely seen in an infant or a young child, the convulsions occurring on successive days just before the appearance of the fever, proving their origin. An editorial in the *North Carolina Medical Journal,* 1882, vol. x., states that there is no more fatal disease among young children in malarial districts of the South than malarial eclampsia. The statement is also made that September and October are the months in which this complication is most generally met with, and usually between 11 A.M. and 5 P.M., the season of the year and the time of day when the ordinary paroxysm of malarial fever is most frequently encountered. Such frequency of malarial eclampsia is not met with in Philadelphia; at least it has not been my experience during the last ten years at the dispensary of the Children's Hospital, where large numbers of cases of intermittent fever are seen every spring and autumn. Meigs and Pepper (*loc. cit.*) and J. Lewis Smith[2] consider it not a very rare thing for the fever to be ushered in by a convulsion. In the pernicious form of intermittent fever this symptom is frequently seen.

TYPHOID FEVER.—Convulsions are of rare occurrence in this disease in children; they are to be considered as of the same nature as those occurring in scarlet fever, measles, etc.

PNEUMONIA.—Convulsions are a rather uncommon symptom in pneumonia, either of the croupous or the catarrhal variety, and they are more frequently seen in very young than in older children. The opinion generally held is that convulsions are most apt to complicate pneumonia when the disease is situated at one or other of the apices of the lung: thus, Juergensen[3] states that the most severe cerebral symptoms usually occur in connection with pneumonia of the apex, setting in with a rapidly-developed fever, and quotes Heinze as holding the same opinion. Among others holding these views strongly may be mentioned Eustace Smith,[4] Goodhart,[5]

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[1] A Study of Morbid and Normal Physiology, Smithsonian Contributions to Knowledge, 1880; also, Pepper's System of Medicine, art. "Thermic Fever," vol. v. p. 394.

[2] Diseases of Children, 4th ed., p. 285.

[3] Ziemssen's Cyclopædia, vol. v. p. 102.

[4] Clinical Studies of Disease in Children, Philadelphia, 1876, p. 49.

[5] Diseases of Children, edited by Starr, Philadelphia, 1885, p. 344.

Loomis,[1] Hanot,[2] and Damaschino.[3] Holt,[4] on the contrary, after a study of one hundred and seventy-three cases of cerebral symptoms in this disease, holds that they are not intimately associated with apex disease, such association occurring in only one-fifth of apex cases, but that they occur more frequently when the disease is extensive and the temperature high (one in three of the cases). He considers that convulsions in this disease belong almost without exception to infancy, being rarely met with after two years of age, the time of onset of the disease being the period when they are usually seen. Coutts (*loc. cit.*), quoting from the practice of Eustace Smith in the East London Children's Hospital, states that convulsions occur at the onset of pneumonia in about five per cent. of the cases (two in forty-one). Eustace Smith (*loc. cit.*) does not believe that there is any relation between apex pneumonia and convulsions, pneumonia of the apex being especially short and favorable, in his opinion.

TOXÆMIC CONVULSIONS.—Under this head should be included those convulsions occurring in uræmic conditions, as well as those which follow the administration of poisons. Eustace Smith considers infants to be very susceptible to the influence of lead, and he has several times seen convulsions follow the internal administration of this drug, and apparently directly due to it.[5] Convulsions occur in uræmia when there is no elevation of temperature: so that there cannot be much doubt that they are due to the action on the nervous centres of the morbid materials circulating in the blood. In the acute infectious fevers we have, as previously mentioned, besides the impregnation of the system with the special poison, the secondary very important factor of fever, which is produced by the action of this poison on the heat-centres: so that two important causes of convulsions may thus be acting together.

Some of the convulsions occurring in gastro-intestinal disorders may properly be placed in this class, as they are possibly caused by the poisonous influence of ptomaines developed in the stomach and intestines, as suggested by Senator.[6]

WHOOPING-COUGH.—Meigs and Pepper mention convulsions as a complication of this disease in 12 out of 208 of their cases (5.76 per cent.), and quote Rilliet and Barthez as having seen it in 5 out of 29 cases (17.24 per cent.). Roger[7] has met with this complication in hospital practice only 15 times in 431 cases (3.48 per cent.), and in his experience it is most frequently seen in infants still at the breast, or at least before the third year of life. Convulsions never usher in this disease, but either occur in the course of a marked attack or precipitate the fatal termination in complicated cases, which complication is usually broncho-pneumonia. Their cause is threefold:

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[1] Pepper's System of Medicine, vol. iii. p. 329.

[2] Du Traitement de la Pneumonie aiguë, Paris, 1880, p. 56.

[3] La Pneumonie aiguë chez les Enfants, Paris, 1867, p. 63.

[4] Medical Record, April 7, 1888.

[5] On Disease in Children, 1884, p. 279.

[6] Quoted by Henoch, loc. cit.; also Berlin. Klin. Wochenschr., 1868, No. 24, and Zeitschrift f. Klin. Med., Bd. vii. H. 3.

[7] De la Coqueluche, vol. ii. p. 582.

1st. The nervous element which is an integral part of whooping-cough.

2d. The high fever which often accompanies severe cases.

3d. The venous condition of the cerebral circulation due to the severe paroxysms of coughing or to the pulmonary complication, or to both. Serous effusion into the ventricles should be mentioned here as a consequence of the venosity and a cause of the convulsions.

MALFORMATIONS AND DISEASES OF THE HEART AND GREAT VESSELS.—From what has been said in the foregoing pages of the important *rôle* played by disturbed circulation in the etiology of convulsions in children, it is evident that they would frequently be found in association with malformations and diseases of the heart and great vessels, particularly in the condition known as morbus cœruleus.

IRRITATION OF PERIPHERAL NERVES.—A burn or scald produces a much more profound impression upon a child than upon an adult, and convulsions are often thus caused. There is great shock and a profound impression on the circulation, the arteries contain but little blood, and all the internal veins are engorged, particularly those of the abdomen, as proved by frequent autopsies. Although the condition is generally described as one of active congestion of all the internal organs, it is more truly a secondary venous congestion. When convulsions occur in this condition they are usually preceded by symptoms of profound prostration. Irritation of the skin in eczematous and other eruptions has been reported as a cause of convulsions in children, as has the irritation caused by pins. Tickling has also been reported as a cause. Convulsions due to the stings and bites of insects must be of rare occurrence in this country at least, although they are reported in warm climates.

Earache as a cause of convulsions should always be borne in mind, and this may occur entirely independently of any meningitic trouble, and be due simply to the pain. Huguenin[1] makes the statement that "changes in the calibre of cerebral arteries occur in irritation of even a distant sensitive nerve," and Nothnagel[2] holds the same view. Sudden and violent pain is known to cause marked changes in the cerebral circulation, and fainting is a very frequent sequence; convulsions have been known to follow a faint produced in this way. Huguenin (*loc. cit.*) reports a case following a quickly-performed circumcision, and a similar case occurred to the writer when an interne at the Pennsylvania Hospital,—viz., on removing a small piece of bone from a crushed finger, the patient cried out with pain, fell to the floor in a faint, and immediately became generally and very severely convulsed; on recovering consciousness he stated that he had always enjoyed good health and had never had a previous convulsion.

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[1] Ziemssen's Cyclopædia, vol. xii. p. 434.

[2] Ibid., vol. xii. p. 13; also, Virchow's Archiv, Bd. xl.

Brown-Séquard's guinea-pigs may be considered as experimental examples of peripheral irritation producing convulsions in predisposed animals. Convulsions frequently take their rise from the irritation produced by the presence of old cicatrices, on the head or elsewhere; but this would be considered more properly under the head of epilepsy, and is mentioned only to impress upon the reader the effect of peripheral irritation. Genital irritation should also be mentioned here as a cause of convulsions. Fissure of the anus has been reported as a cause of convulsions in nurslings.[1]

FRIGHT AND OTHER STRONG EMOTIONS.—Angel Money[2] considers fright and terror as a kind of cortical epilepsy, attended with violent discharge of energy and accompanied by great derangement of intellect or even total loss of consciousness.

Fright has frequently been known to cause convulsions, and the effect on a "nervous child" is more pronounced than on one not so constituted. There is produced a sudden and profound alteration in the peripheral circulation, as is evident to all in the sudden blanching of the face, and the convulsions which sometimes follow immediately upon a fright are due to this vaso-motor spasm. Dickson[3] thinks that there must exist a predisposing cause before fright can act in this way. Niemeyer[4] considers these convulsions to proceed from "excitement of the cerebral ganglia transmitted thence to the medulla oblongata." A fit of crying or of anger has occasionally been known to end in a convulsion; but here in addition to the emotional factor there is a marked interference with the respiratory function. Other violent emotions may act in the same way.

LARYNGEAL CAUSES.—Any irritation or inflammation of the larynx, such as catarrh, or even the trickling of saliva or milk into the larynx or the drying of mucus upon the glottis during sleep, may be the starting-point of a laryngeal spasm which may end in a general convulsion. Crying or sobbing may also act by causing spasm of the glottis, and both Mackenzie and Cohen[5] speak of the catching of the breath produced by tossing a child into the air in play as a cause. The sleeping state seems to predispose to an attack.

Mantle[6] reports a case of severe laryngeal spasms, followed by general convulsions, which were due to the irritation produced by an elongated uvula, finally cured by excision of this part. He advises an examination of the throat in all cases as a matter of routine.

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[1] Maurice Raynaud, Gaz. Hebdom. de Méd. et Chir., 1881, p. 239.

[2] Diseases of Children, 1887.

[3] Dynamics of Epilepsy and Convulsions, Guy's Hospital Reports, 3d ser., 1873, vol. xviii. p. 181.

[4] Practical Medicine, revised ed., vol. ii. p. 370.

[5] Diseases of the Throat and Nasal Passages, New York, 1879, 2d ed., p. 629.

[6] British Medical Journal, February 8, 1890.

Diseases of the Brain and Spinal Cord.—Convulsions due to the various causes previously enumerated do not depend upon any gross lesion of the brain or spinal cord; there remains a division of convulsions which have such conditions as an exciting cause,—viz., poliomyelitis anterior acuta, and other acute spinal affections; tumors of the brain; the various kinds of meningitis; cerebral hemorrhage; embolism and thrombosis; injuries to the head and brain from accident or during difficult labor; concussion of the brain and cord, etc.; and the reader is referred to the appropriate sections in this work for further information upon these subjects. A few words in regard to the general mechanism by which convulsions are produced in these affections will not be out of place in this connection. Mary P. Jacobi[1] states that nearly fifteen per cent. of the cases of poliomyelitis anterior acuta have convulsions as a prodromal symptom, and Erb[2] considers that the pathogenic explanation of these initial brain-symptoms has not yet been found; but if a peripheral irritation can so affect the nerve-centres as to induce convulsions, it certainly seems probable that such a sudden impression upon the spinal cord as occurs in this affection would be sufficient to do so. J. Hughlings Jackson[3] brings forward the hypothesis that these initial convulsions may be due to the action of ptomaines the result of disintegration of the nervous matter of the anterior horns on the "ponto-bulbar" centres (lowest tier). It is probable that the convulsions in the various brain-disorders are due, not to an arterial congestion, as is frequently thought, but to an inhibition of the controlling power of the cerebrum over the lower ganglia, produced by anæmia due to vaso-motor spasm or to intracranial pressure causing impaired nutrition by interference with the blood-supply. An unstable condition of the higher centres may also exist, causing explosive development, or rather liberation, of nervous force in the gray matter of the motor areas. Intracranial pressure in young infants is made manifest by the bulging and increased tension of the anterior fontanel. The first effect of increased flow of arterial blood into the brain is an exaltation of cerebral functions, but if the pressure becomes greater the capillaries are compressed, and there is a consequent slowing of the circulation and a deficient supply of oxygenated blood, so that in truth the effect may end in being the same as in asphyxia or in cerebral anæmia. When the intracranial pressure is due to a venous impediment from the start, the same ultimate condition obtains. Pressure due to serous effusions acts in a similar manner. The symptoms following a severe apoplexy are due to a great extent to the consequent disturbance of the circulation, which is generally regarded as an anæmia.

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[1] Pepper's System of Medicine, vol. v.

[2] Ziemssen's Cyclopædia, vol. xiii.

[3] Lumleian Lectures on Convulsive Seizures, Lancet, March 29, 1890.

SYMPTOMATOLOGY.

It may seem unscientific to speak of the symptomatology of a symptom, but, if the remarks with which this article opened be remembered, it is hoped the liberty will be allowed. For convenience of description this portion of the subject may be divided into three heads: 1st, laryngismus stridulus; 2d, the more general "sympathetic" disturbances, and those seen in the so-called "essential" convulsions or eclampsia proper; 3d, those convulsions consequent upon gross cerebral lesions.

The last division is one which cannot be discussed in this article. The reader is, therefore, referred to the articles on the diseases and surgery of the brain and spinal cord to be found elsewhere in this work. The difference between the first and second divisions must not be considered one of kind, but only one of degree; they have both many of the same predisposing causes, and the first may merge at any time into the second; they deserve, however, separate mention.

LARYNGISMUS STRIDULUS.—**Synonymes.**—Spasm of the glottis; Child-crowing; Cerebral croup; Inward convulsion, etc. Considerable confusion has arisen on account of a rather careless nomenclature as applied to this affection and to simple catarrhal laryngitis: thus, Mackenzie uses the term "spurious croup" as synonymous for both disorders; Meigs and Pepper and Jacobi consider "spasmodic or false croup" a synonyme of simple catarrhal laryngitis, while Mackenzie, Cohen, Steffen, Johnson, and others apply these terms to laryngismus stridulus.

Laryngismus stridulus is a comparatively rare form of local convulsion which is usually characterized by a spasmodic closure of the glottis, and in severe cases by spasm of all the other muscles of respiration. The prominent symptom of this is the peculiar crowing sound accompanying the efforts at inspiration.

The diaphragm and external muscles of respiration may be the parts affected, and the glottis escape, in which case there will be no crowing inspiration. Eustace Smith has found this condition in weakly or prematurely-born infants only.

The attack passes off in a few seconds or minutes, generally in an ordinary fit of crying. It usually comes on towards midnight, the child having been put to bed apparently in its ordinary health, or else having been fretful and cross for a day or two, and having possibly had an occasional slight catch in its respiration. The little patient awakens suddenly, makes a few short spasmodic efforts at respiration, which are followed by a long crowing or whistling inspiration resembling somewhat that heard in whooping-cough; the closure of the glottis then becomes complete, and all sounds cease. The face, which has been slightly flushed or pale, now becomes livid,—this being particularly noticeable about the mouth; the eyes are staring wildly or else are rolled upward; the head is retracted, and in severe cases there is marked opisthotonus; the thumbs may be bent into the palms, the fingers closed over them, the wrists flexed, and the forearms pronated. With this there is strong abduction of the great toes, while the remaining toes are strongly flexed,—"carpo-pedal contractions." The backs of the hands and the insteps appear swollen, and the skin may assume a shiny aspect. After this condition has lasted for a short period, during which dissolution seems imminent, the crowing inspiration is again heard, the spasms relax, and the attack ends in a fit of crying or coughing. Instead of this mode of termination, it is possible for death to occur during the first paroxysm, or at the height of the attack general convulsions may ensue. The epiglottis may become firmly wedged in the chink of the glottis by the spasm and remain there even after the latter relaxes, according to Cohen,[1] and in these cases, unless relief is obtained, the attack speedily ends fatally. The child may recover entirely after such a seizure as has been described and have no repetition of the trouble, or there may be several paroxysms on the same night or on the following night about the same hour, the child being apparently well in the interval. An attack of laryngismus may come on in the daytime, and need not be severe; it may come on during nursing; the child then drops the nipple, rolls up its eyes, gives a crowing inspiration, and may or may not flex the thumbs into the palms; upon recovering, which it does in a few seconds, a second attempt to nurse may cause a repetition of the symptoms. Such an attack as this may follow an exhibition of temper, or even an ordinary crying-spell, and is frequently misinterpreted as being an evidence of bad temper. A condition resembling the "status epilepticus" may be developed, in which the child does not fully recover from one paroxysm before it passes into another. As before said, the attacks are apt to come on at night or just as the child is awakening; they may occur from time to time, or there may be long intervals between them. Fever is absent, as a rule; if it is present, it is probably due to the same cause as the attack. Sweating of the head is a very frequent symptom in children affected with laryngismus, and is due to the rachitis which is usually present. The "carpo-pedal" contractions, above mentioned, are often entirely absent, and when they are marked, Mackenzie considers that they may be accompanied by great pain.

Cheadle[2] believes this condition to be identical with tetany, and, as it is always accompanied by laryngismus, this has been usually described as a symptom of the latter,—in his opinion erroneously. He quotes Abercrombie[3] as the first to differentiate these two affections. The pulse during an attack is small and sometimes almost imperceptible; when the anterior fontanel is still open, there is marked bulging during the stage of venous engorgement.

General Convulsions.—General convulsions are usually preceded by signs of perturbation of the nervous system, although Grancher[1] considers these of little importance. The child sleeps with its eyes half open, showing the white sclerotic beneath the upturned corneæ, starts and cries out in its sleep, grits its teeth, twitches its mouth, and occasionally a smile (risus sardonicus) passes across its face; there may be also a tendency to flexion of the thumbs in the palms. A gazing into vacancy, usually to one side and upward, with an expressionless face, is sometimes met with as a prodromal symptom, and during this condition the child's attention cannot be aroused. This may pass off in a few seconds or may be the immediate precursor of the attack. Laryngismus stridulus, as before mentioned, may merge into the more general convulsion.

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[1] Diseases of the Throat and Nasal Passages, 2d ed., 1879.

[2] London Lancet, May 7 and May 14, 1887.

[3] Thesis for Degree of M.D., Cambridge, Ballière & Co.

These prodromal symptoms may be absent, and the paroxysm may come on suddenly; but this need not follow the prodromal symptoms even when they are present.

General convulsions may present themselves in many forms; the attack may resemble in every respect a severe epileptic paroxysm; it may be tonic throughout its whole course, or violently clonic; it may be partial, affecting only a single group of muscles, or any number until all the muscles are affected; one variety may merge into another.

The tonic and the clonic spasms are essentially of the same nature, and, as in epilepsy, the latter coincide with the stage of cyanosis, and are probably due to the action of the carbonic acid of the venous blood upon the lower cerebral centres, not as a direct excitant, but as an interrupter and final arrester of the previously-existing spasm, so that, as Gowers (*loc. cit.*) expresses it, the clonic spasm is only the tonic spasm spread out.

In the attack of true infantile eclampsia, where there is no organic lesion as an exciting cause, it is very unusual for the movements to be strictly confined to one limb, or even to one side of the body, although they are nearly always more pronounced on one side than on the other. Simon[2] states that the right side is the one that is most severely implicated. Is this because most children are or will be right-handed? and does the converse proposition hold true?

The well-marked paroxysm cannot be described more clearly or with less exaggeration than is done by Meigs and Pepper[3] in the following words: "The child often utters a cry, loses consciousness, and is seized with powerful tonic contraction of the voluntary muscles; the eyes are for a moment fixed and staring, and then drawn obliquely upward under the upper lids, so that the white portions of the balls alone are visible for an instant between the partially-open lids; the trunk is rigid and stiff, the thorax immovable, the respiration suspended by rigid spasm of the respiratory muscles; the face, for a moment pale, usually becomes livid and congested, and the veins of the neck are distended. This stage of tonic spasm is followed quickly by the stage of clonic spasm, in which involuntary and most irregular and convulsive movements occur. The eyes are rarely fixed in one position, but are constantly agitated in various directions, from side to side, or upward and downward; very often there is the most violent strabismus; the eyelids are sometimes opened, and at others shut; the pupils may be contracted or dilated. The muscles of the face next enter into contraction, and occasion the most hideous contortion of the features, the mouth is distorted into various shapes, the lips are often covered with a whitish or sanguinolent froth, and the jaws tightly clinched together by tonic spasms, or agitated by convulsive movements, so as to produce grinding of the teeth. The trunk of the body is also sometimes variously contorted by clonic convulsions; the head is usually strongly retracted upon the trunk, but in other instances is drawn to one side or violently rotated. The muscles about the front of the neck enter into action, and alternately elevate and depress the larynx; the tongue, when it can be seen, is observed to be moved in different directions, and is sometimes caught between the teeth and severely bitten. The extremities, particularly the superior, are more violently convulsed than any other parts. The fingers are drawn into the palms of the hand; the forearms are flexed and extended upon the arms by short, rapid, and generally rhythmical movements; the hand is quickly pronated and supinated upon the arm, or, finally, the whole upper extremity is twisted and distorted into various positions, which it is impossible to describe. The inferior extremities undergo similar movements, but almost always in a less degree than the upper. The respiration during the attack is irregular, sometimes suspended by rigid spasm of the respiratory muscles, and sometimes accelerated."

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[1] Convulsions in Children, Gaz. Méd. de Paris, January 21, 1888; also review in Archives of Pediatrics, Philadelphia, May, 1888, p. 305.

[2] Des Convulsions chez les Enfants, Gaz. des Hôp., 1882, tome lv. pp. 428–436.

[3] Op. cit., pp. 542, 543.

The face, instead of being livid, may be pale throughout the whole of a severe attack.

The attack ends by the movements gradually diminishing in violence, leaving the child in a deep sleep or else in a state of stupor. The child may have only one such attack and recover entirely, or after apparent recovery may pass into a second, and this may be repeated several times with considerable intermissions of comparative health, or one attack may pass into another with scarcely any intermission, and this state, which is analogous to the status epilepticus, may last for hours, the child becoming more and more stupid after each attack, until it may become profoundly comatose, and even die in this state. Considerable fever is often present in the status epilepticus, or *eclampticus*, if the expression be allowed. During the paroxysm the heart's action may be irregular; it is usually tumultuous. It is very difficult to make any observations on the pulse, on account of the incessant muscular action, and authorities differ widely in their statements in regard to this. In epilepsy, an analogous state, Echeverria[1] gives drawings of sphygmographic curves, according to which the pulse before the seizure was higher, distinctly dicrotic, and accentuated. Nothnagel (*loc. cit.*) states that the iris is generally dilated at the beginning of an attack of epilepsy, and this is usually the case in eclampsia, as it frequently is in the artificially-produced eclampsia in animals.[1]

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[1] Quoted by Nothnagel, Ziemssen's Cyclopædia, vol. xiv. p. 229.

When convulsions are entirely confined to one side there is the strongest probability that they are due to gross cerebral lesions, and when they persistently occur in one part, are confined to this, or spread to others in some one particular order of sequence (even should they subsequently become general), then the presumption becomes still stronger, and, in fact, almost amounts to a certainty, that a focus of disease exists in the motor area of the cortex, or in the subjacent white matter, at the point corresponding to the part primarily affected in each paroxysm. This division of the subject properly belongs to the articles on gross cerebral diseases, to which the reader is referred.

In the general convulsions, consciousness is lost from the beginning, but in the partial attacks above described, this may be retained throughout, or not lost until by the successive implication of neighboring areas in the "discharge" the convulsion becomes general.

It is rare for the bowels to be mechanically evacuated during a severe paroxysm by the force of the muscular movement. According to Simon (*loc. cit.*), the secretion of urine is entirely suspended in eclampsia, and the subsequent discharge of urine is a critical phenomenon which announces with certainty the approaching termination of the attack or the end of a series of them, although a few more spasms may occur before their final cessation. The temperature of the body varies according to the cause of the convulsion: in laryngismus stridulus and in the so-called essential convulsions it is usually normal, or nearly so, except in the status eclampticus, as before mentioned. The same is true of uræmic convulsions, whereas in those ushering in the acute diseases there is high fever,—103° F. and 104° F. and above.

DURATION AND COURSE.

Each individual attack of laryngismus is generally of but short duration,—a few seconds, perhaps,—but one paroxysm may scarcely be recovered from before another commences, and this condition may last for hours. As the condition underlying this neurosis is a chronic one, the attacks are liable to return from time to time whenever an exciting cause is present. The child may have but one attack, or these paroxysms may return for months, or even years, and yet recovery take place. Long periods may intervene between the paroxysms, or as many as thirty or forty may occur in twenty-four hours (Steffen). In general terms it may be said that the tendency to return extends over several months. In the more severe attacks, general convulsions are apt to supervene.

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[1] H. C. Wood, A Study of Morbid and Normal Physiology, *loc. cit.*

General convulsions differ more decidedly in their duration and course than the local variety above described. When they usher in an acute disease they are apt to be single, or at least repeated but once or twice, unless towards the close of the disease, when they indicate complications, and are apt to return more frequently.

When they are simply due to peripheral irritation, or to reflex impressions starting from the gastro-intestinal mucous membrane, they are generally single, and are apt to be relieved on the removal of the cause; but if this is not done, other attacks are probable. The duration of the paroxysm itself varies from a few moments to half an hour or more. Meigs and Pepper have seen a case which lasted twelve hours; they may even last longer, but this long duration is rare and usually consists of a repetition of attacks. When the convulsions are due to gross intracranial lesions, the course is apt to be entirely governed by the character of the exciting disease.

When death is due to the convulsions, whether of the local or the general variety, and not to the exciting cause of the paroxysms, it may occur in three ways: 1st, by apnœa, the child dying suffocated; 2d, in consequence of the changes occurring in the brain due to the venous stasis, effusion of serum, hemorrhage, etc.; 3d, by exhaustion in consequence of the frequent repetition of the attacks.

PROGNOSIS.

The prognosis of a symptom presenting itself in so many forms, and due to so many conditions, both functional and organic, of necessity will vary exceedingly. A few generalities may be allowed before entering into the discussion of the prognosis in special cases. A child that has been exhausted by long illness has on this account a great diminution or even an abolition of its reflex excitability, so that convulsions occurring in this condition are of much more serious import than an equally severe attack in a robust child, and usually betoken grave cerebral mischief.

The mere discovery of albumen in the urine should not of necessity lead to the diagnosis of kidney-disease, as it may be due to the presence of fever or possibly be in consequence of the severity of the paroxysm. Huppert,[1] quoted by Nothnagel,[2] has found albumen after *every* fully-formed epileptic seizure, the albuminuria lasting from three to four hours; but other authorities have failed to verify this statement. Mabille[3] failed to find it in thirty-eight cases, either before, during, or after the seizures. The discovery of large quantities of albumen at any one time, or of its continuous presence in smaller quantities, particularly if casts are found, should lead to the probable diagnosis of kidney-disease, with the consequent grave prognosis.

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[1] Virchow's Archiv, vol. lix.

[2] Ziemssen's Cyclopædia, Amer. ed., vol. xiv. p. 233.

[3] Annales Médico-Psychologiques, 1880, 6th ser., vol. iv. p. 415.

Where no exciting cause can be discovered, or where the fits continue after the exciting cause of the first attack is removed, the prognosis is somewhat doubtful, and the probability that they may become permanent must be entertained. No convulsion should be looked upon as of little moment, no matter how prominent an exciting cause may apparently be discovered, for this may have been but the last increment of excitation that was needed to produce a convulsion in reality due to some severe organic disease; besides, a purely reflex convulsion, when repeated, may leave behind some indefinable and intangible impression which may be the starting-point of future attacks, a "habit" being thus induced which is possibly in part identical with epilepsy.

If a positive exciting cause can be discovered, the fit is in all probability eclampsia, particularly if the child has been in previous good health. A temporary paresis of an arm or a leg is not a proof of central lesion unless the weakness lasts for some hours, when such a prognosis becomes probable, whether the lesion is primary or secondary.

Convulsions are to be looked upon as one of the modes of dying, and thus theoretically may have nothing to do with causing the fatal termination. It is partly owing to the frequency of this final symptom that the dread has arisen in the minds both of the medical profession and of others as to the very unfavorable prognosis of all convulsions.

The proportion of deaths is a very difficult thing to estimate, as the records of a large number of attacks and their subsequent history are wanting. The mortality records are also to be looked upon with suspicion, as previously stated. Convulsions of themselves may be the immediate cause of death, but this termination is rare in comparison with the recoveries, and it should be borne in mind that in proportion to their frequency they less often point to grave organic mischief in children than in adults.

West[1] computes that 30.5 per cent. of all deaths under one year occur from diseases of the nervous system, that 73.3 per cent. of these latter deaths are due to convulsions, and that of the total deaths under five years of age 24.3 per cent. are due to diseases of the nervous system, of which 54.3 per cent. are due to convulsions. This is equivalent to saying that 22.35 per cent. of all deaths under one year, and 13.19 per cent. of all deaths under five years, are due to convulsions.

Of the total deaths under ten years in my tables,—viz., 86,769,—7508 were ascribed to convulsions, a percentage of 8.65, which is not nearly so high a mortality as that given by West; but this may be owing to the ages of the cases included in my calculations.

Convulsions in very young children—that is, within the first few days of life—are in all probability due to meningeal hemorrhage, generally produced by injury received during difficult labors; they are of grave import.

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[1] Diseases of Children, 5th Amer. ed., 1874, p. 44.

Osler[1] states that in children dying shortly after birth in asphyxia or convulsions these symptoms are usually due to this condition, and Nothnagel[2] makes an analogous assertion.

In older children this statement does not apply, and the probability of the attack being of reflex origin is increased. If fever is present, particularly if it is high, the accompanying convulsion probably announces the onset of one of the acute diseases, or it may mean thermic fever, and the prognosis varies exceedingly, as will appear later.

The prognosis of eclampsia, so far as it relates to the effect on the future health of the patient, is a very important question.

Many authorities agree that convulsions in infancy are intimately connected with the epileptic diathesis. Féré[3] considers that scarlatinous eclampsia predisposes to epilepsy in after-life. Coutts (*loc. cit.*), after a study of 68 cases of infantile convulsions, in which the health in later years was recorded, found that 31 were the subjects of neuroses in later life: thus, 11 had epilepsy (8 grand mal and 3 petit mal); 6 were somnambulists, and one of these had melancholia, and another hystero-epilepsy with suicidal tendencies; 3 had melancholia alone; 4 chorea; 7 periodical migraine; and of the 37 remaining cases, 6 were eccentric and irritable, and nearly all the remainder were in intellect below their brothers and sisters who had not had convulsions.

Gowers[4] found 180 cases out of a total of 1450 cases of epilepsy in which the attacks began during the first three years of life; nearly two-thirds of the cases beginning in infancy the condition of origin of which could be ascertained, arose from the so-called dentition convulsions; and, ascribing to the same cause a similar proportion of the cases respecting which no history was forthcoming, he found a total number due to this cause which constituted 7 per cent. of all those investigated.

A few authors doubt whether infantile convulsions hold any causal relation to this disease; but enough has been determined to make us watch each case with care and be reserved in our prognosis.

Rickets is considered to hold a causal relation to epilepsy; and, as this condition is present in many cases of eclampsia, it may be a partial explanation of the facts stated above.

Arlt[5] originally called attention to the fact that lamellar cataract has been found to be associated with a previous history of infantile convulsions, and Hutchinson[6] shows that this condition of the lens is usually associated with dental defects other than those which he believes to be an evidence of inherited syphilis. He considers the dental "erosions," as they are erroneously called, to be due to the mercury which has been, in all probability, given for the convulsions which were the cause of the cataract, but also allows that the nervous disturbance which attends the convulsions may exert some influence on the growth of the teeth.[1] Magitot (*loc. cit.*) believes that the cataract and the defective teeth are both due to infantile convulsions, and confidently asserts that "l'érosion dentaire, dans ses formes *d'échancrure, de sillon, de nappe*, etc., est le signe rétrospectif constant de l'éclampsie infantile." Rachitis and measles are also capable of producing these dental defects: so that they cannot be considered as positive evidence of previous convulsions. The cataracts and the dental defects should be looked upon as of the same nature as the deformities and markings on the nails which follow periods of serious disturbances of nutrition,[2] except that the former are permanent and the latter transient.

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[1] Cerebral Palsies of Children, Medical News, Philadelphia, August 11, 1888.

[2] Ziemssen's Cyclopædia, vol. xii. p. 178.

[3] Éclampsie et Épilepsie, Archives de Neurologie, 1884, tome viii. p. 48.

[4] Epilepsy and other Convulsive Diseases, 1881, p. 22.

[5] Quoted by Wells, Treatise on the Diseases of the Eye, 1869, p. 219.

[6] Imperfect Teeth and Lamellar Cataract, Transactions of the Pathological Society, London, 1875, vol. xxvi. p. 235 *et seq.*

The convulsions ushering in the exanthemata are not of so serious a prognosis as those occurring later in the course of these diseases.

In scarlet fever, convulsions at the beginning are of very grave significance, although some patients may recover; but after the appearance of the rash they usually indicate a fatal termination. In malignant measles convulsions are of frequent occurrence at the onset, but if the child has been in previous good health, and the epidemic is of ordinary severity, convulsions at the onset are of no particular significance; not so, however, are those attacks occurring later in the disease, when this symptom usually indicates a fatal termination by some complication. The same is true of scarlet fever. Convulsions are rarely seen in the other exanthemata.

In whooping-cough, convulsions are of very grave significance. Ozanam[3] has seen but one child restored to health after this complication. In the experience of Meigs and Pepper five out of twelve cases were fatal. Roger (*loc. cit.*) does not consider this symptom necessarily of fatal significance in cases otherwise uncomplicated, but when complicated with a broncho-pneumonia, death in his experience usually followed.

Convulsions are quite frequent in young infants in malformations of the heart and great vessels, and are usually fatal, being the cause of death in fifty per cent. of the deaths from cyanosis.[4]

Toxæmic convulsions are also of grave significance, whether occurring as a symptom of uræmia or on account of poisoning. In such wasting diseases as cholera infantum, diarrhœa, etc., convulsions are not uncommon, and usually indicate the beginning of the end.

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[1] Loc. cit., and discussion on M. Magitot's paper "Sur l'Érosion des Dents considérée comme signe rétrospectif de l'Éclampsie infantile," Transactions of the International Medical Congress, Seventh Session, 1881, vol. iv. p. 128.

[2] J. M. Da Costa and Morris Longstreth, Transactions of the College of Physicians, 1877, vol. iii. pp. 109 and 113.

[3] Quoted by Steffen, Ziemssen's Cyclopædia, vol. vi. p. 706.

[4] Keating and Edwards, Diseases of the Heart and Circulation, etc., Philadelphia, 1888

SEQUELÆ.—Among the sequelæ that occasionally occur after general convulsions may be mentioned hemiplegia; contractures; defects of speech and hearing; lamellar cataracts and defective teeth; local palsies, such as squinting, etc.; and occasionally, in addition to these, some mental deterioration. Macroglossia has been regarded by some as an occasional sequel. These may be the effect of the paroxysm itself or may be caused by the original lesion giving rise to the convulsion. The prognosis as regards recovery from these complications is unfavorable, though they are not necessarily of unfavorable significance otherwise.

LARYNGISMUS STRIDULUS.—There are two points to be considered in the prognosis of this rather uncommon affection: 1st, the paroxysm itself, and, 2d, the underlying general condition giving rise to the spasm. The ordinary attack is not so fatal as some observers report, although death frequently happens, either from the severity of the paroxysm or, more rarely, from the mechanical wedging of the epiglottis into the chink of the glottis: nevertheless the prognosis is always rather serious, on account of the possibilities. Jacobi[1] considers that the child will get well unless within the first four to six weeks some untoward circumstance happens. When the paroxysm is a very pronounced one and is accompanied by carpo-pedal spasms or merges into general convulsions, the prognosis becomes very grave, for then other attacks are likely to follow, in any one of which the child may die.

The spasm may be so sudden in its onset that the crowing inspiration is absent, all respiration ceasing instantly. This spasm usually relaxes only in death.

Reid[2] states that of 289 cases which he collected, 115 died; and Meigs and Pepper state that of 61 cases, 4 died of intercurrent or consecutive diseases, while of the remaining 57, 32 were cured, and 25, or about 48 per cent., died of the malady itself; adding these cases together makes a total of 350, with 140 deaths, a percentage of 40.

When the paroxysm has passed over, the underlying condition remains, and the prognosis of this, if it be rickets, is not usually unfavorable. If an exciting cause other than this can be found and is removable, the outlook is favorable.

PATHOLOGY.

In diseases where the pathology is doubtful, or where the lesions to be expected differ and are not very pronounced in character, the views previously entertained of the cause underlying the disorder unfortunately frequently color the description of the conditions found at the autopsy; and eclampsia is not an exception to this rule.

The post-mortem appearances detailed by authors a few years ago differ materially from those of more recent date, the former finding signs of active congestion of the brain, and the latter passive congestion or anæmia. But little light has been thrown by autopsies upon the condition of the nervous system which gives rise to convulsions. In true eclampsia, varying conditions of the brain and membranes have been described, such as congestion of the cerebral veins and sinuses with transudations of serum, but these are more probably the effect rather than the cause of the paroxysm. The gray matter of the brain has been several times noticed to be paler than usual, but Steffen,[1] on the contrary, states that a greater or less degree of hyperæmia of the brain-membranes and of the brain itself has always been found, and sometimes with effusion of blood or œdema. A significant fact in regard to the causation of true eclampsia is the state of the alimentary tract, which, in the autopsies where any mention is made of its condition, frequently presents appearances indicating acute indigestion, the stomach sometimes being full of undigested food.

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[1] On Craniotabes, American Journal of Obstetrics, vol. iii., November, 1870.

[2] Infantile Laryngismus, quoted by J. Lewis Smith, loc. cit., p. 443.

When the convulsions are due to foci of disease, or to general systemic disorders, the appearances found after death will necessarily vary with the exciting cause.

To cite all the conditions found after death in convulsions in children would necessitate enumerating nearly all the pathological conditions to which childhood is liable.

In laryngismus stridulus post-mortem examinations show nothing more positive. Jacobi (*loc. cit.*) states that the condition found is "a positive absence of hyperæmia in the brain, and no or very little blood in the heart and cutaneous veins."

Different inflammatory conditions are sometimes found in the mucous membrane of the larynx, and occasionally the epiglottis may be found incarcerated in the chink of the glottis.

DIFFERENTIAL DIAGNOSIS.

For the sake of facility in diagnosis, convulsions may be grouped, somewhat arbitrarily, into eight classes:

I. Convulsions may be an evidence of reflex irritation from some temporary and comparatively trivial disorder, such as an overloaded stomach, irritation of teeth, earache, worms, constipated bowels, genital irritation, etc.

II. They may constitute an initial symptom of one of the exanthemata or of some other acute disease, thoracic, spinal, or cerebral, or may be due to thermic fever.

III. They may indicate that the child is suffering from some general condition of ill health, such as indigestion, rickets, cyanosis, etc.

IV. They may indicate gross intracranial disease of a more or less chronic nature.

V. They may be a symptom arising during the course of one of the acute diseases, such as whooping-cough, or may be an evidence of toxæmia in the exanthemata or in kidney-diseases.

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[1] Ziemssen's Cyclopædia, vol. vii. p. 1012.

VI. They may be the result of a serious traumatism, such as injury to the head, a violent hemorrhage causing cerebral anæmia, or a severe burn or shock.

VII. They may be one of a series arising without assignable cause, and constitute epilepsy.

VIII. They may indicate nothing more than a mode of dying, particularly in those diseases which are accompanied by diarrhœa and vomiting.

If the child is well nourished and plump, and if there is no fever, the attack is in all probability to be placed in Class I. It may be an attack of laryngismus stridulus; but should there be high fever, it is more likely that one of the acute diseases is about to manifest itself. Should the child be rickety, the sources of irritation mentioned in Class I. are still more potent, so that Classes I. and III. are frequently found associated.

For the differential diagnosis of Class IV. the reader is referred to other sections, but the general statement may be made that convulsions confined to one side, or to the face and arm, or to either of these alone, returning frequently and maintaining the same character of initial movements ("primary movements" of Horsley and Beevor),[1] and of the order of invasion, should suggest strongly their organic nature, particularly if consciousness is retained throughout, or is present in the earlier part of the paroxysm. The same probability of organic origin exists if the child is under-nourished and weakly, and therefore in a condition in which reflex excitability would naturally be in abeyance. Convulsions occurring in Class V. should offer no difficulty of diagnosis, as the primary disease will be so marked that a mistake would be very unlikely. The same is true of Class VI. There is, probably, no essential difference between the processes at work in the causation of the epileptic and of the eclamptic paroxysm, and it is impossible to define where the latter ends and the former begins. As previously mentioned, the first attack in a young child previously healthy is probably eclamptic; the same probability holds good even if there have been previous attacks, provided a cause for the seizure can be found; but if the child has had many before, and if they arise without definite cause, or arose from a definite cause at first which is now no longer in existence, then it becomes more probable that the attack is epilepsy, particularly if the child has passed the period of infancy.

TREATMENT.

The section upon treatment is naturally divided into two portions: 1st, that appertaining to the paroxysm itself, and, 2d, that appertaining to the predisposing cause,—viz., the systemic condition of which the paroxysm is merely a symptom.

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[1] Topography of the Cerebral Cortex, American Journal of the Medical Sciences, April, 1887, p. 347.

The physician will frequently be compelled to treat an attack in ignorance of the exciting cause, as this manifestly cannot be fully investigated while the child is being convulsed; but he should avoid falling into the error of treating all cases alike, as if the convulsion was a disease instead of a symptom of varying significance. It would be manifestly improper to treat an attack due to excessive fever or to sunstroke in the same way as one due to reflex irritation from digestive disturbances. Fortunately, in our perplexity, there are several agents which are eminently useful in quelling nervous outbreaks, however they may be produced, thus giving us an opportunity of discovering and possibly removing the exciting cause.

The opinion generally held, and particularly by the public at large, is that a convulsion in a child is an evidence of acute congestion of the brain, and to combat this condition the warm or hot bath, with or without mustard, is advised by the majority of authorities, in order to draw away the blood from the brain to the skin by causing an expansion of the peripheral vessels. This treatment certainly has the sanction of custom, and so universal is the belief in it that, in all human probability, the child will be placed in the bath before the arrival of the physician. That this is universally applicable and beneficial admits of grave doubts, as will be shown later.

Meigs and Pepper[1] consider that "it is a good rule always to place the child, no matter what is the cause of convulsion, if it be a severe one, in a warm bath (96° or 97° F.)," and this is undoubtedly a safe rule; but a hot bath (100° to 110° F.), such as is often employed, may be decidedly harmful, especially if the convulsion follows the ingestion of food, as the bath certainly tends to render the digestion of the recently-taken nourishment still more difficult. No one would think of giving a healthy child a hot bath immediately after a meal, for this very reason, as the withdrawal of blood from the stomach and internal organs to the skin would most likely be productive of harm.

A. A. Smith[2] considers that a hot bath always does harm, as the child usually has a convulsion or two immediately after it is given. This last statement is, perhaps, overdrawn, although a warm bath would certainly be the less harmful.

If the bath is given and does not very soon afford relief, it should be suspended. Should high fever exist, the warm bath, being below the temperature of the body, exerts a powerful influence in reducing the latter, and thus does good; not so, however, the hot bath, which would be positively harmful here. Cold to the head is usually advised in connection with the preceding, and where there is fever this is useful, but otherwise its utility is doubtful. Where there is anæmia of the brain as the exciting cause of the attack, the hot bath and cold affusions are contra-indicated. The conclusion therefore is that the hot bath is harmful and contra-indicated in infantile convulsions, and that the warm bath is a comparatively safe and sometimes useful treatment in the "essential" and some of the "sympathetic" class, although in the latter, where there is fever, a cool sponge-bath would be more efficacious.

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[1] Loc. cit., p. 551.

[2] New York Medical Record, 1880, vol. i. p. 521.

As remarked under etiology, dentition is frequently considered as the cause of the attacks, and, in consequence, lancing of the gums is advised and sometimes ruthlessly practised. The older authorities advise this more strongly than those of more recent date; thus, Marshall Hall counsels deep lancing of the gums "twice or even thrice daily;" but such universal lancing is horrible, and the operation should not be performed unless there be swelling, heat, and redness over one or more advancing teeth, when it will give decided relief, as it will when no nervous phenomena accompany the process; the same rule should guide us in both conditions. Needless lancing of the gums only serves to increase peripheral irritation, and thus tends to produce the very symptom the operation was intended to relieve. When the gum is of a natural color and hard and not swollen, it should most decidedly not be cut. Henoch (*loc. cit.*) strongly deprecates the use of the lancet.

Should the child be robust and have been in previous good health, and if there is no fever, the probability is that digestive troubles are the cause of the attack, and an emetic, supplemented by a large injection, will be indicated. The emetic becomes peremptory should there be a history of the recent ingestion of unsuitable food. A heaping teaspoonful of a thick mixture of alum and syrup of ipecac may be given as soon as the child can swallow, or the fauces may be tickled with the finger or a feather. Should it be thought that the indigestible food has passed into the intestine, a laxative should be given, and this may be calomel, or, in some cases, castor oil, or simple syrup of rhubarb, either alone or in combination. An enema will also be of service here.

We now come to a class of remedies that are of great service in convulsions of all kinds. I refer to the bromides and chloral hydrate. These may be used alone or in combination. If the child can swallow, from two to five grains of bromide of sodium or of potassium, preferably the former, on account of the less likelihood of its causing disturbance of the stomach, with from one to two grains of chloral, may be given in solution every fifteen minutes for from four to six doses at one or two years of age.

If the child is unable to swallow, from five to ten grains of chloral may be given by enema in a small quantity of warm water, and repeated in a short time if the convulsions do not lessen or disappear. The use of chloral by injection is one of the most effectual means of checking the paroxysm, and should compression of the rectum laterally with the fingers not suffice to retain the enema, a soft-rubber male catheter may be employed with which to administer the injection as far up the bowel as possible.

Cheadle (*loc. cit.*) advises the hypodermic use of chloral in from one- to three-grain doses; but Dastre[1] states that this method of administering the drug should be abandoned, on account of the almost certainty of consequent abscess. The writer has had no experience with this use of chloral.

Most attacks of general convulsions not due to organic disease will yield to this treatment, but, should the attack prove intractable, other means must be tried.

Opium is of great value in quelling nervous excitement, but intense venous engorgement of the brain, either primary or secondary, and coma are contra-indications. The most certain method of administration is morphine by hypodermic injection, and it should be used without hesitation should the attack be a severe one and not yield to the treatment mentioned above.

For a child one year old, from one-twenty-fourth to one-thirtieth of a grain may be given with but little danger, and repeated if necessary. But few of the text-books speak of morphine hypodermically in this connection in children. After its use the child will frequently fall asleep and awake much better.

A. A. Smith[2] considers that opium in the form of laudanum or paregoric should be given in all ordinary cases over four months of age, to quell excitement, and then other treatment be instituted; and that pain is an important indication for its use, unless this be due to too full a stomach or to tight lacing, etc. Plant[3] is of the opinion that those cases which are due to cerebral anæmia from loss of blood or profuse diarrhœa, where the features are pale, the fontanel depressed, and the child under one year, particularly demand opium.

Chloroform and ether by inhalation also possess great power in suspending convulsions, and, in a severe attack, should be administered at once; but caution must be exercised in their use, although children bear anæsthetics better than adults. Henoch (*loc. cit.*) considers any other initiative treatment a waste of time.

Nitrite of amyl is well known to exert beneficial effects in certain forms of epilepsy, and its use might naturally be expected to be of benefit in cases of eclampsia. Eustace Smith (*loc. cit.*) and Bridger[4] both speak highly of its use in this connection. The journals contain reports of a few cases in which it was successfully employed, while some writers state that they have seen no benefit whatever from its use. In those cases in which there is anæmia of the brain due to spasm of the arterioles it would seem to be especially indicated. Its action in depressing the functional activity of reflex motor centres would seem also to make it particularly applicable in cases due to purely reflex causes.

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[1] Étude critique des Travaux récents sur les Anesthésiques, Revue des Sciences Médicales, etc., Paris, 1881, p. 747.

[2] American Journal of Obstetrics, 1880, vol. xiii. p. 705.

[3] Archives of Pediatrics, 1884, vol. i.

[4] London Lancet, April 22, 1882, p. 668.

Other remedies have been mentioned by authors as of service,—viz., asafetida, valerian, musk, etc.,—but the treatment mentioned deserves the most confidence. Blisters and leeches to the nape of the neck and behind the ears have been recommended, but are rarely, if ever, needed in "essential" and "sympathetic" convulsions; the same may be said of venesection.

When the attack is accompanied by high fever, and is probably a prodromal symptom of one of the exanthemata, a *warm* bath or a cool sponge-bath will act beneficially in tending to reduce the fever and at the same time favor the appearance of the rash. Bromides and chloral will be needed also. When the child is suffering from sunstroke, the main indication is to lower the temperature, and the cold bath or sponging, the cold pack, antipyrin, and possibly morphine hypodermically, should be employed.

Should pain be the exciting cause, anodynes will be needed. This is especially true of earache, when hot anodyne applications and morphine or cocaine instillations into the ear may materially aid in addition to the usual antispasmodic treatment. One or two leeches to the ear will be of benefit here.

Where the paroxysm is due to uræmia, there should be no hesitancy in the treatment, and the kidneys, skin, and bowels must be made to relieve the system overcharged with morbific products. The reader is referred elsewhere for fuller details.

If a strong malarial element is present, quinine should be given after the convulsions have been quieted.

Besides the general rule laid down for the treatment of convulsions, nothing more definite can be said here in regard to those attacks which are due to gross lesions of the nervous system, as the consideration of this portion of the subject is beyond the scope of the present paper.

For the more local forms of convulsion, known as laryngismus stridulus, a somewhat different treatment from that for general convulsions should be employed. The child should be raised into the sitting posture, as this gives it a better opportunity to breathe and removes one possible cause of the attack,—viz., pressure on the occipital bone, especially if craniotabes is present. All useless crowding about the little patient by anxious friends and relatives must be prevented. Slapping the child's face or chest with the end of a towel dipped in cold water, or holding ammonia or acetic acid to its nose, may make the patient take a sudden inspiration and thus abort the attack. A sponge wrung out in hot water and applied to the throat, or ice to the epigastrium, as directed by C. D. Meigs,[1] may sometimes have the same effect. Ice or spray over the larynx, or a small piece of ice introduced into the rectum, has also been known to cut short a paroxysm. The fauces may be tickled with a feather or the finger to excite vomiting, for this act is frequently known to interrupt the paroxysm. Should there be incarceration of the epiglottis, as rarely happens, this should at once be released by the finger; if this fail, tracheotomy must be resorted to. Steffen (*loc. cit.*) never saw a favorable result after tracheotomy. Emetics cannot be administered by the mouth during a severe attack, but they relieve where there is a continual stridor. Apomorphine hypodermically has been suggested by Steffen;[1] but this is a somewhat hazardous procedure, and if used at all it should only be in sthenic cases.

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[1] Meigs and Pepper, loc. cit.

Adler[2] reports a case of hysteria in an adult simulating laryngismus stridulus in which this drug acted in a very satisfactory manner, much better than nitrite of amyl: so that it seems possible that in suitable cases this remedy would be an efficacious one in severe laryngeal spasm in children.

Ether and chloroform by inhalation will at once cut short the attack, but, as before mentioned, should be used with caution, and not be trusted to incompetent persons. Nitrite of amyl might be tried if it could be administered early enough.

As in the more general attack, the bromides and chloral are of decided benefit, particularly the latter.

Mackenzie and Steffen (*loc. cit.*) both speak highly of the use of musk during the attack, or as soon after it as possible, in one-and-a-half-grain doses.

The same remarks apply to lancing of the gums as were made under the head of general convulsions.

Should the immediate paroxysm be over, whether it be of the general or the local kind, the condition usually remains which predisposed to the attack, and this must be attended to. The diet of the child should be carefully inquired into and properly suited to its age and condition, and every measure that will improve the hygiene strictly enforced; this is of the utmost importance. Bromides should be given after both forms of convulsions, in order, if possible, to prevent a return.

If an attack of laryngismus is feared, either from the symptoms or from the fact that the child has had a paroxysm on the preceding night, opium and one of the bromides may be given in combination,—viz., from two to five drops of laudanum and from five to ten grains of bromide of sodium or potassium,—and repeated if necessary. This may stop the impending attack in the same manner in which it acts in laryngeal spasms due to simple catarrhal laryngitis, although the two affections must not be confounded. Antipyrin has rendered good service in my hands in the latter condition, and, although the opportunity has not arisen for its administration in laryngismus stridulus, I should not hesitate to use it, and should expect benefit from it. Perceval[3] has recently employed this drug with success in twenty-four cases of laryngismus stridulus accompanied by convulsions, no case requiring more than five grains of the remedy.

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[1] Ziemssen's Cyclopædia, vol. vii. p. 1022.

[2] University Medical Magazine, March, 1889, p. 351.

[3] Antipyrin in Laryngismus Stridulus, London Lancet, 1888, vol. ii. p. 961.

When rachitis is present, treatment addressed to this condition is essential; proper supervision of the diet, cod-liver oil, iron, fresh air, change of location, etc., will be indicated, and thus the tendency to the attacks of laryngismus may be ultimately eradicated. Any other systemic disease that is present should receive full attention and be treated appropriately.

Richardson[1] has used peroxide of hydrogen and ozonic ether, alone or in combination, for stridulous spasm in whooping-cough, with apparent benefit, and, although the author knows of no case of laryngismus relieved by the use of these drugs, it would seem as natural to expect relief in the latter condition as in the former.

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[1] The Asclepiad, 1887, p. 54.

EPILEPSY.

By LANDON CARTER GRAY, M.D.

Synonymes.—Latin, Morbus sacer seu comitialis; French, L'Épilepsie; German, Fallsucht; Spanish, Mal caduco.

Definition.—The term epilepsy (from ἐπιλαμβάνω, "to seize") is used to denote convulsions, either tonic or clonic, of one limb or of several, or of the whole body, with or without loss of consciousness; or loss of consciousness of a certain character; or certain phenomena acting as equivalents to the typical symptoms.

Clinical Symptoms.—In the typical attacks of epilepsy the patient suddenly loses consciousness, utters a sharp automatic cry, falls, has a series of tonic muscular movements of short range, and quickly passes into a condition of general clonic convulsion, which lasts for several seconds or minutes, when the patient partially returns to consciousness, being apparently in a dazed condition, and then gradually sinks into what seems like a deep sleep but is really a return to unconsciousness, this quasi-slumber usually lasting for an hour or more. But the deviations from this type, which is known as *grand mal*, or *major epilepsy*, are innumerable. The most frequent variation consists simply in a loss of consciousness, either without any convulsive movements or with only very slight ones. This is known as *petit mal*, or *minor epilepsy*. In other cases, especially where the heredity is dying out in a family, there will be convulsive movements of the fingers alone; in others there will be simply a sensation of vertigo, which, indeed, very frequently occurs between the attacks of *grand mal* and *petit mal;* in others there will be various sensations which are called *auræ;* and in still others there are conditions of double consciousness, as it is called, in one of which a person may lead his usual life and in the other lead a totally different one. The most frequent form, however, is either the *grand mal* or the *petit mal*.

The countenance in these attacks varies greatly, being sometimes pale at the outset, and sometimes suffused. It must be borne in mind that statements in regard to this matter are extremely unreliable, as the by-standers are usually too much agitated to be accurate observers. The physician himself seldom sees an attack, unless he be a resident of a hospital: thus, while it would be difficult for me to remember the number of cases that I have treated in twenty years of professional life, it is very few indeed that I have seen during an attack.

Hystero-epilepsy is a curious mixture of hysteria and epileptoid symptoms, varying in degree from the pronounced hysterical attack to the frightful cases which are illustrated in the recent French writings, but which are seldom, if ever, seen in this country. All, however, differ from true epilepsy in the admixture or predominance of purposive or seemingly purposive movements instead of rigidly tonic or clonic movements of shock-like character; and opisthotonus is a non-epileptic symptom that is occasionally observed.

Procursive epilepsy is a variety recently described by Mairet, consisting of running movements forward, but otherwise with the usual phenomena of epilepsy. It may alternate with ordinary epilepsy or precede or merge into it.

Laryngeal vertigo, so called, is undoubtedly, in my opinion, a variety of epilepsy, and, although it has not as yet been described in children, the possibility of its occurrence in them should be borne in mind. It consists simply of a slight loss of consciousness, occasionally with light tonic or clonic movements, and is connected with some laryngeal, tracheal, or bronchial affection, such as laryngeal tumors, asthma, etc. The cases reported have ceased after the cure or removal of the seeming cause, but the histories have not extended over a sufficient length of time to exclude the possibility of recurrences.

Certain dream-like states may precede the typical manifestations of epilepsy, or take the place of them. They are simply degrees of impairment of consciousness. Of the latter, however, the conditions of double consciousness are the most marked. One patient of mine, a boy of fourteen, would pass days in wandering about the slums of New York, sometimes earning wages in some light occupation, without arousing the faintest suspicion in those about him, until some morning he would return to his normal condition, when he would quietly return home.

The epileptic insanities are treated in another article of this Cyclopædia.

We shall have occasion, in speaking of the pathological anatomy of the disease, to dwell upon the fact that epilepsy is but a symptom, just as is cough or fever, so that we may divide the epilepsies into those that are due to recognizable organic disease, those that are reflex, and those that we may call idiopathic. For the present we will speak of the clinical characteristics of the idiopathic form of the ordinary *grand mal* or *petit mal* type. This has certain peculiarities which are of great importance from a therapeutic and occasionally from a diagnostic stand-point, and pre-eminent among these characteristics are to be noted the nocturnal recurrence of some, the quasi-periodicity of others, the association of migraine, and the temporary response, generally in a favorable way, to slight changes in the environment or the treatment.

The nocturnal recurrence of epilepsy may very readily be overlooked, and should always be suspected in a child who is hereditarily predisposed to the malady, or in one who complains of waking in the morning exhausted and pale.

The quasi-periodicity of many epilepsies is a matter to which I first called attention some five years ago. It will be found that many individuals subject to this disease will have a recurrence of their attacks at certain times. Thus, females are more especially subject to it at or about the menstrual period, whilst in others it will be found to occur at certain times of the month, or at certain intervals, these intervals consisting sometimes of months and in some rare cases even of years. This may be utilized for therapeutics, as I shall point out farther on.

The association of epilepsy with migraine is a fact to which I also called attention for the first time some five years ago, and in these cases the epilepsy alternates with migraine, the migraine disappearing when the epilepsy appears, and the epilepsy returning when the migraine disappears. By this I do not mean to say that all cases of migraine are subject to epilepsy, but I do mean to say that there is a very close relationship between migraine and epilepsy, and in some cases the connection is so close as to permit of this alternation ; indeed, almost all cases of migraine will be found at some period of their lives to have had a loss of consciousness, with or without convulsive movements, although, of course, this fact is usually strenuously denied. But this association of migraine and epilepsy is of considerable therapeutic significance, as I shall show.

Another extremely curious characteristic of epileptics is the fact that they are so readily influenced by slight changes in the environment and in the treatment. This was first observed as far back as 1828 by the great Esquirol, who relates how, being at the head of the Salpêtrière, which was even then capacious, he divided his epileptics into groups, and put each group upon a different medicine and one group upon some disguised placebo ; how they all improved for a certain length of time, how they all relapsed at about the same time, and how each medicament had as much effect as the placebo, and no more. In addition to this, I have pointed out again and again that epileptics will do well for a time upon any change of treatment, whether that treatment be medical or surgical, whether it consist in cutting off the prepuce, removing the clitoris, extirpating ovaries, doing operations upon the male or female genitalia, using the hot iron or the moxa, cutting the eye-muscles, or even, as I have done myself, etherizing the patient and cutting a piece of skin out of the buttock ; nay, more, I have seen improvement effected in a patient for months by mere change of locality, and I have known of another inveterate epileptic whose fits did not return for months after she had soused herself in a vat of hot water, until the process of cicatrization was completed. These epileptic attacks are prone to recur in very variable spells, occurring every day, often several in a day, for weeks, perhaps months, and then spontaneously disappearing for a variable length of time, to recur again and again in the same old manner.

The pupils of most epileptics, as I have pointed out, are large and variable. Marie and Musso have attempted to show by measurements of the pupil that my statements upon this point are erroneous, but the variability of the pupils renders their measurements questionable, in my opinion. By this statement I do not mean to say that the pupils are not large and mobile in other nervous diseases, but simply desire to call attention to the fact that this is their condition in epilepsy.

The *facies* in most epileptics is unmistakable to a trained eye, although it is almost impossible to describe its fleeting traits. I think, however, that I could make a diagnosis of epilepsy in most cases by the *facies* alone. Of course it is much more marked in the inveterate cases which have been for a long time under treatment by the bromides, and at this period the accompanying listlessness, the pallor, the acne, the coated tongue, and the peculiar breath will greatly aid in the recognition.

The temper of most epileptics is extremely irritable, and especially is this the case in children whose malady has given them ill-advised license.

These characteristics of the so-called idiopathic epilepsy must be remembered by any one who would intelligently treat a case of the disease.

The epilepsies of organic origin, or of probable organic origin, are as various as are the functions of the brain. Hughlings Jackson first called attention to a form of epilepsy in which the convulsive movements were localized in one or more limbs, and to this in the course of time has been given the name of Jacksonian epilepsy. It has been found in many instances to be due to recognizable gross organic disease of the cortical motor centres or the underlying motor tract. The procursive epilepsy is believed by Mairet to be due—upon what grounds we shall learn later on—to cerebellar lesion. Almost any disease of the brain or spinal cord may have epilepsy among its symptoms, and this symptom may also be present with tumors of the brain or cord, the different forms of cerebral and spinal meningitis, hemorrhages of the brain or cord, traumata of the brain and cord, etc.

Etiology.—The factors which may be considered as bearing an etiological causative relationship to epilepsy are—age; sex; heredity; migraine; organic brain, spinal, or peripheral lesions; traumata; lesion of non-nervous organs; hysteria; malnutrition.

Gowers has analyzed fourteen hundred and fifty cases, with this result:

Under ten years	422 cases.
From ten to nineteen years . . .	665 "
From twenty to twenty-nine years	224 "
From thirty to thirty-nine years,	87 "
From forty to forty-nine years . .	31 cases.
From fifty to fifty-nine years . .	16 "
From sixty to sixty-nine years . .	4 "
From seventy to seventy-nine years	1 case.

In this connection I desire to call attention to the fact that many cases of idiopathic epilepsy will begin with a fit in early infancy, after which no convulsion will occur for years, when the attacks will again appear and the case will become one of well-marked epilepsy.

In Gowers's fourteen hundred and fifty cases the percentage of females was 53.4, and of males 46.6 : 36.6 per cent. were of hereditary origin.

The association of migraine with epilepsy has been already mentioned, as has also the connection with organic brain, spinal, and peripheral nerve-lesions and traumata.

In some rare cases epilepsy may be caused by diseases of the heart, of the kidneys, rarely of the lungs, and very rarely indeed of the genitalia.

The association of epilepsy with hysteria is by no means infrequent.

I have seen three cases in which profound malnutrition seems to have been the cause of epilepsy, for when the general health was perfectly restored the epilepsy disappeared, and in one case it has not returned for seven years, in another case for six, and in another case for four, whilst in none of them was there any hereditary predisposition.

Pathological Anatomy.—As I have already said, epilepsy must be regarded as a symptom ; and it may be divided into two great classes,—viz. : 1. Epilepsy from recognizable organic disease ; 2. Epilepsy which we may call idiopathic.

The organic lesions which are capable of causing epilepsy are tumors of the brain and cord ; meningitis, either tubercular, cerebro-spinal, suppurative from disease of the ear, or by metastasis from other organs ; the different forms of myelitis ; the cerebral palsies of childhood, such as hemiplegia, double hemiplegia (diplegia), or paraplegia, and due to such lesions as porencephalitis, embolism or hemorrhage from the cerebral arteries, thrombosis of the cerebral arteries or veins, etc.

The pathology of the idiopathic forms is imperfectly understood at the present day. The old theory of vaso-motor spasm preceding cerebral anæmia or hyperæmia is now almost entirely discarded by neurologists, as it ought to have been long ago, for there has absolutely never been any logical proof of it whatsoever. It is doubtless true that ligature of the carotid artery and great hemorrhage are capable of producing epilepsy, but that the profound ischæmia produced by them was present in every case of epilepsy has never been shown, whilst there are many experiments and facts to prove that the cerebrum of epileptic human beings and animals is not vascularly altered to any marked degree. Vulpian has examined the brains of guinea-pigs artificially rendered epileptic, and has found that they are neither hyperæmic nor ischæmic during the epileptic attack. The deductions as to the cerebral circulation that would be drawn by certain authors from the facial circulation are not warrantable, inasmuch as the latter is by no means an index to the former, for it often happens, especially in cerebral traumata, that the face may be deathly pale at the same time that the cerebral meninges are in a condition of profound hyperæmia. The same remark also applies to the proof that would be drawn from the condition of the circulation in the fundus oculi. In some cases of idiopathic epilepsy a sclerotic condition will be found in the brain, in others some old focus of limited meningitis, especially of the pia mater, whilst in many no lesions are visible. Great stress has been laid upon sclerosis of the cornu Ammonis, but this find has by no means been constant, and it is exceedingly questionable whether it is not a mere coincidence or an effect rather than a cause. Barthez and Rilliet have laid great stress upon a peculiar variety of sclerosis to which they have given the name of tuberous or hypertrophic, and in which the convolutions are found elevated, studded with round or ovoid tuberosities irregularly disposed upon the convex portions of the convolutions, rarely in the fissures, the size varying, but sometimes attaining that of a large nut. These nuclei do not affect the white matter, the gray matter of the cortex, or the central nuclei, the basal nuclei being alone affected. M. Chaslin has recently, at the instance of M. Féré, examined five epileptic brains, and in four of them he has found a condition for which he suggests the name of neurogliar sclerosis. In this condition the convolutions are macroscopically shrivelled, small, hard, smooth, or slightly roughened, without adhesion of the pia mater, and otherwise normal, the pathological alteration extending in a very variable manner over the surface of the cerebrum, with large intervening normal portions, and sometimes reaching to the medulla oblongata, to the cornua, or to the cornu Ammonis. In one instance the alteration was found only in one of the olivary bodies. Microscopically the fundamental lesion is said to have been due to the presence of a number of rough fibrillæ of an uncertain length which had invaded the cerebral tissue, especially the gray cortex. In the author's own words, "In the normal state the first layer of the gray cortex contains certain so-called spider-cells, whose prolongations are scarcely visible. In this condition, on the contrary, this first layer is formed by a bundle of fibrils arranged nearly parallel to the surface of the cerebrum, and it can be distinctly seen to originate from numerous cells with hypertrophied prolongations. In the preparation which I am viewing at this moment there is a place where this transformation invades all the layers, but leaves intact numerous nerve-cells and vessels. It can be seen, moreover, that these fibrillæ in a certain space form in the depth of the cortex a net-work of nodal points, in which lie the cells of the neuroglia. Finally, and I would call attention particularly to this fact, this first layer is studded in places by large compact bundles, which are evidently formed from these fibrillæ. I would observe, in passing, that the vessels which remain do not present a trace of inflammation, there being simply, in certain points, a hyaline transformation of the capillary wall." M. Chaslin is convinced, following the views of Ranvier, that a distinction should be made between the connective tissue of mesodermic origin and the neuroglia, which is of epithelial or ectodermic origin, the latter embracing the Müller's fibres of the retina, the fibres and cells of the neuroglia in the spinal cord, and the slightly differentiated prolongations of the spider-cells in the brain; and this neurogliar sclerosis, which he describes for the first time, he believes to be entirely distinct from a sclerosis of the connective or mesodermic tissue, for he asserts that the peculiar fibrillæ of this pathological condition distinctly emanate from the neuroglia-cells, whilst the non-adherence of the pia mater and the relative integrity of the vessels are indirect proof in the same direction. Moreover, he has been able to adduce a histo-chemical proof. These fibrillæ and bundles resist, in sections made after bichromate immersion, the successive action of potassa solution of forty per cent. for ten minutes, as well as washing with water and concentrated acetic acid, whilst they remain colored red by the picrocarmine used after washing in water, and they can be preserved thus in formic glycerin. On the other hand, the connective tissue treated in this manner swells and decolorizes, so that a section of the spinal cord thus treated showed the pia mater swollen and decolorized, whilst the neuroglia was intact. In addition, after the action of alcohol diluted to one-third, these fibrillæ remained colored by the carmine when subjected to acetic acid, whilst all other forms of connective tissue of the body were decolorized. This pathological description is so definitely and judiciously given that it merits attention. Of the five brains that were examined, four were found to be in this condition of neurogliar sclerosis, whilst the fifth was apparently unaffected. The author would have us believe that this neurogliar sclerosis is the condition that has been found in the cornu Ammonis and in the olivary bodies in some cases of epilepsy.

Nor is our knowledge of the *modus operandi* of epilepsy much more definite than our knowledge of the exact pathology. The older theories have been that the medulla oblongata was the portion of the nervous system most concerned in the production of symptoms. Van der Kolk believed that the nuclei of origin in the floor of the fourth ventricle of the hypoglossal nerve were always in a condition of induration, this induration being in proportion to the amount of tongue-biting that was present in the attacks. But this was the airiest of theories, and is scarcely worthy of mention, much less of sober discussion. Then Nothnagel asserted that there was a certain area in the floor of the fourth ventricle, which he called the convulsive centre, irritation of which in some unknown manner was capable of causing epileptic convulsions. This simply added another fact to our knowledge of the different causative lesions of epilepsy. But of late years, as our knowledge of the functions of the cortex of the brain has received so tremendous an impulse from the experiments of Fritsch and Hitzig, facts have accumulated tending to demonstrate more and more strongly that epilepsy in at least a large number of cases, if not in all, is due to direct or indirect excitation of the cortex or of the nerve-strands leading from the cortex to the peripheral structures. It has been shown again and again that electrical or mechanical irritation of the motor centres can cause convulsive seizures of tonic and clonic nature, with loss of consciousness, and it has also been shown that the same effects can be produced by the same irritation of the nerve-strands underlying the cortex. Duret has produced the most wide-spread convulsions by irritating the cerebral membranes, more especially the dura mater. Innumerable lesions of the cerebrum, both of the cortex and the underlying white strands, have brought proof in the same direction. But we must also remember that epilepsy can be produced by such extracranial lesions as diseases of the spinal cord, of the peripheral nervous system, and of many of the non-nervous viscera, by alterations in the blood, such as are to be found in albuminuria, and possibly by malnutrition, and by the action of many toxic agents and various febrile conditions: so that it would be a matter of large assumption to say that in all these various conditions the cortex of the brain must be the part affected. The truth probably is that the epileptic manifestations are due to a peculiar molecular condition of the motor tract which proceeds from the motor convolutions to the peripheral motor structures, the muscles. What this particular molecular condition is we do not know, any more than we know what is the peculiar molecular condition producing chorea, or neuralgia, or tetanus, or hysteria, or any one of the different functional nervous diseases, and we probably never shall know until the time arrives when we shall possess such instruments of precision as will enable us to see the molecular play in a living brain and spinal cord, or until we shall have so advanced in our methods of preparation and staining of the nervous system that we can detect such slight cellular alterations as have thus far entirely eluded our vision. The metaphysical theories are puerile that would explain these cellular alterations in the present condition of our knowledge. Whatever this altered molecular condition may be, there can be no question that it finds expression in epilepsy through the motor tract proceeding from the motor convolutions to the motor structures, the muscles of the periphery. When muscles are convulsed, they can be convulsed only by direct excitation of the muscle itself or of the motor tract leading from the muscle up to the motor convolutions. But some varieties of epilepsy are evidently due to an excitation that extends into this motor tract from some part of the nervous system without it. For instance, some cases are attended with such symptoms as hemianopsia, or word-deafness, or aphasia, indicating a lesion in the corresponding centres of the cortex, and autopsies have demonstrated the correctness of this view; whilst the epileptic convulsions that are observed from lesions of non-nervous organs, diathetic conditions, and the action of toxic agents must be from indirect implication of this motor tract.

Idiopathic epilepsy, therefore, is a neurosis, like neuralgia, migraine, hysteria, and chorea, and it is exceedingly questionable whether the sclerotic changes that have been found are not effects rather than causes.

Diagnosis.—In the diagnosis of epilepsy it must always be borne in mind that epilepsy is a symptom, and the question of diagnosis is simply a question of what the epilepsy is a symptom. The question must therefore always be asked whether it is a symptom of any organic disease, such as lesions of the brain, of the spinal cord, or of the peripheral nerves; whether it accompanies the cerebral palsies of childhood; or whether it bears a distinct relationship to heart-disease, or nephritis, or profound malnutrition.

After all these organic lesions have been excluded, the epilepsy may be said to be idiopathic. Its diagnosis should not then be difficult. The attacks consist of unconsciousness, with tonic or clonic convulsions, or simply of loss or impairment of consciousness, without convulsions, or with very slight ones, needing only to be differentiated from hysteria and simulation.

True hysterical attacks do not have the same sharp loss of consciousness that occurs in epilepsy, and hysterics do not, therefore, fall and bruise themselves or bite the tongue as do epileptics; moreover, the movements in hysteria are more evidently volitional and of wide range, so that the patient rolls and turns herself in bed or assumes various attitudes, whilst in epilepsy the muscular movements are limited in range, consisting only of limited movements of flexion and extension, or of simple rigidity. But it should not be forgotten that true epilepsy may be conjoined with true hysteria, in the so-called hystero-epilepsy.

Simulation of epilepsy is extremely rare. When the simulation is artistically done, as I have seen it by a certain professional thief, who is known to his companions as a "dummy chucker," the detection will not be possible except by close observation, when the unchanged color in the face, the undilated pupils, and the lack of the long sleep afterwards, together with the tendency to overdo the clonicity or the tonicity of the convulsions, will usually lead to detection.

Prognosis.—At the outset of the question of prognosis arises the question as to what shall be considered a cure. Cases have been known to go without an attack for ten, fifteen, or even twenty years, whilst it is not seldom, as I have already said, that a convulsion in infancy will not be succeeded by another for ten or fifteen years, when a whole series may set in. Under these circumstances we must feel a profound uncertainty as to what period of time of freedom from fits shall constitute a cure. In favorable cases immunity from the attacks may be obtained for several years, even as much as six or seven years, as I have known in my own practice. In less favorable cases the violence and the frequency of the attacks may be greatly lessened, so that the life of the patient is made much pleasanter and more useful, and the tendency to mental deterioration is decreased. In some cases, however, the attack cannot be affected by any means at our command. As a matter of fact, most cases of epilepsy of the *grand mal* type can be improved, but only a few of them can be cured in the sense which I have explained. As to which cases can be improved and which cannot, I know no other criterion than the effect of treatment for a month or two. Idiopathic cases which respond promptly to treatment during this period of time will usually continue to do well, but time alone will show as to how long they can be kept without their attacks. The sad fact should always be explained to the patient's relatives that they must make up their minds to continuous treatment for years to attain even this result. The cases of *petit mal* are almost always intractable. I have never yet seen a case materially affected by treatment. Cases of hystero-epilepsy are usually of excellent prognosis, if the patient can be brought under proper control and treatment. The cases which are associated with migraine are usually also of excellent prognosis so far as great improvement goes. The cases of peripheral origin usually improve very greatly, although there is always, in my experience, a lurking convulsive tendency.

Treatment.—The treatment will depend in some degree upon the variety of the epilepsy. Cases of epilepsy caused by intracranial lesions whose location can be determined by our present knowledge of localization should be operated upon, if it be possible to reach the locality affected. The surgeon can easily reach the cortex of the cerebrum and the cerebellum, the centrum ovale of the cerebrum, and all of the subcortical tissue of the cerebellum, and in a certain number of instances our present knowledge of cerebral and cerebellar localization will be a reliable guide to him. But regard must also be had to the nature of the lesion before any surgical procedure is attempted. It would be folly to operate in the hemiplegias, single or double, and in the paraplegias constituting the so-called cerebral palsies of childhood, as they are due to the cerebral losses of substance known as porencephalitis, to hemorrhage, to arterial or venous thrombosis, or to sclerosis or encephalitis, which, if not always, are generally the result of preceding arterial trouble, while it is seldom possible to diagnosticate the exact causative lesion. A proper case of tumor should always be operated upon, although cerebral tumors of children are by no means so easy to diagnosticate as are those of adults. Still, when cephalalgia, cerebral vomiting, neuro-retinitis, and the progressive character of the symptoms are such as to warrant a diagnosis of a cerebral tumor that can be localized in an accessible area, an operation is not only warrantable but called for. Where the epilepsy is evidently due to suppurative trouble extending inward from the ear and causing symptoms of abscess in the temporal lobe, an operation is eminently practicable, and has been done in several instances with great success. When epilepsy has followed a cerebral trauma, the scalp should be carefully shaved, and a cicatrix or depression of the skull searched for, and, if either is detected, an exploratory trephining should always be done, for in several instances of this kind a very slight adhesion of the membranes has been found and removed with good results, or a hæmatoma or depression of the inner table has been discovered. Under antiseptic precautions the trephining is a harmless procedure, and may disclose conditions that could not have been recognized without it.

In a case of epilepsy occurring in a child with some peripheral irritant, the latter should always be removed, although such removal may cause no more than temporary improvement; for even this may be a valuable adjuvant to the treatment with drugs. Any phimosis or adherent prepuce should always be rectified. An irritable clitoris should always be treated, either by soothing applications, or by carefully applying with a camel's-hair brush a sixty-per-cent. solution of nitrate of silver, the applications being made as often as may be necessary. If there is a vaginitis, this should be carefully treated. If there are marked errors of ocular refraction, these should be rectified; but I have no faith whatever in the curative or even palliative effect of removal of insufficiency of ocular muscles. If there is a laryngeal tumor, giving rise to the symptoms of so-called *laryngeal vertigo* or *epilepsy,* this tumor should be removed. A neuroma should be excised. But in all these cases the operation should not be trusted to alone, as the epileptic tendency, once established, as I have said, is very difficult to eradicate.

In cases of epilepsy conjoined with hysteria, the hysterical tendency should be taken fully into account. Too many hysterical children manifesting epileptic tendencies are petted and spoiled by their parents, until they come to have far less than the usual feeble self-control of childhood, and in many instances they become perfect savages. All this should be carefully and firmly met, either by the aid of a trained nurse, or by the thousand methods that will vary in every individual case according to the tact and good sense of the physician and the parents. It is surprising in many instances to see the effect of such moral treatment upon the epileptic manifestations of this hysterical class.

If migraine is associated with the epilepsy, the former should be carefully treated. The best drug with which to do this is a reliable preparation of cannabis indica, preferably the solid extract, beginning with doses of one-tenth of a grain, and gradually increasing to one-third or one-fourth or even one-half grain, according to the idiosyncrasy of the patient. Whatever the quantity, it should be administered three times a day, and its administration should be continued for months. At the same time the epilepsy itself should be treated, in the manner that will be described. In these cases of associated migraine, attention should always be paid, also, to the condition of the digestive organs. In cases of coated tongue or foul breath the administration occasionally of one-twentieth of a grain of calomel every hour for five consecutive hours will be found useful, and should be followed some twelve hours subsequently by a moderate saline laxative; whilst five-minim doses of dilute nitromuriatic acid in a half-wineglassful of water before meals, or, if this does not agree with the child, a wineglassful of Vichy or Giesshuebler water, should be given three times a day for a week or two. Constipation should always be overcome by some gentle laxative, one of the best of which is freshly-calcined magnesia, of which chocolate cakes are now made by many pharmacists.

Every case should be carefully interrogated or observed to determine whether there is any marked periodicity in the return of the symptoms, and at these periods the child should always be kept quiet and special attention should be paid to the medication. In many instances where the attacks return during a certain period that may not extend over a week or a few days, I put the child to bed at this time, or I add to the medication or increase the quantity of the drug which I am administering. In nocturnal cases it may only be necessary to administer the drug at bedtime. The possibility should always be borne in mind, however, of these epileptic attacks being diverted from their usual period of time, breaking out at some other, so that a nocturnal attack may come during the daytime, or at an unusual period of the month. In such cases it will become necessary to keep up continuous treatment, simply paying special attention, in the manner indicated, to the periods.

There is no question whatsoever that the bromides constitute the most valuable means of treatment at our disposal. They should be given in as large doses as may be necessary, unless the idiosyncrasy of the patient prohibits them. Most epileptic children bear remarkably well all the bromide that it may be necessary to administer, but there are occasional exceptions. I have repeatedly given as much as one ounce of the bromide of potassium at a dose without any ill effect, whilst in other cases I have seen collapse produced by a dose of ten grains. Occasionally, also, there are cases in which the bromide actually seems to aggravate the disease.

Bearing these considerations in mind, my routine treatment is as follows: I always begin with ten grains of the bromide of potassium, administered three times a day, unless the attack has a marked periodicity, when I administer the bromide at that time in proportionate doses. In the course of a week I increase the bromide to fifteen grains three times a day, unless the patient has begun to improve, or unless bromism manifests itself. If the patient does not improve, but bears the medicine well, I increase to as much as from thirty to forty grains thrice daily. If the patient manifests symptoms of bromism, but the epileptic manifestations do not improve, I conjoin with each dose of the bromide of potassium five grains of the bromide of sodium, as this combination of the bromides will often increase the effect upon the disease without increasing the constitutional effects of the drug. Having thus got the patient under some control, I keep on with the dose which I have reached. If the patient ceases to improve, or relapses, I make therapeutic use of one of the clinical characteristics of epilepsy which I have described,—viz., the impressionability of the disease by variations in the treatment and the environment. I can usually thus produce some effect upon the epileptic manifestations by a laxative, a brisk cathartic, a change of scene, or a combination with the bromide treatment, as already detailed, of borax, belladonna, or hyoscyamine. These adjuvant drugs I use for only a short time, gradually discontinuing their use, and I endeavor to employ them in one or two doses daily.

Great stress has been laid by some authors upon the loss of uvular reflex, evidenced by the ability to tickle the throat with a feather without causing the patient to gag, as an indication that sufficient bromide has been used. I have tested this again and again, and have seldom found it to be of any value, as I have known cases to grow worse when this reflex had been abolished, and other cases to improve although this reflex was not abolished, and others still to bear increasing doses of bromide although this reflex had ceased.

Of the bromides, however, I never make use of other than the bromide of potassium and the bromide of sodium, as the bromide of ammonium has proved perfectly worthless in my hands and in those of others, except that I have occasionally employed it as an adjuvant in the manner just described. The acne, of which so much has been made, has seldom been a serious factor in my cases, and has usually been overcome by the administration from time to time of moderate doses of arsenic, preferably in the form of Fowler's solution. It has been my experience that a case which will not improve under the bromide treatment will not improve at all, and the only exceptions to this rule which I have met are in some few cases that will do well with borax when the bromide has failed.

The method of administration of the bromide suggested by Dr. Gowers is occasionally very useful, especially when alternated with the modes of treatment that I have just detailed. Dr. Gowers describes his method as that of the maximum-dose treatment. He begins with doses of two or three drachms of bromide of potassium every second or third morning, and increases the dose to four drachms every fourth morning and six drachms or an ounce every fifth morning, these doses being given after breakfast in a tumblerful of water, for they may cause epigastric pain and vomiting if they are not well diluted. He does not increase the dose beyond that which produces transient lethargy and mental dulness. The susceptibility of different patients to these doses varies exceedingly, some being unable to bear more than four drachms, whilst in others, as I have myself seen, one ounce will produce no unpleasant symptom. The maximum dose should be reached in two or three weeks, and repeated three or four times, after which the doses should be gradually reduced, so that the whole course lasts six or seven weeks, when the patient may be left without treatment for several weeks, or even several months, although I have not seen the immunity of six or eight months that Dr. Gowers claims.

Belladonna has not proved of much use to me, except as an adjuvant in the manner described. The dose should be one or two minims of the fluid extract thrice daily, but the drug should be carefully watched. Borax, ten, twenty, or thirty grains three times a day, well diluted, is in some cases of as much efficacy as the bromide treatment, although it is not generally so. In every instance, however, in which the bromides fail in their effect, or in which they disagree with the patient, the borax treatment should be carefully tried, and it is the most valuable of all the adjuvants.

I have never been able to satisfy myself that other drugs than these mentioned are of any value in epilepsy.

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TETANUS.

BY F. X. DERCUM, M.D.

TETANUS (French, Tétanos; German, Starrkrampf) may be defined as a disease which is characterized by the gradual onset of tonic spasm of the voluntary muscles, the spasm beginning, as a rule, in the muscles of mastication, spreading thence to the trunk and limbs, and being subject to irregularly recurring exacerbations of short duration. No definition, however, can in itself be entirely satisfactory, and a detailed description of symptoms is here, as elsewhere, necessary to convey a proper conception of the clinical picture.

Tetanus has an exceedingly extensive literature, having been known from the earliest times. The majority of writers have dealt with the subject in general, and, with the exception of tetanus neonatorum, have not treated of its special relations to children. Childhood including, as it does, all the years from birth to puberty, tetanus has a full claim upon our attention. That tetanus occurs with considerable frequency among children, a brief study of statistics will readily show. In the valuable collection of cases made by Laurie[1] from the records of the Glasgow Infirmary, fifty in number, nine occurred among children. Of one hundred and seventy-one collected by him from various other sources, thirty-five occurred among children. Taking these cases all in all, the relative frequency amounts to nearly twenty per cent. Again, in the collection made by Poland[2] of cases occurring at Guy's Hospital from 1825 to 1857, seventy-two in number, sixteen, or twenty-two per cent., were in children. In the more recent collection by Taylor[3] of cases occurring at Guy's Hospital from the middle of 1866 to the end of 1877, fifty-one in number, twelve, or twenty-three and one-half per cent., were in children. It would be safe to infer, therefore, that the average percentage is about twenty-two. The statistics of Wallace[4] present a decidedly smaller proportion,—that is, excluding the cases of tetanus neonatorum. He reports, in all, two hundred and eighty cases, and from the ages of one to fifteen years twenty-eight cases occur. The significance of this low percentage is, however, impaired when we reflect that the cases collected were those admitted to the Medical College Hospital of Calcutta and included both natives and Europeans. Further, the statistics of Wallace present other anomalies, such as an excessive proportion of European children suffering from tetanus neonatorum as compared with the natives, and we are led to the conclusion that the peculiar social conditions obtaining in India markedly influence hospital admissions. In the Pennsylvania Hospital the experience has also been that a large proportion of the cases of tetanus has been in persons under fifteen years of age. Dr. Frederick A. Packard has collected from the hospital record and placed at my disposal thirty-two cases occurring since 1873. Of these, nine, or almost twenty-eight per cent., were in children under fifteen years of age. This does not include four cases in children, not recorded, but of which Dr. Packard has personal knowledge. To include them would, of course, make the proportion still greater.

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[1] Glasgow Medical Journal, 1853–54, i. 339.

[2] Ibid., 1857, iii. 1.

[3] Guy's Hospital Reports, 1878, Series III., xxiii. 339.

[4] Lancet, August 12, 1882.

Etiology.—Two classes of causes are here to be considered, predisposing and exciting. Among the predisposing factors are age, sex, race, climate, and season.

Under five years of age tetanus—excluding, of course, tetanus neonatorum—is excessively rare. Wallace records five cases (four natives and one European) from one to five years of age. In Packard's collection one instance of five years is recorded. Taking Laurie's collection,[1] we find first one of five and a quarter, next one of six, and then one of six and a half. At seven years the number of cases has already increased to two, at eight to four, and with some modifications the frequency of tetanus now increases as we approach the age of puberty, so that at fourteen years the number has reached eight and at fifteen ten. We find also that at fifteen the greatest frequency obtains of any year in the second decade, which, as Gowers has pointed out, is the period during which the greatest percentage of cases occurs. Further, the cases occurring in the first half of this decade outnumber those occurring in the second. In fine, if inferences drawn from hospital statistics be allowed, it would appear that tetanus is relatively frequent as we approach the years of puberty.

Regarding the relative frequency in the two sexes, we find that males predominate, though the difference is less marked than among adults. In Laurie's and Taylor's cases the proportion is as three to one, while in Poland's cases it is less than this. In adults, on the other hand, the preponderance of males is fully twice this amount. Doubtless the increased exposure of male adults to injury is an important factor in explaining this difference, but it does not altogether suffice, as the statistics fail to show that decided increase in the proportion of males in childhood as we approach the period of puberty which we should otherwise expect.

As is well known, negroes and dark-skinned races generally are especially liable to tetanus, and, though the evidence as regards children is not such as to make a statistical statement possible, it points in the same direction. Especially is this true of tetanus neonatorum.

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[1] Including his miscellaneous collection.

As regards climate, there can be no doubt that here, as in the case of adults, it acts as a powerful predisposing cause. Tetanus is far more frequent in hot countries, as is shown, for instance, by the enormous number of cases recorded by Wallace (*loc. cit.*). From 1869 to 1879, a space of ten years, there were two hundred and eighty cases of tetanus in the Calcutta Hospital. When we compare this with the fact that in the Pennsylvania Hospital there were but thirty-six cases in seventeen years, the overwhelming influence of climate becomes very apparent.

The season of the year also seems to play a part. In Poland's cases the greatest number occurred in the month of June and the least number in February. In Laurie's cases, on the other hand, the greatest number occurred in October and the least in August. Strangely enough, the maximum number of Wallace's cases occurred in December, the minimum in July. These cases are obviously too few upon which to base a generalization, but the probability is that very changeable seasons—seasons associated with extremes of heat and moisture—favor the development of tetanus. The prevailing impression in this country, that summer is especially the season of tetanus, is justified by the records of the Pennsylvania Hospital, the larger number of cases occurring in June and July. In temperate climates, however, season does not seem to be as potent a factor as in hot climates. The absolute locality makes little difference, as the disease is met with in mountain, valley, and plain, inland and on the coast, in moist and in dry places.

Lastly, the previous condition of health of the patient appears to exert no influence.

Among the exciting causes of tetanus two especially are recognized,—trauma and cold. Those resulting from trauma are by far the most frequent and most dangerous. The injuries may be of the most diverse character, though they all agree in almost always—if, indeed, not invariably—involving a lesion of the external integument. Bruises, burns, crushes, cuts, tears, bites, and especially punctured wounds are among the traumata found. As regards children, there is nothing that distinguishes the wounds from those occurring in the adult. We find, however, that in the child the parts most frequently injured are the foot and leg, while in the adult they are the fingers and hand. Indeed, the proportion of injuries of the hand and fingers is very small in children. Doubtless the fact that the feet of children of the class who apply to the hospitals for relief are frequently altogether unprotected, and also the fact that in the adult the risk of injury to the hands through manual labor is much increased, largely explain this difference. Further, it is interesting to note that in the tetanus of children wounds of the head are rarely the exciting causes, while in adults they are far from uncommon.

The wound may possess almost any character. It may vary from an insignificant bruise to a frightful laceration, and may result from the most diverse accidents. In looking over the various collections of cases, however, we soon recognize that punctured wounds and wounds involving violent disruption or dissolution of tissue are by far the most frequent. Cuts and incised wounds generally are very rarely the exciting causes of tetanus. For instance, among all the cases under fifteen years reported by Laurie, Poland, and Taylor, we find cuts recorded but three times, while punctured wounds, lacerations, and burns occurred some fifty times.

Further, it does not depend upon the kind of tissue that is wounded. The idea that wounds of tendons are most frequently related to tetanus falls to the ground when we reflect that the parts most frequently exposed to injury, the hands and feet, are necessarily largely tendinous in their make-up, and also that wounds of the trunk, such as burns and scalds, not involving tendons, are disproportionately frequent. The same reasoning applies to the involvement of nerves.

The wound itself may be in almost any condition. It may, on the one hand, present a healthy appearance and be actively engaged in the process of healing or may even be completely healed; or, on the other hand, it may be suppurating, foul, and even gangrenous. According to Thamhayn, the wound at the time of the appearance of symptoms is most frequently in the stage of cicatrization. It is rather the exception, he states, to find the wound badly constituted. Watson, on the other hand, makes the significant observation that tetanus is more apt to occur with dirty wounds and in abscesses with suppressed suppuration than after fresh and clean injuries.

Among the exciting causes—doubtful in value, perhaps, but mentioned by various writers—are alcoholic intoxication, over-exposure to the sun, violent emotion, inflammation of serous membranes, and, lastly, worms. How it is possible that these various causes may become active factors in the production of tetanus will be discussed under the head of pathology.

Symptomatology.—As in other diseases, there is in tetanus a period of quiescence intervening between the exposure to the exciting cause and the appearance of symptoms. In the vast majority of cases it lasts several days or even weeks, and suggests very strongly a period of incubation. A few cases, like that of Robison cited by Gowers, present an almost incredibly short period, the symptoms coming on after the lapse of a few minutes. Whether in these cases earlier factors had been at work, so as to make this tetanic explosion possible, cannot be determined. In view, however, of the recent remarkable advances in the pathology of tetanus, a suspicion to that effect is strongly justified. We all know how careless and unobservant the average man, especially the hospital patient, is with regard to events bearing immediately upon his disease, and how readily minute and insignificant traumata are forgotten. At any rate, the fact remains that tetanus makes its appearance in the average case from four or five days to two weeks after the inception of an injury, and this time does not vary for children. In striking an average of the time in Laurie's cases, both for ages under and ages above fifteen, we find it to be about eight days for the former and nine for the latter. The difference of one day is certainly without significance. We find, however, slightly less variation in the former, the shortest time being two days and the longest eight, while for adults the shortest was one and the longest sixteen. This is also probably without special significance. Occasionally excessively long periods are reported,—periods extending over several months,—but the probability is that in these cases tetanus does not arise from the original trauma, but from some secondary complication, such as bed-sore, or from exposure to cold.

Preceding the onset of the muscular spasm, the patient is apt to be much depressed. He is restless and ill at ease, anxious and fearful. He loses his appetite, feels chilly and thirsty, and at times fever is present. He may remain in this condition several hours or even several days. Finally a feeling of stiffness is noticed in the jaws and in the back of the neck. Pains variable in character accompany this sense of stiffness, and are also referred to various other portions of the body. The muscles of mastication and of the back of the neck are the ones in which these sensations, as well as actual spasm, first make their appearance, though this rule is not invariable. Occasionally the muscles of deglutition are first involved, and the patient notices an uncomfortable sensation in swallowing or perhaps a difficulty in accomplishing the act. Less frequently the sterno-mastoids are the first, or among the first, to be involved, and may even present the condition of unilateral spasm. More rarely still, truncal muscles, or the muscles of the extremities or of the part which is the seat of the trauma, are the earliest to exhibit symptoms. The pains vary greatly. There may, indeed, be nothing more than a vague, unaccustomed sense of discomfort. On the other hand, they may be very intense, and may rapidly increase as the disease advances Frequently they are referred to the head and temples, the back of the neck, and the jaws.

Gradually marked spasm of the muscles of mastication makes its appearance. The patient now notices a distinct difficulty in separating the jaws. This becomes more and more marked until the jaws become fixed in position, and the condition known as trismus, or "locked-jaw," is established. The masseters and temporals become excessively rigid, and from these muscles the rigidity spreads successively to the muscles of the back of the neck, the trunk, and the extremities. As the intensity of the spasm increases, the muscles become hard and board-like to the feel. This is especially true of the abdomen. Generally the spasm is accompanied by severe pain. The patient now assumes a striking and peculiar position. It is one of extreme extension. The head and trunk are bent forcibly backward, producing the condition known as opisthotonus. The expression of the face is also remarkable and characteristic, and when once seen is rarely forgotten. A repulsive grin, the "risus sardonicus," distorts the features. The lips are slightly parted, the upper lip being pressed against the teeth and the lower slightly everted, while the angles of the mouth are forcibly extended and depressed. At the same time the brow is raised and corrugated, and the eyes are fixed and seemingly sunken in their sockets. The expression as a whole is that of a hideous grimace, horrible to look upon, and suggestive of the most intense suffering.

The opisthotonus and the risus sardonicus are of course assumed involuntarily, and are merely the result of the overaction of the dominant muscular groups. However, in exceptional cases special muscular groups are alone or excessively involved, so that the average position is departed from. Thus, at times, instead of the head and trunk being bent backward, the body is simply rigid and *straight,* the condition being then termed orthotonus. More rarely, it is said, the body is bent forward, producing emprosthotonus, or to one side, producing pleurothotonus. It has never been the fortune of the writer to observe either of the last-mentioned positions. Gross irregularities in the order and degree of muscular involvement, however, doubtless occur, and we should remember that great extremes may be met with. It is stated, for instance, that the masseters and temporals may be so little involved that the depressor muscles of the jaw may determine the mouth being held wide open instead of rigidly closed (Thamhayn, Gowers); but certainly this condition must be excessively rare. Lastly, the symptoms may be imperfectly developed, the spasm beginning in the regular order, but ceasing after a time to spread, or spreading with extreme slowness.

A special form of tetanus, which should be mentioned, is the cephalic tetanus of Rose. Its chief peculiarity is that, associated with spasm of the muscles of deglutition and of the muscles of the jaws, there is a complete paralysis of the facial nerve on one side. In these cases the wound is found upon the face or head. In eight cases collected by Bernhardt[1] the wound was in the immediate neighborhood of the orbit in six cases, upon the eye in one case, and upon the temple in another. The paralysis of the facial nerve is always upon the side of the wound. The cause of this paralysis is not known, but it is probably reflex in origin. No lesion of the nerve has been found. In an autopsy by Lannois[2] the nerve was carefully dissected out, but failed to reveal any change to either the naked eye or the microscope. Betz[3] collected, including the cases of Bernhardt, sixteen cases. These were distributed as follows. Nine were adult males, two middle-aged women, one a girl of eighteen, and another a boy of ten. In three the ages were not stated. Twelve of the entire number proved fatal, among them being the boy. It should be stated that the dysphagia is generally very marked, and that this form of tetanus has in consequence also been termed hydrophobic tetanus.

The condition of tonic spasm having been established in a given case, a new feature, an irregularly-recurring paroxysm of exacerbation, makes its appearance. The contraction of the muscles is suddenly intensified, and the opisthotonus and risus sardonicus become accentuated. The paroxysm may be very slight, causing but little change in the patient's position and appearance, or it may be so violent as to raise the patient from the bed by the intensity of the opisthotonus produced. At times, indeed, the legs become as rigid as though all their joints were ankylosed, and as though they were of one piece with the trunk. The arms may also be involved, especially the upper arm, but to a less extent than the legs. The forearms and hands are affected, if at all, but slightly. Occasionally the paroxysm, instead of being evenly diffused over the muscles, is concentrated or especially marked in certain groups. Again, instead of being a wave of intense tonic contraction, it may at times be somewhat convulsive or clonic in character.

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[1] Zeitschrift f. Klin. Med., Berlin, 1883–84, vii. 410–424.

[2] Lyon Méd., 1889, lxii. 380.

[3] Memorabilien, Heilbr., 1885, N. F., v. 7.

In duration the paroxysm varies from a few seconds to a fraction of a minute,—rarely longer. Occasionally, however, the attacks recur so frequently that a number of them may be merged together and the apparent duration much increased. If the seizure be severe, the thorax and diaphragm may become absolutely fixed, and complete arrest of respiration may ensue. The face grows livid and purple, and the patient may, indeed, die during the paroxysm.

During the attack the pain in the muscles is much increased, while pain often agonizing in character makes its appearance in the abdomen and back. At times it seems focussed in the epigastrium, or is especially intense between the shoulder-blades, radiating thence about the trunk or even the limbs. It is stabbing, darting, or shooting in character, or it may be replaced by an insufferable sense of constriction. It may, indeed, be so severe that at the on-coming of a paroxysm the patient cries out aloud, and continues to do so as best he may until the fixation of the muscles of respiration makes screaming impossible.

The seizures recur with varying frequency. They may be few and far between, or even, in rare and very mild cases, entirely absent. On the other hand, they may be so frequent that the patient is for hours in an almost continuous paroxysm. Slight causes, such as handling the patient, a draught of air, and especially efforts at voluntary movement, such as attempting to rise up in bed, or even the act of swallowing, may suffice to provoke them. The reflex excitability, both superficial and deep, is enormously increased. Of course this is also the case during the intervals, though to a less extent.

As may be inferred from what has been thus far stated, deglutition and breathing are much interfered with, and sometimes this interference is exceedingly grave. It may be impossible, for instance, to administer food. Every attempt may provoke a paroxysm, accompanied by regurgitation and by strangling. The breathing may be exceedingly shallow and rapid, and may during a seizure cease altogether. At other times it is interrupted by hoarse sounds, the result of ineffectual attempts at speaking. In mild cases, on the other hand, the breathing may during the intervals be almost normal in depth and frequency. The pulse, as a rule, is small and rapid, especially during the paroxysm.

The excretory function of the skin is much increased, sweating, especially during and after the paroxysm, being excessive. The temperature is very variable: at times there is no noticeable departure from the normal, but not infrequently there is a rise to from 101° to 102° F. or more. This rise is generally maintained throughout, with or without remissions. Sometimes there is an especial rise just preceding the termination in fatal cases, and this rise may be extraordinary. Temperatures of 108°, 110° F., and even higher, have been recorded, and the rise has continued post mortem. The fever in tetanus is altogether independent of septic, inflammatory, or other complications. It appears to be purely nervous in origin, and probably arises from a rapid and progressive paralysis of the heat-centres. It is not improbable that in the cases of extremely high temperature there is really an intrinsic heat-stroke.

The urine is diminished in quantity and high-colored. Micturition is frequently accomplished with difficulty, and occasionally the catheter alone is efficacious. The bowels are, as a rule, obstinately constipated. In the first place, the patient takes very little food, and, secondly, the act of defecation is rendered difficult by the participation of the external sphincter in the general spasm. The muscular coat of the bowel as well as that of the bladder is not involved, the spasm appearing to affect the voluntary muscles only.

The appetite is generally lost, though this loss may be in part only apparent, as the patient, knowing the pain and consequences of efforts at swallowing, may refuse food on this account. Thirst, on the other hand, is occasionally intense, brought about, no doubt, by the excessive sweating. The tongue, when it can be seen, is generally found coated.

The mental faculties are undisturbed, the mind being clear throughout. Delirium or confusion is not present, unless it be perhaps in cases where narcotics have been freely used or towards the termination of fatal cases when exhaustion has supervened. Sleeplessness and restlessness are constant factors. Spontaneous sleep is infrequent and of short duration.

In lethal cases the symptoms persist until the patient dies of exhaustion. This may occur gradually, or quite suddenly after a severe and prolonged paroxysm, when heart-failure appears to be the immediate cause. At other times cyanosis, induced either by spasm of the glottis or by the general fixation of the respiratory muscles, is the most prominent factor. Intercurrent troubles, connected with or independent of the wound, bed-sores in late cases, etc., may also play a part in bringing about or hastening the fatal result.

In cases that recover, the paroxysms gradually become less frequent, less intense, and of shorter duration. Little by little, general muscular resolution sets in; but the disappearance of the spasm does not occur in all the muscles at the same time, nor even symmetrically. Soon there is an increased ability to take food, and then follows a gradual return of strength. Gradually the spasm becomes less and less marked, and finally disappears entirely, or a slight stiffness may persist in the muscles for some time and may even be noticeable in the gait.

TETANUS NEONATORUM.—There can be no longer any doubt that the tetanus of new-born children is identical with that of adults. The experiments of Beumer and of Peiper (see section on Pathology) are conclusive. However, a brief special mention of the subject is warranted. Like ordinary tetanus, it has an exceedingly wide geographical distribution. It is known not only in India, the West Indies, and the Southern States of America, but also in the mountains of Switzerland, the highlands of Scotland, and the Arctic cold of Iceland; but, in whatever situation it be found, it is most frequently associated with filth and ignorance. Among the most common etiological factors are irritation and inflammation of the umbilical cord, injuries to the head or other portions of the body during birth, circumcision, cold, and dampness. Whether any value whatever should be attached to the assertion made by Sims and his followers as to displacement of the occipital bone, is exceedingly doubtful. In the first place, as admitted by Sims, the condition is not uniform, the bone being sometimes displaced inwardly and sometimes outwardly; further, it is exceedingly difficult, in the light cast lately upon the pathology of the disease, to imagine how treatment by change of position alone could affect the progress of the disease; and, lastly, it is very evident that some at least of the cases cited were not cases of tetanus neonatorum at all, but of some other affection.

Tetanus neonatorum comes on generally from the fourth to the eighth day, occasionally, however, not until some days later. Niemeyer makes the significant observation that it never occurs except between the first and the fifth day after the fall of the navel-string. The child at first is restless, cries out during its sleep, and looks much distressed. It soon refuses to take or becomes incapable of taking the breast. The nipple, if seized, cannot be retained, and the milk is regurgitated, there being marked difficulty in swallowing. On attempting to insert the finger into the mouth of the patient, we find that the jaws, though not absolutely closed, are more or less fixed. The face, also, is now seen to be involved. The mouth is puckered, the lips being slightly depressed. The eyes are closed, apparently through spasm of the orbicularis, while the forehead is thrown into wrinkles. The countenance as a whole looks drawn; it is pale or even bluish in color. In many cases the trismus predominates very largely over the other symptoms, and sometimes the spasm fails to spread to the trunk and limbs. However, the general involvement may be excessive, and the limbs may be more rigid than in ordinary tetanus. The reflex excitability is heightened, handling and attempts at feeding provoking paroxysms. The paroxysms are generally severe, though the spasm during the intervals is, as a rule, less intense than in other cases. Breathing is much impeded, the child sometimes dying of suffocation. Death supervenes, usually from exhaustion, in from a few hours to two or three days. The mortality is very great. Wallace reports thirty-four cases with twenty-nine deaths, but even this showing must be regarded as rather favorable. It should be stated that in many cases the bowels are constipated throughout. There is also now and then fever.

Pathology.—Until within recent years vague and abstruse theories were the only ones advanced. The absence of constant findings post mortem, the variable condition or even the entire absence of the wound, as well as the inconstancy of the other etiological factors, rendered an explanation exceedingly difficult. Heretofore the theory of the exclusively nervous origin of tetanus has been maintained, and at present even is held by Gowers. Too much, however, is left unexplained, and, indeed, the view of the exclusively nervous origin constitutes a mere begging of the question. It is supposed, for instance, that the nerve-cells are in such a condition that under slight peripheral irritation they expend their nerve-force with excessive rapidity,—explosively, as it were; or, as Gowers puts it, there is "a diminished resistance to the evolution of nerve-force by the cells." This certainly is not an answer to the question, but merely a restatement of the problem. The difficulty still remains as to how this condition of the motor apparatus is brought about.

Of late years most interesting discoveries have been made, the starting-point of which was an observation by Nicolaier[1] that the insertion of small particles of earth beneath the skin of mice, guinea-pigs, and rabbits produced symptoms in every way analogous to those of tetanus. Further, pus obtained from the wounds thus produced was frequently capable of communicating the disease by inoculation to other animals. In addition, microscopical examination revealed the presence of bacilli, and one of the forms appeared to be specific, though it was not isolated. Cultures of these bacilli, though impure, likewise proved capable of producing the disease in animals inoculated with them.

Rosenbach[2] confirmed the existence of the bacillus of Nicolaier in the pus obtained from the wound of a case of tetanus, as did also Bonome.[3] The latter obtained the bacillus from the decubitus of a patient who died of tetanus following a fracture of the vertebræ. Inoculation of animals with particles taken from the region of the sore resulted in tetanus. Injection into the veins resulted negatively. Brieger[4] made cultures of the bacilli, and obtained a number of poisonous ptomaines, which by inoculation provoked the same symptoms in animals as had followed the experiments of Nicolaier. He named the principal ptomaine tetanin. The others allied in character he named tetanotoxin, spasmotoxin, and spasmodin. Further, he also succeeded in isolating tetanin from the amputated forearm of a case of tetanus. Carle and Rattone, cited by Brieger, had also injected the contents of an acne pustule found upon a man dead two hours of tetanus into the sheaths of the sciatic nerves of rabbits, and succeeded in the majority of cases in provoking fatal tetanus. From these rabbits the disease was again communicable to others. Ferrari succeeded in producing tetanus in animals by inoculating them with blood and cerebro-spinal fluid of a woman who died of tetanus following ovariotomy. Vanni and Giarré also succeeded by using blood of tetanus alone.

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[1] Deutsche Med. Wochenschrift, 1884, No. 52.

[2] Archiv f. Klin. Chirurg., Berlin, 1886–87, xxxiv. 306–317.

[3] Giorn. di r. Accad. di Med. di Torino, 1886, Ser. III., xxxiv. 759.

[4] Deutsche Med. Wochenschr., 1887, xiii.

Further, the following interesting contributions to the subject have been made by Beumer.[1] He relates the case of a patient in whom a splinter received under the finger-nail while playing tenpins had caused fatal tetanus. Particles of this splinter inserted subcutaneously in animals likewise resulted in fatal tetanus. He also relates a similar case of a boy who acquired tetanus as a result of a punctured wound of the sole of the foot. In this instance small particles of stone collected from the place where the boy had last been playing produced tetanus in animals on inoculation. Beumer further experimentally proved the existence of the tetanus-bacillus in soil obtained from various depths, especially the upper layers, as well as in materials from various sources,—the sea-shore, the field, the garden, and even the dust of rooms. He also produced tetanus in animals by inoculating them with fragments removed from the navel of a child dead of tetanus neonatorum. Peiper[2] obtained similar results by inoculating animals with fragments of skin from cases of tetanus neonatorum.

Among those who have made successful inoculations from man to animals and from animals to animals should be mentioned Hochsinger,[3] Morisani,[4] Eiselberg,[5] and Giordano.[6] Almost all observers identified either in the blood or the pus of the wound the pin-headed, bristle-shaped bacillus of Nicolaier. Most successful of all, however, was Kitasato,[7] who succeeded in isolating the bacillus and producing absolutely pure cultures. Morphologically this germ proved identical with the bacillus of Nicolaier, and when inoculated it yielded results identical with those obtained by previous observers. Kitasato also demonstrated that its virulence is most persistent, undergoing, unlike other germs, no diminution in repeated cultures, and, also, that it is possessed of extraordinary vitality.

Verneuil,[8] who has given great attention to the subject of tetanus, concludes, from the study of a large number of cases, that traumatic tetanus occurs most frequently in persons who have to do directly or indirectly with horses, such as farm-laborers, stablemen, carmen, farriers, veterinary surgeons, etc., and also among gardeners, who deal largely with earth in the fertilization of which horse-manure has been used. There is, indeed, every reason to believe that the tetanus of man and that of the horse are identical diseases and dependent upon the same bacillus.

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[1] Berliner Klinische Wochenschrift, 1887, xxiv. 541, and Zeitschrift für Hygiene, 1888, iii.

[2] Centralblatt f. Klin. Med., Leipzig, 1887, viii. 777.

[3] Centralblatt f. Bacteriologie u. Parasitenkunde, Jena, 1887, ii. 145.

[4] Progresso Med., Napoli, 1887, i. 785.

[5] Wiener Klin. Wochenschr., 1888, i. 232 *et seq.*

[6] Centralblatt f. Bacteriologie u. Parasitenkunde, 1888, ii. 623.

[7] Deutsche Med. Wochenschr., 1889, xv. 635. For a detailed description of the bacillus the reader is referred to the original paper.

[8] Revue de Chirurgie, 1889, vii. 757, also Revue Scientifique, 1888, xli. 225.

Shakespeare[1] made subdural inoculations in animals after the manner of Pasteur, with matter from the brain and cord of a horse dead of tetanus, with the result of inducing tetanus in the animals experimented upon. His conclusions are very interesting. He maintains, first, that "traumatic tetanus of the horse and of the mule is at least sometimes, if not always, an infectious disease, transmissible to animals, and very probably to man. During the evolution of the affection a virus is developed and multiplies, which virus, injected into the cranial dura of another animal, produces the same infectious disease." Secondly, "This virus is found in the bulb and spinal marrow of the animal which is the bearer of the affection. Analogous to the rabic virus, its virulence can be increased by subdural inoculation in series ; like the rabic virus, it is susceptible of attenuation by desiccation in the air at a moderate temperature, and, like the rabic virus also, its effects are more intense when inoculated subdurally than when inoculated into the skin or in the muscles of the back." Finally, he believes that we may admit that tetanus observed in man is transmitted to him, directly or indirectly, by a domestic animal, and in particular by the horse. The last conclusion is open to objection, inasmuch as even Verneuil admits that there is a numerous class of cases in which no relation, direct or indirect, can be traced between the patient and any equine influence. Berger,[2] for instance, records six cases, in but four of which equine influence existed. Of the remaining two, one appeared to be an instance of contagion from man to man, while the other was probably directly of telluric origin.

Of great interest are the experiments of Rietsch[3] in this connection. He inoculated guinea-pigs with the dust of hay, with the result of producing tetanus in the animals so treated. From the guinea-pigs he inoculated an ass, which also died of typical tetanus. Giordano (*loc. cit.*) also inoculated animals with bits of straw taken from the locality in which his case of tetanus had received the initial injury, with results in every way similar to those of Rietsch. Lastly, Abadie[4] relates the circumstances of an epidemic occurring during the Franco-Prussian war at the village of Ardennes. All the available houses were filled with wounded, and it became necessary to improvise a hospital in the church. Straw was accordingly placed upon the church floor, and the wounded were lodged in the straw.

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[1] Transactions of the Ninth International Medical Congress, 1887, 373.

[2] La France Médicale, 1888, i. 866.

[3] Semaine Médicale, August 7, 1888.

[4] L'Union Médicale, 1888, Sér. 3e, xlvi. 893.

Several of these cases were attacked by tetanus in the course of a few days and succumbed, while of the wounded lodged in the houses of the village not one was affected.

A considerable number of instances of contagion from man to man are on record. One of the most remarkable experiences is that related by Amon.[1] He had attended and dressed a case of tetanus with compound fracture. The next day he had occasion to remove an adherent placenta from another patient. Eleven days later this woman also developed tetanus. Ball[2] records the following instance. The first patient was a case of tram-car injury, amputation at the shoulder, tetanus, and death. Six weeks later he operated for radical cure of hernia, the second patient being placed in a bed adjoining that in which the first had lain. This patient also died of tetanus. There had been no tetanus in the hospital for eight years previously. Adams[3] reports a similar instance. The first case was one of amputation of the toe; death from tetanus. The second was one of ligation of hemorrhoids, the patient being placed in the same room as that occupied by the first case; death from tetanus. Jacquinot[4] relates an epidemic of four cases occurring in the surgical wards of the children's hospital. Other instances might be cited. The following was furnished me by Dr. F. A. Packard. A lad was admitted to the surgical wards of the Pennsylvania Hospital suffering from compound fracture of the forearm. He soon developed tetanus. Another lad, who was convalescent from an extensive scalp-wound which had not yet entirely healed, was set as a watch over the first boy. In a few days, although previously doing well, the second boy also developed tetanus, from which he died.

The most important of the recent contributions to the literature of tetanus is undoubtedly that of Guelpa.[5] He points out that the bacillus of tetanus does not have a nidus in any organ. It is found, for instance, in the central nervous system in but five per cent. of the cases examined. In the pus, however, especially in that from the deeper portions of the wound, it is always found, and Brieger's discovery of the ptomaine tetanin shows how this soluble poison may be absorbed from the wound and thus produce tetanus. Further, Guelpa does not believe that rheumatic tetanus exists in the true sense of the word. Causes which determine rheumatism may favor the development of tetanus, but only as adjuvant causes,—that is to say, as causes which render the organism more apt to be influenced by the pathogenic microbe or by its secretions. It is certain, moreover, that many cases of tetanus are pronounced rheumatic because physicians do not know or are not able to find the avenue of entrance of the microbe.

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[1] Muench. Med. Wochenschrift, 1887, xxxiv. 427.

[2] Dublin Journal of Medical Science, 1887, Series III., lxxxiv. 457.

[3] Lancet, 1888, ii. 419.

[4] Revue Mensuelle des Maladies de l'Enfance, 1889, v. 508.

[5] Journal de Médecine de Paris, January 13 and 20, 1889.

There is, again, another method of penetration into the animal economy of the bacillus and its products, which Guelpa has been able to confirm by sight. We know that the bacillus of tetanus is found in hay, in earth, on walls, etc. What, then, is there surprising in the supposition that man and animals should introduce this source of infection by the digestive tract, and that, as is occasionally observed in anthrax and tuberculosis, it may be the intestinal mucous membrane, made vulnerable through some predisposing cause, which is the seat of inoculation? The experiments of Brieger prove that the bacilli of Nicolaier are readily cultivated in meat, milk, etc., and that they secrete their characteristic ptomaines in these media.

Guelpa cites the case of Zabludowsky in which the tetanic spasm ceased after washing out the stomach, and he also relates the old story of Betoli, of a number of slaves who had eaten of the flesh of a steer dead of tetanus following castration, and who themselves contracted tetanus, some of them dying. It is interesting in this connection to state that Sormani,[1] although he failed to induce tetanus by feeding to dogs and rabbits tetanus cultures as well as the flesh of animals dead of tetanus, showed that the virulence of the bacillus was unchanged by its passage through the digestive tract, the fæces of animals so fed proving tetanogenic when inoculated. May it not be possible that such influences as cold, over-exposure to the sun, alcoholic intoxication, the presence of worms, etc., act by making the intestinal tract in some way vulnerable?

Guelpa's conclusions regarding the nature of tetanus are exceedingly interesting and important, and are as follows:

1. Tetanus is an infectious disease.
2. Rheumatic tetanus, in the true sense of the word, does not exist.
3. Although the horse is one of the animals most apt to contract this disease, tetanus is not of equine origin. It would be more correct to attribute it to telluric origin; but this would be too restricted. We believe it preferable to affirm simply that it is of microbic origin.
4. The symptoms of tetanus are not direct effects of the microbes, but occur in consequence of the toxic substances generated by them.
5. During the first manifestations of tetanus at least, the multiplication of the microbe is limited to the site of the infection. It is only later and quite rarely—in six per cent. of the cases, according to Rosenbach—that the bacillus becomes generally diffused through the organism.
6. Although opposing the nervous theory of tetanus, we must admit that the nervous system possesses an excessive susceptibility altogether peculiar to the action of the micro-organisms or the products generated by them.

The above conclusions are eminently justified by the facts in our possession, and they are probably final.

Morbid Anatomy.—The changes found in the tissues after death have been various and of doubtful import. At times the investigation yields none but negative results. At other times marked hyperæmia of the brain and cord and of their membranes is found. Minute hemorrhages and even extensive extravasations of blood sometimes occur, as do also effusions of serum. In tetanus neonatorum there is generally marked fulness of the spinal veins, often associated with actual escape of blood. Microscopically various appearances have been noted by Rokitansky, Demme, Wagner, Fechner, Lockhart Clark, and others. They have been variously described as hyperæmia, exudation, granular degeneration, centres of softening in gray and white matter, nuclear proliferation together with the formation of fat-granules and amyloid bodies, etc. Certainly, if any interpretation is to be placed on these observations it is that the changes met with are the result of rapid tissue-destruction; they are, so to speak, the marks of devastation left by the storm.

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[1] Riforma Med., Napoli, 1889, v. 1202.

The peripheral nerves are at times found perfectly healthy. At other times neuritis with marked swelling is found near the site of the wound. Foreign particles have also been found embedded in a nerve-trunk, or the latter has been badly crushed and torn.

The voluntary muscles are generally unusually pale. Occasionally minute extravasations of blood are seen, and here and there individual bundles of fibres are found ruptured. Fatty change and granular degeneration are also noted.

In tetanus neonatorum the umbilical cord may present nothing abnormal, or it may be the seat of more or less marked inflammation. There may be phlebitis and even a localized peritonitis.

Diagnosis.—The symptoms of tetanus are, as a rule, so clearly marked that only in very exceptional cases can a doubt legitimately arise. The condition which, of all others, most closely resembles the clinical picture of tetanus, is that of strychnine-poisoning. In the first place, however, strychnine-poisoning presents in its earliest phase none of the general malaise and oppressive fear which precede the outbreak of tetanus, but, instead, a feeling of exhilaration and restlessness. Again, we notice that the muscular symptoms, instead of developing gradually, come on with great suddenness, and the spasms, instead of beginning in the jaws and the back of the neck, begin in the extremities or in the whole body simultaneously. The muscles of the jaws are the last to be involved, and, furthermore, they remain relaxed in the intervals between the paroxysms. In fact, in strychnine-poisoning there is none of that persistent tonic rigidity which is so typical in tetanus. Very slight stiffness may, indeed, be present in the intervals of the convulsions, but it is rare. Again, the hands and forearms, almost never involved in tetanus, are here involved to an extreme degree and at an early period. There is also an absence of the risus sardonicus; at least, there is nothing approaching it in the quiescent intervals.

In both tetanus and strychnine-poisoning the reflexes are much increased.

In tetanus, however, this is not observed until the disease is well developed, while in strychnine-poisoning it is one of the earliest symptoms. Lastly, the existence of a recent trauma would have special significance. The mental condition is in both instances unimpaired, and affords no element of differentiation.

Hysteria in a major form, though rare, occurs with sufficient frequency in children to warrant a word. There may, indeed, be present a general rigidity, interrupted now and then by markedly opisthotonic spasms. However, paroxysms, atypical and bizarre, are apt to make their appearance also. Further, the risus sardonicus is absent. The countenance is frequently extraordinarily placid, or now and then a beatific expression or an expression of anger or fear is present. In the intervals emotional phrases and ejaculations, sobbing, crying, hysterical gestures, etc., may be indulged in. Consciousness, also, is apt either to be lost or to be much perverted.

Tetany offers very few points of similarity to tetanus. In the vast majority of cases it commences by a tonic spasm of the forearms and hands, these parts assuming characteristic and peculiar positions, which it is not necessary to detail here. The legs are next affected, and in severe cases the trunk also participates. The distinction from tetanus is very simple.

Very rarely perhaps it may be necessary to distinguish between tetanus and cerebro-spinal meningitis; but the history of the onset, of chill and fever, the presence of petechiæ, the characteristic attitude, the obvious involvement of cerebral functions, etc., would give abundant grounds upon which to make the diagnosis. In hydrophobia, also, the history of a bite, and the absence of trismus, of general tonic spasm, and of the opisthotonic paroxysm, will serve for differentiation. Even in the dysphagic and cephalic forms of tetanus the absence of the true rabic paroxysm and the presence of a unilateral complete facial palsy will answer the problem.

Lastly, the reader should be cautioned against the various forms of local spasm which occasionally accompany wounds. Rarely in true tetanus slight spasm first makes its appearance in the injured limb, but it is soon followed by involvement of the muscles of the jaws and the back of the neck.

Prognosis.—The death-rate in tetanus is very high, and it is practically never possible to give a favorable prognosis. Among children, indeed, the mortality is at times greater than in adults. For instance, of all of Laurie's Glasgow cases of fifteen or under, not one recovered.[1] Of the cases over fifteen, fourteen and one-half per cent. recovered. In Poland's collection but one of the cases of fifteen or under, sixteen in number, recovered. Of the adults, on the contrary, fifty-six in number, eight, or sixteen per cent., recovered. Taylor's statistics show a slightly lower mortality-rate for children, three out of twelve recovering. Of the cases collected by Packard from the records of the Pennsylvania Hospital, nine in number, but one recovered.

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[1] Loc. cit. Of the miscellaneous cases collected by Laurie from various sources fifty per cent. of the cases aged fifteen or under recovered. The bulk of these cases had doubtless found their way into medical journals by the very reason of their recovery, and are therefore valueless for statistical purposes.

Our opinion regarding a given case is influenced not only by the age of the patient, but also by three other factors. First, the more rapid and severe the development of symptoms, the more unfavorable is the outlook. The converse, however, does not hold good, inasmuch as cases relatively mild at first may suddenly grow frightful in their severity. Secondly, the prospect of recovery is, of course, lessened in cases in which crushing accidents, scalds, or burns have added a surgical increment to the patient's burden. Lastly, our opinion is influenced by the duration of the case. Other things being equal, the longer the duration of the case the better is the prospect of recovery. The majority of the fatal cases terminate within a few days. Thus, of Laurie's Glasgow cases three died on the first day, fourteen on the second, seven on the third, five on the fourth, two on the fifth, and so on, the latest fatal cases occurring on the tenth day. In Poland's cases eight died on the first day, seven on the second, seven on the third, nine on the fourth, one on the fifth, five on the sixth, ten on the seventh, and two on the eighth. The longest fatal case lasted thirty-two days. Of Taylor's cases three died on the first day, five on the second, eleven on the third, seven on the fourth, three on the fifth, and two on the sixth. In other words, the bulk of the cases die within the first five or six days. Occasionally, however, death occurs as late as the tenth, twelfth, twenty-second, twenty-third, or twenty-eighth day, or even later. One of Taylor's cases died as late as the one hundred and nineteenth day. However, in these late cases death is not to be ascribed to tetanus, but, as a rule, to some complication, such as bed-sores.

Lastly, it should be stated that the prognosis in so-called "idiopathic" or "rheumatic" tetanus is much less unfavorable than in ordinary traumatic tetanus.

In casting about for other elements of prognosis we are disappointed. Neither the location of the wound, nor its condition, nor the presence or absence of fever, is of import. Excessive temperature is, of course, of grave significance.

Prophylaxis.—The recent and brilliant discoveries in the pathology of this hitherto obscure disease will, it is to be hoped, yield rich practical results. Almost invaluable, for instance, is the knowledge of the single fact of its contagious and infectious nature. Certainly the first lesson to be learned is that every case of tetanus should be rigidly isolated, and that surgeons should practise excessive precaution in passing from patient to patient. An instructive lesson is that furnished by the experience of Berger[1] and Nélaton. Berger had in his wards a man suffering from a deep burn which had opened the metacarpo-phalangeal articulation. It was found necessary to amputate, and the patient subsequently developed tetanus. Under Nélaton's care was a boy who had been run over, and in whom there were multiple contused wounds, and in addition a large abrasion on the anterior aspect of the thigh. It happened that Berger, who had been present at an operation by Nélaton, requested the latter to see with him his case of tetanus. They accordingly proceeded to Berger's patient, accompanied by the interne and some of the externes of the service. Along each side of the patient's bed a strip of board had been placed, to prevent his falling out. Upon one of these strips Nélaton rested while the dressing was removed. He inspected the wound, but did not touch it, and, on leaving, went to the ward containing the little lad who had been run over. Both himself and his interne now cleansed their hands, finishing by a wash of corrosive sublimate. They removed the dressing, took out some sutures, trimmed off some sloughs, etc., washed the wound with corrosive sublimate, and applied salol. They did not, however, finish the dressing, leaving that to an externe. The latter, it was afterwards remembered, had also been at the bedside of the tetanus patient, and had not been seen to wash his hands. Nélaton's patient promptly developed tetanus,—though, fortunately, he recovered.

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[1] La France Médicale, June 21, 1888.

It can readily be seen that it is more than ever important that the discharges and dressings of wounds in cases of tetanus should be at once destroyed, and that on the termination of a case no other disposition should be made of the bedding than that of consigning it to the flames. Further, the room should not again be used for surgical purposes,—not, at least, until most radical and thorough cleansing and disinfection have been practised. It is doubtful, indeed, whether it be possible to carry this out effectively, especially when we reflect upon the hardiness and extraordinary vitality of the germs. Kitasato (*loc. cit.*), let us remember, showed that, though they are killed by a temperature of 100° C. in a few minutes, they can successfully withstand a temperature of 80° C. (172° F.) for an hour. Further, they resist a five-per-cent. solution of carbolic acid for ten hours, and only after fifteen is their vitality absolutely destroyed. A solution of five per cent. of carbolic together with one-half per cent. of hydrochloric acid is effective only after five hours. Corrosive sublimate in the strength of one to one thousand is effective only after three hours; though when associated with one-half per cent. of hydrochloric acid it destroys the germs in thirty minutes. Certainly it seems as though modern methods of antisepsis can prove but a weak and imperfect barrier against these germs. Further, how long they may possibly linger about a hospital ward is suggested by the experiment of Kitasato, in which they were adherent to silk threads, desiccated over sulphuric acid, and afterwards kept in ordinary air; months afterwards they retained their virulence.

The frequent connection between tetanus and equine and telluric influences should lead us to regard with especial apprehension all wounds that have been contaminated with earth, horse-manure, hay, etc., and to exclude such influences by all possible care in ordinary and surgical wounds. These influences are perhaps excluded with ease in the ordinary civil hospital; not so, however, during the exigencies of war, as the lamentable story of Abadie already cited fully illustrates. Certainly here are facts which demand the gravest attention on the part of military hygiene.

Treatment.—As regards the actual management of the individual case of tetanus, we are confronted first by the knotty problem presented by the wound. Every possible method of dealing with it seems to have been practised, with, in the vast majority of cases, a negative result. Let us analyze the condition of the wound in the light of recent developments and see what indications present themselves. Guelpa, we remember, concludes that "during the first manifestations of tetanus at least, the multiplication of the microbe is limited to the site of the infection," and, further, that "it is only later and quite rarely that the bacillus becomes generally diffused through the organism." Further, he points out that the bacillus is always to be found in the *deeper* portions of the wound; and this fact seems to have an especial significance. It certainly suggests that from the superficial portions of the wound the bacillus is soon expelled by the defensive process of discharge and suppuration, while in the deeper portions of the wound, ready escape not being possible, the bacillus is retained sufficiently long to mature and secrete its ptomaines. The very conditions demanded by this view are presented in the kinds of wounds most frequently found in tetanus. The punctured wound permits the lodgement of the germ deep within the tissues, with but a narrow, sinuous, and readily-obstructed outlet. The lacerated wound and the deep burn expose intermuscular septa, open up the sheaths of nerves and tendons, joints, etc., and permit of the establishment of numerous and tortuous tracts of infection. Lastly, successful inoculation in animals is made not by rubbing the virus upon an abraded surface, but by introducing it beneath the skin, into the sheaths of nerves, and beneath the dura of the brain.

If these inferences be correct, the first indication for treatment is very clear. Upon the very earliest signs of tetanus the wound should, other things being equal, be freely incised and its innermost recesses exposed. Guelpa proposes that the wound be curetted, the tissues freely released, and, if necessary, deep excisions of tissue made. These directions, of course, cannot apply to every individual case, but must be adopted according to circumstances. Guelpa calls especial attention to the fact that it is in the thrombi of veins coming from the wound that the principal nidus of the microbe is frequently found. The operation being completed, Guelpa proposes to irrigate the wound with a strong solution of corrosive sublimate, —from five to ten parts in a thousand,—or even with a strong solution of nitric or sulphuric acid. The propriety of this proceeding seems to the writer highly questionable. Certainly abundant and frequent irrigation will accomplish quite as much, and add nothing in the way of shock or suffering. Indeed, the surgical interference should be judicious in the extreme, and should be limited to free opening up of the wound, the excision of ragged edges and sloughs, the removal of clots, thrombi, etc., and thorough washing, the wound being kept open, so that the washing can be repeated at intervals.

The mere fact that recoveries have occasionally occurred after nerve-stretching and even amputation is of no significance, inasmuch as both death and recovery have occurred indifferently after the most diverse methods of treatment. Certainly in the present state of our knowledge nerve-stretching has nothing to recommend it, while amputation is probably rarely justified. It should be remembered that the average case of tetanus comes under medical care after the symptoms are fully developed and when some degree of general infection has taken place. Unless indicated upon general surgical grounds, amputation should not be practised, lest the shock and irritation of another trauma be added to that from which the patient is already suffering.

So much for traumatic tetanus. How shall we deal with the avenue along which, according to Guelpa's theory, infection takes place in "idiopathic" or "rheumatic" tetanus? Evidently the indication is to treat the digestive tract antiseptically, in addition to free purgation. The drugs that naturally suggest themselves in this connection are salol and salicylic acid. Either of these could be administered in full doses, followed some hours later by a brisk saline cathartic. This preliminary treatment could do no harm, and certainly deserves trial.

The general indications for treatment now remain to be considered. First and foremost, every effort must be made to maintain the strength of the patient. At the very threshold we are met by the mechanical difficulty of administering food; even when the trismus does not of itself prevent the taking of nourishment, the mere effort at swallowing is apt to provoke the paroxysms. All nourishment should, of course, be liquid, and yet in extreme cases even this cannot be administered by the mouth. At times, when the "lock-jaw" has been excessive, a tooth has been extracted in order to gain additional space. The propriety of this measure is, however, for reasons already advanced, open to question, and especially because the method of feeding now so extensively and successfully practised among the insane, of passing a soft rubber catheter through the more spacious of the two nasal cavities and sufficiently far back until the tube has fairly passed into the œsophagus, is here especially applicable. The catheter being warmed and carefully oiled, but little irritation is produced, and by means of it any desired quantity of liquid nourishment can be given. It may be, however, that the operator is so frequently interrupted by the paroxysms, or that the presence of the catheter in the pharynx provokes these to so great an extent, that he will be obliged to desist. However, it is a method which should always be tried in preference to extraction of teeth or rectal feeding. The last-mentioned should be a *dernier ressort.*

The food should consist of milk, or milk with the addition of raw eggs, or—better, perhaps, than either—peptonized milk. The quantity, of course, must depend upon the age of the patient, but, other things being equal, the maximum amount of food should be given. Alcohol also is here of undoubted service, not only by stimulating the digestion of the nourishment taken, but also because of its steadying action upon the heart and its sustaining powers generally. Besides, there can be no doubt that it largely diminishes the horrible psychic depression from which the patient suffers.

Just as in strychnine-poisoning, everything that can possibly produce peripheral excitation should be removed or avoided. The bedclothes should not be uncomfortably heavy, and it should be the effort of the nurse to keep such portions as are in immediate contact with the body smooth and evenly arranged. It should be also seen to that the dress of the patient is loose and open about the neck, and that there is nowhere any constriction or impediment. In the necessary attentions to the patient, all sudden touching of the surface by cold hands or unnecessary turning or raising of the body should be avoided. It should be remembered that at times so slight an impression as a breath of air is sufficient to bring on a paroxysm. Absolute quiet of every kind is to be the order of the day.

Finally, drugs directly sedative to the nervous system are indicated. Their object should be twofold: first, to allay the excessive spasm, and, secondly, to produce sleep. To accomplish these ends numerous drugs have been used. Foremost among them stands bromide of potassium. Large doses frequently repeated should be given, and, if there be great difficulty in administering it by the mouth, it may be injected into the bowel. That it is of very decided value cannot be doubted. H. C. Wood has collected thirty cases in which recovery followed its use, though in ten of these other drugs were given in addition, and in one small bleedings and etherization were also employed. It must be conceded that bromide of potassium is one of the few drugs that promise an alleviation of the symptoms.

Next in value comes chloral. This drug tends both to produce sleep and to act as a motor depressant to the cord. It must be given, like the bromide, in large doses, if benefit is to be expected from its use. Its use is, however, not unattended with danger to the heart, especially in children, in whom cardiac depressants seem to act with unusual readiness. To some degree this difficulty may be met by the cautious use of digitalis. Further, chloral presents such palpable advantages that the risk of its depressive action on the heart can, other things being equal, be ignored. Certainly, in a disease the mortality of which is so high, no time is left for vacillation. It should be used first in moderate and then in larger doses, the pulse being carefully watched, and alcohol and digitalis being used as indicated.

Urethan is also a drug which may prove of value, though it has failed to attain a high reputation as an hypnotic. It is deserving of extended trial, because it appears to have a direct depressant action on the cord without entailing any depression of the heart. Jackman has reported a successful case.

Frequently it is not possible to procure the much-longed-for sleep and quiet by means of chloral alone, and at such times it is well to combine the treatment with the use of morphine. Morphine is a valuable adjuvant, but must be used with discretion. As a rule, it is well borne, *i.e.*, in moderate doses, but we should remember that in the average case respiration is already much interfered with, and that the too free use of the opiate may favor the development of a fatal cyanosis. These remarks apply with especial force to children. However, the suffering of the patient is often so great, so evident, that its use to some extent, at least, is absolutely demanded.

Very frequently the best results are achieved by a combination of remedies. Either the bromide, chloral, and morphine may be given together at stated intervals, or, what is perhaps better, the bromide and chloral may be administered in the same dose, while the morphine may be used hypodermically as occasion arises. Occasionally it may be well to combine the morphine with some other hypnotic, such as paraldehyde, neither drug being given in full physiological doses. This was recently successfully carried out in a case under the writer's care.

In addition to the various remedies used to produce sleep and to lessen spasm, active measures are frequently called for during the paroxysm. Chloroform inhalation has often been used to mitigate the attacks, as has also ether. Complete relaxation, however, never results unless the anæsthetic be pushed to its full effect; and, as may be supposed, this is not unattended by danger. Especially is this true of chloroform. The patient's breathing is already much embarrassed, and care should be taken that the anæsthetic is well mixed with air. Nitrite of amyl has also been used to break up the paroxysm, and certainly deserves more extensive trial. H. C. Wood speaks highly of its value, and in a case recently under the observation of the writer the convulsions were undoubtedly ameliorated by its use, the patient finally recovering. Gowers, however, states that at Guy's Hospital it was observed that the spasm became more intense at first, though slighter afterwards.

Various other remedies have from time to time been used in the treatment of tetanus, but with no result that would justify confidence. Among these is bloodletting, now happily fallen into disuse, and the indication for which it would, indeed, be difficult to see. Warm baths were also at one time thought to exercise a beneficial action in relaxing the spasm; but the difficulty attending their use, and the unavoidable and injurious handling of the patient, make their utility more than problematical.

Calabar bean also has been extensively used, and has the advantage of being capable of subcutaneous administration. It appears really to possess some value. In the cases collected by Roemer and by Wood quite a notable percentage, something over fifty per cent., recovered. This evidence is, however, open to the objection made elsewhere regarding isolated cases collected from miscellaneous sources. We are far more apt to glean the truth from carefully-preserved hospital records. For instance, among eight cases treated at Guy's Hospital by Calabar bean,[1] but one recovered; nor does this result seem to have varied when the remedy was fortified by the action of other drugs, such as chloral and morphine. In six cases of mixed treatment but one recovered. When using Calabar bean the alkaloid is to be preferred. H. C. Wood fixes as a moderate dose for an adult one-fifteenth of a grain of eserine repeated every three hours. If no effect is produced, the dose should be increased. Bouchut, cited by Wood, found that one-twentieth of a grain given hypodermically to children produced very decided symptoms, which passed off in a few hours. It would seem, therefore, that a properly-proportioned dose would hardly be accompanied by danger.

Among the various other drugs that have been used are cannabis indica, belladonna, conium, tobacco, lobelia, and curare, all, it is needless to say, without fixed result. Cannabis indica is certainly very inferior to opium, the purposes of which it only in a slight measure fills. Curare, again, is so excessively dangerous that, although its action is sufficiently well known, its use must always be regarded as purely experimental. Though first suggested many years ago, the uncertain composition of individual samples of the drug, and the consequent uncertainty in its dosage, have prevented, among other things, its practical application except in isolated instances.

Passing in review the various points in the treatment of tetanus, we have, in addition to the surgical treatment of the wound, *first*, the maintenance of absolute quiet; *secondly*, efficient feeding, including the administration of alcohol; *thirdly*, the use of various drugs to allay the motor excitement and to produce sleep. I would suggest that these drugs be used in the average case as follows. Bromide and chloral should be given together in full doses, proportioned, of course, to the age of the child; these doses should be repeated about every four hours. In addition, morphine, or morphine combined with some other hypnotic, should be given at intervals according to circumstances. Should symptoms of cardiac weakness come on, digitalis should be added to the treatment and the amount of alcohol increased. Lastly, nitrite of amyl should be used to combat the paroxysms, and, if inefficient, chloroform or ether should be substituted.

If it be impossible to administer the bromide and chloral by the mouth, recourse should be had to the rectum, and, if this latter expedient fail to make an impression, hypodermic injections of eserine should be resorted to. The intravenous injection of chloral, at one time practised, is inadmissible.

Lastly, the patient should be closely watched by an attendant previously instructed in the methods of artificial respiration, for, should profound cyanosis make its appearance, as it is apt to do at the end of a paroxysm, artificial respiration must be promptly carried out. At other times atropine, digitalis, and possibly ammonia, should be used subcutaneously. It is not improbable that every now and then a desperate case may be tided over by extreme care and watchfulness.

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[1] Taylor, op. cit., p. 358.

The bowels should, of course, be opened, though this is often a matter of extreme difficulty. Saline cathartics are to be preferred.

As regards the excessive sweating, it does not seem to call for special interference. Guelpa, indeed, goes so far as to recommend pilocarpine, with the idea that the noxious ptomaines will be excreted by the skin. This is, however, purely hypothetical.

ATHETOSIS.—CATALEPSY.—THOMSEN'S DISEASE.

By GEORGE W. JACOBY, M.D.

ATHETOSIS.

Definition.—Athetosis (from ἄθετος, "without fixed position") is a name first used by Hammond in 1871 to specify a peculiar disorder of motion, characterized chiefly by continuous spasmodic movements of the fingers and toes, and by an inability to maintain the affected parts in any position in which they may have been placed.

History.—Since Hammond's description, cases have been published by numerous observers, among whom are T. C. Shaw, Clifford Allbutt, Eulenburg, Oulmont, and Gowers. The thesis of Oulmont, in addition to a few personal cases, presents a *résumé* of all cases published prior to 1878. Of late years the original definition of Hammond has not been strictly adhered to, and cases have been described under the name of athetosis which apparently should not be so termed.

Etiology.—In the majority of published cases either no cause is assigned or the supposed cause cannot be accepted by an impartial critic. Thus, in one case of congenital athetosis the mother during pregnancy was the subject of undue psychic excitement. In another case (Landouzy) the affection appeared after a severe fright during childhood. Traumatism (Hughes) and cold (Walker[1]) have also been credited with its causation. In a large number of cases the patients had previously been afflicted with epilepsy, delirium tremens, or psychoses of various character. It has frequently been observed in imbecile children (Shaw).

Secondary athetosis is frequently encountered in childhood as a sequela of polioencephalitis (Strümpell). This fact is clearly demonstrated by Oulmont's data. Of eleven cases of athetosis following upon ordinary hemiplegia, three occurred in childhood. Twelve cases following upon hemiplegia with epilepsy occurred in children whose ages varied from a few months to seven years. Sex has no influence upon the production of the disease; neither is there any preponderance of one side of the body affected over the other.

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[1] Case of a boy. Athetosis occurred suddenly after a bath in very cold water.

Pathology.—Autopsies in children are unknown, and cases dating from childhood furnish few reliable data. We are, therefore, obliged to draw our conclusions from the autopsies made in adults. In reference to secondary athetosis, Kahler and Pick have clearly proved that all post-hemiplegic disturbances of motion are due to a lesion of the pyramidal tract ascending between the thalamus and the lenticular nucleus. The various forms of disordered motion are to be explained by simple irritation and by total or partial destruction of this system of fibres. In all cases of post-hemiplegic athetosis, anatomical examination has shown the lesion to be in the vicinity of this tract. The focus was in the left thalamus in a case of Gowers's; Weir Mitchell, Lépine, and Landouzy found lesions of the striata. In twelve autopsies cited by Kahler and Pick, the parts mainly involved were the thalamus, the lenticular nucleus, and the internal capsule. It is very probable that athetosis, as well as all the other post-hemiplegic disorders of motion, is caused by an irritative process affecting the pyramidal tract at any part of its course. Bernhardt, in a case of hemiathetosis post-hemiplegica, found foci of softening in the right striatum and one focus also in the left. The right anterior communicating and the right Sylvian artery were obstructed. Ewald in a case occurring during the course of a dementia paralytica, in addition to signs of acute and chronic meningitis, found two circumscribed foci of softening in the first left temporal convolution. Rosenbach in a case of bilateral athetosis occurring during the course of a case of tabes dorsalis, in addition to the sclerosis of the posterior columns, found a focus of softening in the posterior lower and external end of the *right* lenticular nucleus. A case of this kind (athetosis and tabes), but without autopsy, was also seen by Berger. Murrell in a case of his found the right corpus striatum to be almost entirely destroyed, the thalamus appearing healthy. Landouzy in a case of right hemiathetosis dating from childhood found an old lesion situated in the anterior part of the left lenticular nucleus.

Cases of athetosis without hemiplegia are probably produced by an irritative central process not sufficiently extensive to produce a paralysis (Berger). In one of Oulmont's cases of primary athetosis a focus of softening was found in the lenticular nucleus and the striatum of one side. Lauenstein's case is of importance in so far that the athetosic movements, which were limited to the fingers of the left hand, occurred eleven days before death in a patient suffering with cardiac disease, no paralysis being manifest. The autopsy revealed a fresh focal lesion at the anterior end of the right thalamus.

These facts, together with Démange's case (conclusive of cortical disease), corroborate the statement that a lesion at any part of the pyramidal tract may produce athetosis. In cases dating from childhood, Gowers, on account of its varied position, extent, and frequency of occurrence, believes the athetosis to be due to impairment in the nutrition of the growing motor cells.

Symptoms.—The spasmodic movements appear comparatively suddenly, certain prodromi generally having been overlooked or disregarded.

The previous history will often reveal the pre-existence of motor disturbance. As a rule, a unilateral paralysis or paresis will be found to have been present prior to the development of the athetosis. This is then secondary to some other affection of which the paralysis is a symptom.

The spasms, which constitute the main symptom of the athetosis, are very characteristic. They are localized exclusively, or nearly so, in the peripheral ends of the extremities, affecting the fingers, hand, toes, or foot. As a rule, they are unilateral. In rare cases the muscles of the neck and face are affected, causing continual distortion of the face and mouth. The tongue may also be affected (Strümpell), rendering speech indistinct and labored. The spasms are always of greater intensity in the fingers than in the toes. They are frequently entirely wanting in the toes, very rarely in the fingers. In a boy aged fourteen (Bernhardt) and in another case (Tison) the movements were confined to the lower extremity. The movements when seen in the fingers and hand are those of adduction and abduction, alternating with movements of supination and pronation. They are entirely involuntary, always slow, extensive, and rhythmical, executed with a certain regularity and even monotony, representing a species of irregular incoördination. They make the impression of labored voluntary movements. The term mobile spasm (Gowers) expresses the condition clearly.

In some cases hyperextension of the fingers, with positions of subluxation due to a relaxation of the tendons, is noticed. The movements are generally very active, but are continually changing in intensity and become less marked when the attention of the patient is distracted. Those present in the toes and feet are of the same character. The movements are present at all times, and in the majority of cases persist during sleep. Only in exceptional cases can they be voluntarily suppressed, and then only for a brief period. In a case of our own, as also in Hammond's original case, energetic compression of the wrist-joint produced temporary cessation of the movements.

Atypical cases, with periodical presence of the spasms, have also been observed. Transitory contractures of the wrist and ankle also occur, the characteristic mobile spasm disappearing and the special feature of the athetosis being temporarily lost. A remarkable fact is that these athetosic movements may continue indefinitely without causing any complaint of fatigue, although the natural functions of the affected limbs, the hand and fingers especially, become greatly impaired, causing the voluntary movements to be slow and labored (Bernhardt, Rosenbach, Berger). Generally the affection is confined to one-half of the body (hemiathetosis), but cases are known in which both sides were affected (athetosis bilateralis).

The most frequent form of athetosis is the secondary or symptomatic form, in which the movements appear in the paralyzed limbs of hemiplegics usually at a time when partial retrogression of the paralysis has taken place. In a case of Barr's the movements first appeared in the non-paralyzed side, subsequently implicating the other. The secondary form is always a symptom of a cerebral focal lesion, and constitutes a special form of post-hemiplegic disturbance of motion (Gowers), clinically differing materially from post-hemiplegic chorea (Weir Mitchell, Charcot). In these cases the hemiparesis, hemianæsthesia, atrophy, and contracture of the muscles, exaggerated tendon reflexes, disorders of intelligence, abnormal irritability, and epileptic attacks, when present, are also symptoms of the focal lesion. In a limited number of cases the athetosis has developed primarily without the precedence of other symptoms in the affected limbs. Of this idiopathic form the most interesting variety is the congenital athetosis (Strümpell, Bourneville, and Pilliet). The majority of primary cases are also bilateral (Bourneville, Warner). Primary cases have been described by Landouzy, Gnauck, Oulmont, Eulenburg, and Kirchhof.

Diagnosis.—If we confine ourselves strictly to the definition and the description, the diagnosis of the affection is not difficult. The strict localization of the movements in the fingers and toes not being acknowledged by all authors, the diagnosis will consequently depend almost entirely upon the character of the movements. In children the diagnosis must always be made between a hemichorea and a hemiathetosis. The localization of the affection is important here. Whereas in athetosis only the peripheral parts of the extremities, the hands and the feet, are affected, in chorea such is not the case. If the face alone is affected, the disorder may be considered choreic and not athetosic. The movements themselves are also entirely different in character in the two disorders. In chorea we have the quick, irregular, misdirected movements, increased by voluntary efforts, and irregularly spread over all the muscles. In athetosis the regularity and monotony of the movements form a sufficient contrast to this picture. In some cases, both factors, localization and character of the movements, will be requisite for a correct diagnosis; these are the exceptional cases of athetosis in which the movements partake somewhat of a choreic character. Thus, a case of primary athetosis has been described which gradually became transformed into a chorea (Leube), and also a case of chorea posthemiplegica in which the choreic movements assumed an athetosic character (Bernhardt).

Prognosis.—The affection is generally chronic, and remains unchanged for years. Recovery occurs, if at all, only in the rarest cases. A case of primary athetosis of Gnauck resulted in complete recovery, and Case 28 of Oulmont's thesis was slightly improved. Improvement of secondary cases has been mentioned by Hammond, Gowers, and Proust, but no cases of cure are known. When death occurs it is not due to the athetosis as such.

Treatment.—In reference to the treatment of the affection little is to be said: thus far the various remedies have proved of little avail. Electricity, the hope of many, is of no value. The nature of the lesions known to produce athetosis is of such a character as to preclude their possible removal by remedial agencies. In the cases which improved, the remedies employed were galvanism (Oulmont), bromide of potassium, mercury (Oulmont), iodide of potassium (Gowers), setons, chinin, and nerve-stretching (Hammond).

LITERATURE.

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CATALEPSY.

Definition and History.—The term catalepsy, formerly employed to denote a special form of disease, is now very generally used to denote a special symptom occurring in a variety of diseased conditions of the nervous system. The use of the word catalepsy itself dates back to the ancients, Greek writers using it in the significance of a surprise, a seizure.

Whereas Hippocrates employs the word κατάληψις to represent a convulsive disorder, Galen describes the affection in a manner which entirely coincides with our views. During the period intervening between Galen and Hoffmann (1692) the most varied disorders were confounded under this name. The "*flexibilitas cerea*" of the limbs was first described by Sprengel. Osius (1799), Tissot (1807), Fleisch (1812), and others gave clear descriptions of the condition. Gradually, however, the views and descriptions of the various authors became more and more dissenting, until the title "catalepsy" embraced descriptions of affections like chorea, epilepsy, hysteria, etc. Even at the present day the precise significance of the term as understood by the various authors varies greatly, the only common principle which is retained by all as a *sine qua non* being the waxy flexibility.

Etiology.—Spontaneous catalepsy, the term being used in contradistinction to induced catalepsy (the cataleptic condition produced in certain subjects through the influence of a loud noise, a dazzling light, prolonged fixation of an object by the eyes, or as a sequel of the lethargic condition in hypnotized subjects), may occur in both sexes and at all ages. It is, however, most frequent in the female sex and in early adult life. The affection is unusual in early childhood. To my knowledge the youngest case on record is that of a child three years of age, described by A. Jacobi. Monti records eleven cases encountered in children. The sexes were about equally affected, and the ages varied from five to fifteen years. Eulenburg speaks of catalepsy at five years of age; Schwartz saw a case in a boy of seven, and Hovey in a boy of eight. The neuropathic constitution seems to play as great a part in the production of catalepsy as it does in the production of other nervous affections. We therefore not infrequently find catalepsy occurring in families in which insanity and drunkenness are present, and in which the mode of life and a faulty mode of education also may have had an influence upon its production. We do not here refer to those cataleptic conditions met with as part and parcel of certain psychoses.

Etiological importance has been attached to blows and maltreatment (Schwartz, boy aged seven, chorea followed by catalepsy, after maltreatment), and to emotional disturbance or sudden alarm (Glas, catalepsy in a girl of ten, after fright while bathing). Retarded sexual development and chlorosis were in some cases supposed to exert an influence upon its production. In three cases of ten referred to by Landouzy, catalepsy occurred after sudden suppression of menstruation. Masturbation and reflex irritation have also been credited with its production. In short, every factor which is of significance in the production of hysteria is spoken of here; it is also a fact that catalepsy is most frequently met with in hysterical individuals, and that hysteria is one of the most important elements in its production. We furthermore encounter a cataleptic condition in certain cases of chorea, in various psychic depressive and exalted conditions (melancholia attonita, mania, ecstasy), and in various organic cerebral affections, especially meningitis.

Of interest also are the artificially-produced forms of this disorder. Without entering upon the various hypnotic experiments for the production of catalepsy and hemicatalepsy, it is worthy of note that Lasègue has produced a transient catalepsy by simply covering the eyes of hysterical patients, and that Strübing, in a cataleptic subject, could at any time produce an attack by the transverse passage of a galvanic current through the head.

Pathology.—Our anatomical knowledge of catalepsy is very slight.

Schwartz, in the case cited, found softening of the striatum and thalamus and exudation into the dura spinalis. Meissner in another case (man, cataleptic, then epileptic, and finally hemiplegic) found upon autopsy an epithelioma in the anterior cerebral fossa, growing from the dura. The right cerebral cortex and the striatum were softened. No deductions of any kind are admissible from this scanty material, and we can therefore only surmise the pathogenesis from the character of the symptoms. Authors have here allowed their imagination full sway. Henle believed in a depression of cerebral action, and considered catalepsy analogous to syncope. L. Meyer believes that the mental impression produces an increased muscular tonus. Hammond speaks of paralysis of the will, masked epilepsy, etc. Eulenburg considers the cataleptic muscular contractions as reflex, and says they must be increased if the voluntary impulse from the cerebrum is abolished, particularly if the reflex inhibitory centres are lost. Soltmann thinks that a connection is possible between the physiological contraction of the muscles in the new-born and the contractions in catalepsy.

No matter how we endeavor to explain the process, we have apparently a continuous stimulation of the muscles by some of the motor centres: that these are in a condition of overaction on account of loss of some inhibitory action seems probable; but which influences are at work in keeping the muscles in such a state of innervation that they are in a condition of contraction just sufficient to overcome the force of gravity and to enable them to retain the limb in the position in which it may be placed, is totally unknown.

Symptomatology.—In an analysis of the symptoms presented by the various cases we must differentiate between those of the paroxysm itself and those of the causal disease. The attacks themselves sometimes show certain prodromal symptoms. Dizziness, headache, hiccoughs, cramps of the stomach, tremor, heaviness of the limbs, impairment of memory, fainting-spells, have all been classed as such.

As a rule, the attacks occur suddenly, and even if prodromal symptoms are present they are generally disregarded. The entire muscular system becomes rigid, and the patients remain fixed and immovable in the position in which the attack overtook them. The muscles feel hard and tense, and this rigidity is at first excessive, but after a short time the limbs submit to passive movement, any position in which they may be placed being retained for a few minutes. The muscular contractions produced by the electric current disappear with the discontinuance of the current (M. Rosenthal). There is always a certain amount of resistance to passive movements, and this, on account of the impression made upon the operator as though he were moulding wax, has been termed *flexibilitas cerea.* Any desired position may thus be imposed upon the limb. The leg, arm, hand, etc., may be placed in any special position (in contradistinction to hypnotic catalepsy, no command need be issued nor remark made), and it will there remain until it falls in consequence of the laws of gravity. The countenance is usually void of any expression, the eyes remaining closed or open, according to their condition at the beginning of the attack. The pupils are dilated and reactionless. The state of sensibility varies; in profound cases there is complete anæsthesia, in others sensation is normal, and in still others there is hyperæsthesia (Puel, Lasègue). Reflex excitability is absent; in severe cases even the corneal reflex is not present. The patellar tendon reflex is retained. Consciousness is generally lost, but in some cases it may be partially or entirely (Mosler) retained. The temperature is either reduced or normal; pulse and respiration are weak, hardly observable, but regular. The attacks may last from a few minutes to several hours, and the recovery is either gradual or sudden. In some cases they recur with remarkable regularity, but their frequency as well as their severity varies in each individual case.

Diagnosis.—The diagnosis of the paroxysm presents no difficulty whatever. If we bear in mind the characteristic form of muscular contraction, the *flexibilitas cerea,* it is hardly possible to confound the attack with any other disorder. Cases of simple trance should not be termed cataleptic, on account of the absence of muscular rigidity. The diagnosis of the causal affection is more difficult. The cataleptic phenomena alone give us no clue, but the associated symptoms occurring during the attack as well as in the intervals are important. In all cases of hysterical catalepsy the presence of other hysterical symptoms will reveal their nature. The assertion that catalepsy may be mistaken for tetanus, apoplexy, or syncope is unwarranted, if its characteristics are borne in mind.

Prognosis.—It is rare for death to occur during an attack of catalepsy; the prognosis, so far as life is concerned, is good; but whether a recurrence of the attack can be prevented is more difficult to answer. The nature of the causal affection will here be the determining factor. In the majority of cases the attacks recur at intervals during the entire life of the patient.

Treatment.—In the treatment of this affection we must above all endeavor to remove the producing cause of the abnormal condition. That this is not easily accomplished, and frequently cannot be done at all, is shown by the ill success which attends our efforts in the treatment of other severe hysterical conditions. The general nutrition of the child must be improved by dietetic or medicinal means; above all, good food and fresh air are to be recommended, and then hydrotherapy, massage, gymnastics, and bitter tonics will be found of value. The former depletory mode of treatment, by means of venesection, diaphoretics, emetics, etc., cannot be too severely condemned. Specifics do not exist. For the cure of catalepsy, as for that of all other diseases in which the treatment is difficult, innumerable remedies have been recommended. Chinin, purgatives, anthelmintics, chloride of sodium, electricity, etc., have each been credited with the cure of certain cases.

During the attack itself very little is to be done. The attack generally ceases by self-limitation, independently of any remedies employed. Those cases in which the attack may be interrupted by a pail of cold water or the actual cautery are probably not true cases of catalepsy. In the majority of cases the use of the faradic brush or the actual cautery is valueless. Pressure over the supraorbital foramina, as well as over the ovaries in female patients, may be tried. In a case of Strübing's, both manipulations were capable of interrupting the attack temporarily.

All that we can do is to keep up the nutrition of the patient, making use of the stomach-tube, if necessary.

MYOTONIA CONGENITA.

(THOMSEN'S DISEASE.)

Definition.—This disease, which is named after the physician who, himself afflicted, first attracted general attention to it by his thorough description, is a peculiar disorder of the voluntary movements, characterized by an inhibition of these movements, due to a stiffness and tension of the muscles, and occurring particularly after a period of inactivity.

History.—In 1876 the disease, as it occurred in himself and in four generations of his family, was first described by Dr. J. Thomsen. Twenty-three cases occurred in this family. The title employed in his description was "Tonic spasms in voluntarily moved muscles." The only references to this class of cases which can be found prior to 1876 are one by Sir Charles Bell and another by Leyden. Since Thomsen's publication reports of similar cases have been received from nearly all countries. Many of these cases have only certain symptoms in common with the affection described by Thomsen, while others, in addition to the characteristic symptoms, present phenomena which point to the existence of central nerve disorder. The most complete publication upon the subject, also containing an analysis of the positive and doubtful cases published prior to 1886, is Erb's monograph on Myotonia Congenita. The number of cases analyzed in this book is twenty-eight; this number does not include the cases (twenty-two or twenty-three) occurring in Thomsen's own family, two of which only have been described, and these without any objective examination. The case described by us was the twenty-ninth, and since then a few more cases have been published. This number includes only the pure and typical cases.

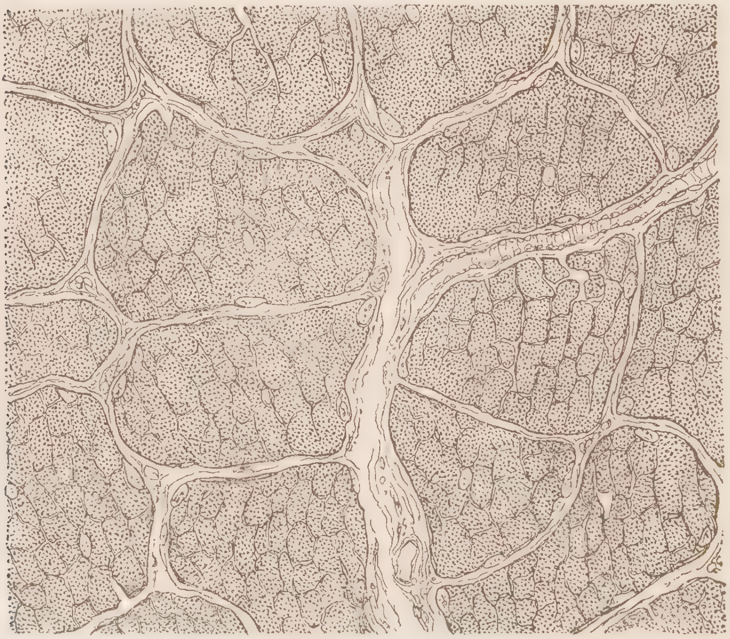
Etiology.—The etiology of the affection demonstrates that the most important factor in its production is heredity. In the majority of cases it is a family disease, and in nearly all the patients the affection was noticed in early childhood, as soon as the child was obliged to make systematic use of its muscles. In a number of cases, in addition to the presence of the same affection in other members or in collateral branches of the family, other neuropathic disorders were present.

Thus, Weidmann gives the history of a patient, of whom one brother was epileptic and another had died in consequence of a meningitis; Knud Pontoppidan describes a case in whose family numerous neuropathic disorders were found; and in the family of a patient of Bernhardt's a number of consanguineous marriages had occurred.

Males seem to be more frequently affected than females. Of the influence of other causes, particularly psychic emotion, we know little: cases have been described by Seeligmüller, Peters, and Engel in which the origin of the affection is ascribed to fright. Engel's case is probably not a genuine case of Thomsen's disease. Climate and country may have some influence upon its production; the affection appears to be more common in Scandinavia and Germany than in France and England, and is exceedingly rare in America.

Pathology.—Autopsies do not exist. In a number of cases pieces of muscle have been either excised or removed by the harpoon *intra vitam.* Thus, microscopical examinations of muscle were made by Ponfick, Petrone, Jacusiel-Grawitz, Knud Pontoppidan, Rieder, Erb, and ourselves, but by all, with the exception of Erb and ourselves, with purely negative results. Erb found the changes in the muscles to consist of an enormous hypertrophy of all fibres, and great proliferation of nuclei, with altered appearance of the minute structure (indistinct transverse striation, homogeneous appearance on transverse section, formation of vacuoles in the fibre). In addition, a slight increase of the perimysium is noticeable. (Fig. 1.)

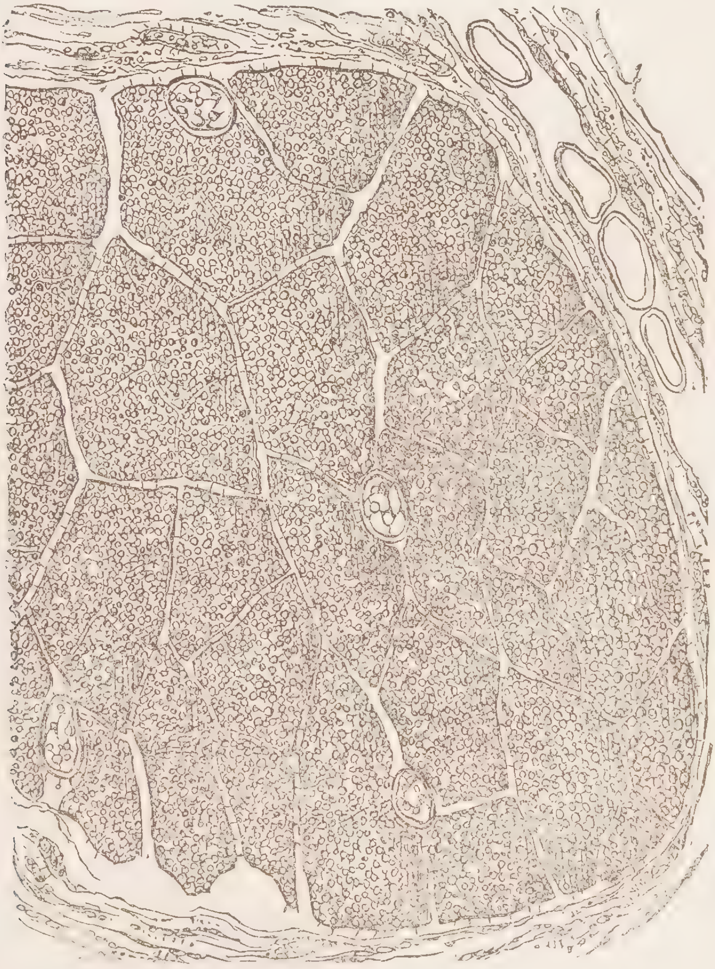
Fig. 1.



Myotonia congenita. Quadriceps femoris. Transverse section. × 300.

In addition to these points emphasized by Erb, we were able to demonstrate another change which is exceedingly characteristic. This is the distinct division of the fibre into angular fields, varying in size, and similar to the faintly-indicated Cohnheim's fields of normal muscle. The splitting up of the fibre can in many places be traced into extremely minute fields, and in many instances is so well marked that comparatively wide gaps are visible between the angular fields. Almost everywhere transverse sections show (with high powers, 1200) a lack of continuity between the groups of sarcous elements. (Fig. 2.) The threads of protoplasm which normally connect the sarcous elements among themselves are almost everywhere broken. In the normal slits, corresponding to the boundaries of the sarcoplasts, as seen in longitudinal section, no protoplasmic connections are visible. (Fig. 3.)

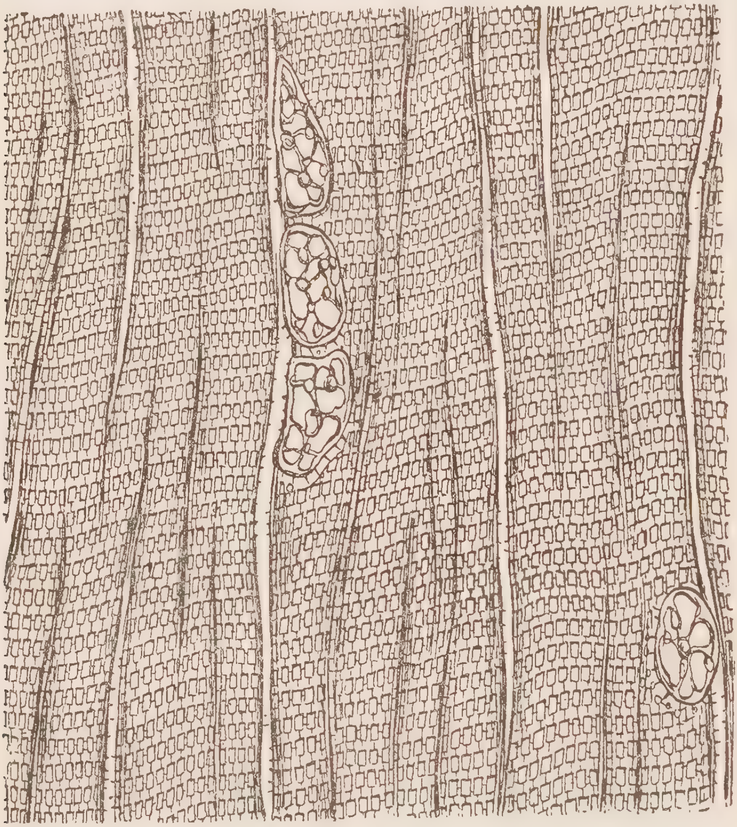
Fig. 2.



Myotonia congenita. Quadriceps femoris. Transverse section of a muscle-fibre. × 1200.

Microscopically as well as clinically, therefore, Thomsen's disease appears to be an affection of the muscles alone. That the disease is a congenital malformation of the muscular fibres is proved by the augmented size of each individual fibre and by an increase in number of nuclei and in the breadth of the perimysium. It is obvious that a considerably larger number of embryonic sarcoplasts must have entered into the construction of each individual fibre than is the case in normal development. Such a muscle necessarily has a considerably larger number of sarcous elements, or, which is synonymous, more contractile matter, than a normal muscle, and therefore the contraction of such a muscle is more liable to become exaggerated, or, to use another expression, tetanic. This is seen under the microscope in the form of clusters of sarcous elements aggregated to a close contiguity. In normal muscle the motor nerves are known to terminate in the form of plates beneath the sarcolemma, but upon the surface of the muscle-fibre. The continuity between these motor end-plates and the adjacent sarcous elements is established by delicate threads of protoplasm. The

FIG. 3.



Segment of a muscle-fibre from myotonia congenita. Quadriceps femoris. Longitudinal section. × 1200.

continuity throughout the muscle-fibre is preserved by such filaments interconnecting all the sarcous elements in every direction. We can thus conceive how the nerve-impulse is transmitted from the motor nerve to the terminal plate, thence into the adjacent sarcous elements, and finally into all the contractile particles of a muscle.

In Thomsen's disease the motor nerves and motor end-plates do not show any deviation from the normal, so that the nerve-impulse is transmitted into the muscle-fibre in the same manner as in the normal condition. The result of this reception of impulse will be a contraction, which, especially after a certain rest, will be a hypercontraction, or, rather, tetanus. This tetanus leads to an agglomeration of a certain number of sarcous elements, with a break in the continuity of the contracted clusters. In consequence of this tetanus, the nerve-influence is inhibited for so long as the tetanus lasts. After the lapse of a few seconds the tetanic contraction will subside, the continuity between the hitherto separated groups of sarcous elements will become re-established, and the propagation of nerve-influence will be again rendered possible. We can thus understand the peculiar reaction of the muscles to the various stimuli when applied directly to them; but why the muscles should react differently to indirect stimulation is still inexplicable.

Symptomatology.—The disease is characterized by an inhibition and awkwardness of voluntary movements, occurring particularly after a period of rest, gradually disappearing, and remaining absent as long as the exertion is continued. The peculiarity is already noticeable in children when they first begin to play; they are awkward in their movements and cannot compete with their playmates in such games as require full and rapid control of the muscles.

Subjectively, the affection consists in an inhibition of the rapid and prompt execution of all voluntary movements, in consequence of a stiffness and tonic spasm occurring in the muscles. After a period of rest, upon any exertion, the spasm will be at its height: the muscles are then completely stiff, and movements can be executed only with the greatest difficulty, if at all: thus, in our patient, if he while running accidentally stubbed his toe, the entire body became stiff, he fell to the ground, and was then unable to arise until the spasm had passed away. The spasms always pass off soon, and during the intervals the patients do not differ from healthy persons. Thus, a patient who upon getting out of bed in the morning cannot walk at all, on account of the spasm, will after repeated attempts succeed in making a few steps, and will then be able to walk for hours without any trouble; the next period of rest will, however, again bring on the spasm. Various grades of severity are found in various patients. Generally, all the voluntary muscles, with the exception of those of the face, tongue, and eyeballs, are thus affected; in our case these muscles did not differ from those of the rest of the body. Certain influences seem to increase the severity of the symptoms,—above all, long-continued rest, even standing; cold, cold baths, and also great heat, will produce this result. Of great importance in the production of the spasm are psychical excitement, sudden fright, sudden sensory impressions, as a false step, etc. Moderate active exercise tends to ameliorate the condition of the patients. In every other way they appear normal; no other symptoms of nervous disorder are ever present; nutrition, etc., is perfect. Objectively we find disorders present which also are entirely confined to the voluntary motor system. The muscles show an increase in size, in most cases a truly athletic development; the force of the muscles is, however, always less than their size would lead us to suppose. Fibrillary twitchings or disorders of sensation are not present. The tendon reflexes are generally normal, sometimes reduced, and even vary at different times in one and the same patient. Peculiar changes in the mechanical and electrical excitability of the muscles are found, which have been summed up by Erb under the name of myotonic reaction (My.R.) The mechanical excitability of the nerves is normal, that of the muscles is increased. In our case, a blow with the percussion-hammer produced a slow contraction of the irritated fibres, they remaining contracted and forming a distinct groove in the muscle, which lasted from twenty to forty seconds. The faradic excitability of the nerves is also normal, that of the muscles is increased and altered. Here, also, a slow, tonic, persistent contraction occurs. Very strong currents produce persistent contractions from both nerve and muscle. Single opening shocks always produce quick, lightning-like contractions from nerve and muscle. The galvanic excitability of the nerves is normal, while the muscles show an increased excitability with qualitative change. The kathodal closure contraction and anodal closure contraction are about equal, or the A.C.C. is stronger than the K.C.C. Marked local furrows and depressions are formed under the excitation electrode. Erb in his cases noted undulatory, rhythmical contractions moving from the kathode, where they originated, to the anode, where they ceased. These contractions followed each other "like the waves of water produced by a falling stone." In our patient they were at no time observed.

Diagnosis.—The diagnosis of the affection now presents no difficulty, on account of the characteristics of the "myotonic reaction:" as far as known, no other affection gives a similar result to electrical examination. Simulation can, as Erb puts it, always be detected by a few blows with the percussion-hammer and a few anodal and kathodal closures with the galvanic current.

Treatment and Prognosis.—No treatment seems to exert any influence upon the affection. Patients in time learn to avoid certain influences, such as cold, damp air, psychical excitement, etc., and to encourage others, as active muscular exercise, and are thus enabled to lead a fairly comfortable life. The affection lasts during the entire life of the patient, and death when it occurs is due to some other affection.

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EXOPHTHALMIC GOITRE.

By JOHN K. MITCHELL, M.D.

The variety of names which have been given to this affection since its comparatively recent recognition shows the uncertainty in which students have remained as to its pathology. Exophthalmic goitre, Graves's or Basedow's disease, Cachexia exophthalmica, Tachycardia strumosa, Exophthalmos anæmicus, are but a few of the titles bestowed upon a collection of symptoms now usually regarded as nervous in their origin.

To the perfect picture of exophthalmic goitre three cardinal symptoms are necessary,—enlargement of the thyroid gland, palpitation of the heart, and prominence of the eyeballs.

Yet, as Trousseau has pointed out, one of these may be wanting, and still the diagnosis be easily made if the others are present. The one most commonly absent is the exophthalmos, as in the case detailed on page 957.

History.—Stokes,[1] who himself added much to the knowledge of this affection, attributes to Flajani (1800) the observation of the coincident occurrence of cardiac trouble with swelling of the thyroid gland. But the "Clinical Lectures" of Graves[2] of Dublin in 1835 introduced a more accurate and extended description, and Basedow[3] in 1840 insisted for the first time upon the diagnostic importance of the three chief symptoms,—palpitation, exophthalmos, and glandular enlargement. The malady has been studied with good results by Charcot,[4] Von Graefe,[5] Trousseau,[6] and others.

Etiology.—No defined cause is known, but many things seem to act predisposingly. Of the first importance is sex. Trousseau quotes from Withuisen[7] fifty cases, of which but eight were males. Ross[8] says "it affects the female twice as often as the male sex;" but this is a proportion of males far greater than that of most observers. In seven cases tabulated by Von Graefe but one was a male. Dr. A. A. Eshner[1] has collected two hundred and twenty-seven cases,—forty-two males and one hundred and eighty-five females. Of seven cases which I have observed myself, none were in men.

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[1] Diseases of the Heart and Aorta, Dublin, 1853.

[2] Clinical Lectures, Dublin, 1835.

[3] Basedow, Casper's Wochenschr. f. d. ges. Heilk., No. 13, 1840.

[4] Gazette Médicale de Paris, 1856.

[5] Archiv f. Ophthalmologie, Bd. iii., 1857.

[6] Gazette Médicale de Paris, 1862; Clinique Médicale, 1865.

[7] I have been unable to verify this reference.

[8] Diseases of the Nervous System, London, 1883, vol. i. p. 709.

Stokes thought that the disease never occurred before puberty, but since his time many undoubted cases have been observed in children, though the average age of occurrence in Dr. Eshner's table was between thirty and thirty-one. Hawkes[2] relates a case in a girl of six years; Pepper[3] has reported "a fully-developed case" in a girl of ten years, Solbrig[4] in one of eight, and Trousseau[5] had a patient aged fourteen years.

Heredity seems to play but a small part, if any, in the production of the disease: in the case of Hawkes the father is said to have suffered with the same affection. The establishment of the menses in girls, especially if there be difficulty, such as dysmenorrhœa or irregular flow, is of importance, and the trouble is of frequent occurrence in female children at this time. The anæmia and chlorosis which are seen so often about this period have also an important share.

Insanity, epilepsy, and hysteria are sometimes associated with exophthalmic goitre. Graves even suggests that *globus hystericus* is due to a passing swelling of the thyroid gland. Long-continued mental strain, anxiety, and mental excitement act also as causes. It is said that the disease has appeared suddenly after violent emotion.

Of still greater moment is the history of a tendency to nervous affections in the family of the patient.

Pathology.—Important changes in the cervical portion of the sympathetic nerve and the lower cervical ganglia have been found in several cases examined post mortem. A diminution of the nerve-elements, together with an increase of the connective tissue, enlargement and inflammatory alterations in the middle and lower cervical ganglia,[6] and great increase of connective tissue about the cervical sympathetic, have been observed. Other students have failed to find any marked changes in the nerves or ganglia.[7] Various alterations in the heart, true hypertrophy, passive dilatation, insufficiency of the valves, fatty changes in the muscular fibre, have all been noted.

The enlargement in the thyroid gland is usually general, though the right side is slightly more affected in many cases. The hypertrophy is never so extreme as in ordinary bronchocele. The arteries are dilated, crooked, and anastomose freely; the veins are varicose. In some of the autopsies they have been found almost obliterated and reduced to mere fibrous cords.

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[1] Jefferson Medical College Prize Essay, 1888.

[2] Lancet, 1861, p. 130.

[3] Transactions of the Medical Society of Pennsylvania, vol. xii., 1879.

[4] Zeitschrift f. Psychiatrie, 1870.

[5] Gazette Médicale de Paris, 1862.

[6] Peter, Gazette Hebdomadaire, 1864, p. 180.

[7] Jaccoud, Pathologie Interne.

The glandular tissue is sometimes itself increased; in the later stages it has undergone a retrograde cirrhotic change. The cells are healthy or contain cysts filled with gelatinous matter. In the orbit the intraorbital tissues are found in an hypertrophied condition, the deposit of adipose tissue usually great, the veins enlarged and overfull. Atheroma of the ophthalmic artery is often present.

In this confusion of results the pathological anatomy remains somewhat doubtful. It has been asserted that the increased rapidity of heart-action is produced by irritation of the accelerator nerves in the sympathetic. Others hold that the swelling of the gland and the protrusion of the eye are both due to paralysis of the vaso-motor portions of the same nerve; and here it must be agreed that Ross is right in saying that it is a somewhat strained interpretation to assume that one set of fibres is irritated by the same lesion that has paralyzed another set. He himself offers the not very much more satisfactory suggestion that in peripheral nerves both paralysis and irritation sometimes happen from the same cause in one nerve, as may be the case in a neuritis.

But the whole subject is in too vague a state and offers too many contradictions for us to do more than mention these differing observations, waiting for further investigation to settle definitely the pathology of the affection.[1]

Symptoms.—The disease, usually slow and chronic in its onset and running a long course, may be acute both in development and in disappearance. This happens in those cases where great excitement or violent emotion has been the cause. In Solbrig's case (referred to above) the patient had entirely recovered in ten days.

Before any external appearances of the disease can be seen, the patient is irritable, hysterical, and complaining often of palpitation, shortness of breath, flushing, and choking sensations. On examination, the pulse is found greatly hastened, beating 100 or 120 times in a minute in the less severe cases, and in the worse ones too fast to be counted. On studying the chest, it will be found that the area of visible cardiac pulsation is increased;[2] the arteries beat violently at the base of the neck, and an epigastric pulsation is seen. On percussion, the præcordial dulness is generally found more extended than is normal; and on auscultation, a soft blowing murmur at the base of the heart, distinctly transmitted into the carotid, and sometimes into the thyroid arteries, is heard. Next in order is the appearance of thyroid swelling, though it may precede the development of palpitation. The right side is slightly oftener enlarged than the left; both may be equally swollen, or the overgrowth may be markedly greater in one lobe than in the other. The tumor is soft, pulsating, and a thrill like that of an aneurism is communicated to the hand on touching it.

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[1] See also Brown-Séquard, Lancet, vol. i., 1875, who has artificially produced exophthalmos in animals; Boddaert, Bulletin de la Société de Médecine de Gand, 1872; Eulenberg, Ziemssen's Cyclopædia.

[2] Trousseau, Clinique Médicale, tome ii.

Exophthalmos comes last in the order of occurrence, and may vary from a mere prominence of the eyeballs to a degree of protrusion so great that the eyelids are unable to close at all and ulceration and destruction of the cornea result. The appearance given by the staring unwinking eye is very strange, and has even caused the patient to be supposed insane.[1] A sign first pointed out by Von Graefe[2] is of very great importance in the early recognition of the disease. On rolling the eyeball downward, the upper lid, which in health moves in perfect accord with it, follows tardily. In exophthalmos mechanically brought about, as by tumors, this does not occur. This would seem to point to the sympathetic nerve as a partial cause of the protrusion, since Müller has proved that the smooth muscular fibres of the upper lid receive their innervation from the sympathetic. The symptom is not invariably present, but Von Graefe has seen it with a very trifling degree of exophthalmos.

With the ophthalmoscope, according to Becker, the retinal arteries are seen increased in size and pulsate visibly.

The order in which these three symptoms are here given, palpitation, thyroid swelling, exophthalmos, is that in which they commonly occur, but the rule is not without exceptions, and, as has already been said, cases are often seen in which one of the three signs is entirely absent.

Some observers have noticed slight increase, others slight decrease, of bodily temperature in the course of the disease. In my own cases a slight rise of temperature has been always present.

Treatment.—In former days the chief remedies against the disease were quinine, iron, and other tonics, on the theory of its cause being in the anæmic state of the sufferer; nor should such means be neglected. Many men have pinned their faith to many drugs: heart tonics and heart depressants, digitalis, veratrum, belladonna, aconite, iodine and its compounds, bromide, arsenic, ice, and hydrotherapy, have all been vaunted, and may indeed all be useful. Trousseau asserts that iodine increases all the symptoms, and recommends digitalis and ice-bags locally applied to the goitre and to the eyes. Recent writers[3] have found tincture of strophanthus of use. Dr. J. D. Ely[4] thinks that he has cured cases with tincture of phytolacca decandra.

The indications would seem to be to decrease the palpitation by removing any external causes which may produce or add to it, to improve the nutrition should the patient be anæmic, and, while thus treating the symptoms which cause distress, to endeavor to reach that which is probably the original cause of the disease by improving or altering from its morbid state the sympathetic nerve. The first end may be best gained by prescribing avoidance of exertion, and, so far as may be, of excitement. In bad cases it is well to put the patient to bed for a time while carrying out other treatment. The sick person should avoid entirely the ascent of steps. A supine position for some hours during the day should be insisted upon. Meanwhile, tincture of strophanthus in small and frequent doses may be given, and the effect very carefully watched. Ice-bags to the eyes, to the goitre, about the base of the neck, and to the cervical spine should be applied for as long as the patient can bear them, several times in the day. This is a remedy which may be dangerous in persons with weak hearts, and must also be watched.

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[1] Graves.

[2] Deutsche Klinik, 1864, p. 158.

[3] Brower, Journal of the American Medical Association, xi., 1886. The author lays least stress on the more important part of his treatment,—galvanism to the sympathetic.

[4] Medical Age, April 10, 1888.

I have over and over seen excellent results from the bold use of tincture of strophanthus, used at first with caution, the patient being kept at absolute rest, under milk-diet, and locally treated by galvanism, as below described, and by the use, twice daily, of thorough massage of the eyeballs and the throat. This latter means is followed by marked and immediate lessening in the circumference of the neck.

Most important and most useful of all is the galvanic current. This is best applied as suggested by Eulenberg:[1] one pole over the cervical sympathetic, the other over the body of the heart. Eulenberg recommends that the negative pole be placed over the nerve in the neck, on either side alternately, or, better, with a *divided electrode* over both nerves at once. This has decidedly the most influence, but I have not been able to see that there was any choice as to the direction in which the current should pass. With either the positive or the negative electrode pressed firmly down at the base of the neck in the worst case which I have seen, the pulse, which had been beating at 125 or 130, would drop in a few minutes to 98. The treatment must be persisted in for a long while. It may be weeks before any improvement appears. It will probably be months before any lasting gain is made. The current-strength can seldom be borne greater than from two to three milliampères.

Besides the rest and avoidance of excitement already spoken of, the diet should be carefully regulated; coffee and tea should be omitted, and little meat eaten. If possible, a country life should be chosen. Eulenberg also advises a course of chalybeate waters, such as those of Schwalbach in Europe or the iron springs of Saratoga.

The exophthalmos is sometimes so great as to need local treatment. Von Graefe suggests painting the upper lid and the space between the eyebrows with tincture of iodine, the inunction of ointment of the iodide of potash, wet compresses, and even in extreme cases tarsorrhaphy. It is not likely in young patients that the exophthalmos would be sufficiently great to call for so severe a measure, but Von Graefe has done the operation in older persons, and Drs. Levis and Roberts have performed it in this country.

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[1] Ziemssen's Cyclopædia, vol. xiv. p. 102.

The following notes from the case-book of the out-patient department of the Philadelphia Infirmary for Nervous Diseases present an example of the kind of cases mentioned above, in which, while one of the cardinal symptoms was absent, there could yet be no doubt of the diagnosis. They are printed by permission of Dr. Wharton Sinkler, in whose clinic they were taken.

L. A., aged 14. An excellent personal and family history was given, the patient's own health having been perfect until about one year since, when she first had slight menstruation. At about the same time she noticed a rapidly-increasing shortness of breath on very slight exertion. The menses were irregular. The girl was unusually irritable and nervous. After some months a slight swelling of the throat appeared.

When brought to the clinic she had a pulse of 120, a blowing murmur heard in the arteries of the neck and over the body of the heart, much increased by the slightest exercise. The tumor, which was decided but not very large, was perfectly bilateral, soft and pulsatile, and communicated a marked thrill to the hand. No exophthalmos. She was ordered galvanism to the sympathetics three times a week. After a few applications it was noted that within two or three minutes after the current began to pass the pulse fell to 108. Persisting with the treatment, a still more marked improvement took place, and at the present time the promise for a permanent cure seems bright. Her general condition is also greatly better under an ordinary tonic treatment.

HYSTERIA.

By CHARLES K. MILLS, M.D.

DEFINITION AND GENERAL CONSIDERATIONS.

HYSTERIA will be considered as occurring in children before the age of puberty. In the "American System of Practical Medicine" is a general discussion of the affection by the writer, in which juvenile hysteria is not neglected, but it will here be treated of more fully.

In children, as in adults, hysteria may be defined as a functional nervous disease, characterized by special symptoms, any or all of which are related in varying degree to abnormal psychical conditions. Hysteria, like insanity, is therefore possible when the mind has sufficiently developed for the child to be affected for good or evil by its environment, and cases properly called hysterical have been observed in very early childhood. Gillette[1] has reported a case of hysterical paralysis in a girl only eighteen months old. Hysterical hyperæsthesia, neuralgia, aphasia, aphonia, and other phenomena have been recorded almost in infancy. Abortive or incomplete hysteria is more common in boys and girls before puberty than is the fully-developed disease, by which is meant the affection showing various grave, correlated phenomena, such as severe spasm, hemianæsthesia, amblyopia, and paresis. The neurosis in young children is more restricted than in those older, in accordance with their smaller capacity, education, and experience. Hysteria is largely a disease of impulse and feeling, and, as these are less under control in the child than in the adult, flitting hysterical manifestations must be common at an early age; but even the gravest form of the disease may occur in childhood.

Briquet, according to Jacobi,[2] states that twenty per cent. of his cases occurred in children, recording eighty-seven cases, all girls, between five and twelve years of age; Amann collected sixteen from eight to fifteen years in a total of sixty-eight; Althaus, seventy-one below ten years out of eight hundred; Landouzy, forty-eight from ten to fifteen years out of three hundred and fifty-one ; and Scanzoni, four below ten years and thirteen between ten and fifteen years out of two hundred and seventeen.

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[1] New York Medical Journal and Obstetrical Review, 1882, vol. xxvi. p. 66.

[2] American Journal of Obstetrics and Diseases of Women and Children, June, 1876, vol. ix. No. 2, p. 235.

The literature of hysteria in children is not extensive. A lack is particularly to be observed in treatises on diseases of children, as in those of Meigs and Pepper,[1] West,[2] and others. Henoch[3] considers hysterical affections in children at some length, Ashby and Wright[4] have a short but practical chapter, and Goodhart[5] reports a case or two under the head of functional nervous disorders. More attention has been paid to the subject by neurologists and alienists. Georget[6] recorded observations in 1824, and cases were reported by Landouzy[7] in 1846, and, as above mentioned, by Briquet,[8] whose great work on hysteria appeared in 1859. Jolly[9] holds that in childhood, long before puberty, well-marked hysterical phenomena occur, endorsing the opinion of Briquet that in one-fifth of all cases the development of hysteria takes place before the twelfth year. Rosenthal[10] believes in its frequency in young girls, but has observed only two instances in boys.

Articles on hysteria in children, but in small number compared to the immense literature of the general subject, have appeared in German, French, Italian, English, and American medical journals. Among German writers the subject has been discussed by Henoch,[11] Smidt,[12] Mendel,[13] Riegel,[14] Herz,[15] Riesenfeld,[16] Pukler,[17] Engelsburg,[18] and Hirschfeld.[19] Cases have been reported by Italian observers, as by D'Abundo.[20] Bourneville and D'Olier[21] in 1880 reported the case of a young boy stricken with hystero-epilepsy offering all the phenomena manifested by a woman or a young girl, and as early as 1882 Charcot[22] published a lecture on hysteria in young boys; Bourneville and Bonnaire[1] also have reported interesting observations on hystero-epilepsy in a young boy, the case having been cured by hydropathy with gymnastics and internal treatment. Casaubon[2] in 1884 wrote his thesis on hysteria in young boys, and Peuzniez[3] in 1885 his on hysteria in children, collecting the most important facts on the subject.

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[1] Practical Treatise on the Diseases of Children.

[2] Diseases of Infancy and Childhood.

[3] Lectures on Children's Diseases, translated by John Thomson, M.B., etc., New Sydenham Society, London, 1889.

[4] Diseases of Children, Medical and Surgical, London, 1889.

[5] Guide to Diseases of Children, edited by L. Starr, M.D., Philadelphia, 1889.

[6] De l'Hypochondrie et de l'Hystérie, 1824.

[7] Traité complet de l'Hystérie, 1846.

[8] Traité clinique et thérapeutique de l'Hystérie.

[9] Ziemssen's Cyclopædia of the Practice of Medicine, Amer. ed., 1877, vol. xiv.

[10] Clinical Treatise on Diseases of the Nervous System.

[11] Wien. Med. Presse, 1881, xxii. 916–918, and op. cit.

[12] Jahrbuch f. Kinderh., 1880, Bd. xv. H. 1.

[13] Deut. Med. Wochenschr., No. 16, April 17, 1884.

[14] Zeitschr. f. Klin. Med., quoted in Archives of Pediatrics, July, 1884.

[15] Wien. Med. Wochenschr., No. 46, November 14, 1885.

[16] Ueber Hysterie bei Kindern, Diss., Kiel, 1887.

[17] Wien. Med. Wochenschr., 1888, xxxviii. 431–433.

[18] Ibid., 1888, xxxviii. 459–461.

[19] Internat. Klin. Rundschau, September 23, 1888.

[20] La Riforma Medica, Rome, June 4, 1888, No. 128.

[21] Le Progrès Médical, 1880.

[22] Ibid., 1882, x. 985, 1003.

Among English authors Wilks[4] and Gowers[5] in their text-books, and Thompson,[6] Roberts,[7] Barlow,[8] and Springthorpe[9] in journals, have discussed the subject. While American authors on hysteria in children have not been numerous, some of the papers have been original in method of treatment, as, for instance, those of Jacobi[10] and Shaffer.[11] Dessau,[12] Carreau,[13] Gillette,[14] Lee,[15] Bemiss,[16] and Weir Mitchell[17] have recorded interesting observations.

ETIOLOGY.

The old classification into predisposing and exciting is as good a subdivision of the causes of hysteria in children as any other, although it is sometimes difficult to separate one of these classes of causes from the other. Bad education or training, climate, social condition, traumatism, or masturbation might in a certain case be either a predisposing or an exciting cause.

Among the most important predisposing causes may be ranked heredity, improper educational methods, neglect of physical health, the ill effects of bad example, unusual hardship, climate, and depraved conditions of the blood.

The influence of heredity as a predisposing cause of hysteria is generally recognized. The neurotic constitution is, after all, the most frequent predisposition to hysteria, and Briquet, Amann, and others give valuable statistics to show the more or less direct transmission of the disease from parents to children, particularly from mothers to daughters. In such cases both the inheritance and the influence of parental example may assist in producing the disorder. Some years ago I saw in consultation a striking example of this,—a typical case of hystero-epilepsy in a girl eleven years of age, whose mother I had treated several years before for the same affection. The physician in attendance at first regarded the case as one of meningitis, probably tubercular. The spasm was very similar in many of its features to that from which the mother had suffered. She, like the mother, had the hystero-epileptoid status, passing from one attack to another for several hours. After a careful review of the symptoms and the history of the case, the doctor became convinced with me that it was one of grave hysteria. She, like the mother, made a complete recovery.

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[1] Le Progrès Méd., 1882, x. 645–648. [2] Thèse, Paris, 1884. [3] Ibid., 1885.

[4] Lectures on Diseases of the Nervous System, 1883.

[5] Epilepsy and other Chronic Convulsive Diseases, 1885; also, A Manual of Diseases of the Nervous System, 1888.

[6] Lancet, November 3, 1877.

[7] Practitioner, London, November, 1879.

[8] British Medical Journal, December 3, 1881.

[9] Australasian Medical Gazette, January, 1885.

[10] American Journal of Obstetrics and Diseases of Women and Children, 1876.

[11] Archives of Medicine, December, 1879; February, 1880; April, 1880.

[12] American Journal of Obstetrics and Diseases of Women and Children, October, 1880.

[13] Ibid., etc., 1881, vol. xiv. p. 504.

[14] New York Medical Journal and Obstetrical Review, 1882, p. 66.

[15] Ibid.

[16] New Orleans Medical and Surgical Journal, October, 1886, p. 255.

[17] Lectures on Nervous Diseases, etc.

The inheritance of hysteria, as of other nervous diseases, is often not direct. Grasset,[1] for example, has shown the connection between the tubercular diathesis and hysteria, believing that hysteria is often the manifestation of this diathesis. The children of the insane, the epileptic, the alcoholic, and of those suffering from certain organic diseases, particularly affections which insidiously undermine nervous stability, may become hysterical. Children of perfectly healthy parents resist the development of hysteria, even under strong exciting causes. The hysterical constitution is a morbid heritage.

The neglect of the physical health of children is a frequent cause of hysteria, particularly in its minor manifestations. Poor or badly-selected food, imperfect ventilation, too little sunshine, overheating, or exposure, want of cleanliness,—in brief, bad infantile and juvenile hygiene,—lead to the development of hysteria, as to a multitude of other affections.

Habitations and the particular rooms in houses assigned to the children have sometimes a marked influence in the development of hysteria. The children of the poor are compelled to live as best they can, but the rich and middle classes and even the poor can improve the chances for nervous and general health by attention to the opportunities within their reach. If children are compelled to be a large part of their time within-doors, whenever possible the most healthful rooms should be set aside for their use. Chilly, sunless, badly-ventilated rooms sap nervous vitality; rooms of good size and southern exposure should be given preference. Just as the sick, particularly of certain classes, get better in sunny rooms, so those in health, particularly children, will retain their health and powers of resistance to disease in pleasant living-rooms.

"It is really surprising," says a recent writer,[2] "to see the amount of trouble and pains bestowed on the proper housing and feeding of horses and dogs or other domestic animals, while at the same time comparatively little attention is paid to these matters with regard to the rearing of children. Model stables and model kennels abound, while the model nursery is almost unknown. Warming, ventilation, and aspect are all subjects which are thoroughly considered in the stable, while as regards the nursery they are generally left for chance to decide,—though the health of a child is surely more important than that of a horse or a dog."

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[1] Brain, January, April, and July, 1884.

[2] Jessie Oriana Waller, in Nineteenth Century, 1889.

Bad educational methods may act both as predisposing and as exciting causes in children as well as in adults. "Both in our private and public educational institutions the conditions are frequently such as lead to the production of hysteria or confirm and intensify the hysterical temperament. In our large cities all physicians in considerable practice are called upon to treat hysterical boys and girls, the latter more frequently, but the former oftener than is commonly supposed. Hysteria in boys, indeed, does not always meet with recognition, from the fact that it is in boys. Cases of hysteria in girls under twelve years of age have come under my observation somewhat frequently. About or just succeeding examination-time these cases are largely multiplied. The hysteria under such circumstances may assume almost any phase; usually, however, we have not to deal in such patients with convulsive types of the disease."[1]

Social conditions are occasionally active in the development of hysteria. While not a disease of the rich, it is, on the whole, more likely to occur among either the children of the rich or those who, while not wealthy themselves, are willing to sacrifice unduly in order to over-indulge their children. Children who are too much pampered and allowed their own way are sometimes the victims of hysteria. In our large cities, in which all houses are so poorly supplied with grounds, yards, or courts, for out-door exercise, the children even of the well-to-do develop hysteria in the winter and early spring because of undue confinement within-doors. In-door games and amusements failing or cloying, they indulge too much in reading and in effeminate plays. The life of a child should be made as natural, healthful, and happy as possible, in order to provide against nervous breakdown, and this can be accomplished only by a proper admixture of in-door and out-door life, which is too often practically impossible in the winter in cities. If parents cannot supply their children with the amount of air and exercise needed for their health while in the city, they should make every effort to send them to the country or sea-shore early in the spring and for a long period.

Either an enervating climate or one of great variations may develop hysteria in the young. Certain seasonal influences are potent in its development. Many observations have been made by Mitchell,[2] Lewis,[3] and others on the effects of climate and seasons upon chorea, and the facts obtained have about the same significance for hysteria; in fact, the chorea of children is not seldom hysterical in nature. Hysterical chorea and hysteria of other forms are, on the whole, more likely to develop in the spring, and particularly after a severe winter.

Anæmia, chlorosis, or the strumous habit may be the groundwork of hysterical seizures in children, and it is for this reason that iron, arsenic, iodine, the hypophosphites, cod-liver oil, and careful feeding, with abundance of fresh air and sunshine, will often do much for such children. The mistake must not, however, be made of supposing that anæmia or spanæmia is a constant or an almost universal cause of hysteria in children. Well-nourished or overfed children sometimes suffer from hysterical manifestations, and, like the pampered poodle dogs, do better on a little starving and cuffing than on too much care and coddling.

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[1] The writer, in American System of Practical Medicine.

[2] Lectures on Nervous Diseases, etc.

[3] Polyclinic, January, 1887, vol. iv. p. 205.

Various disturbances of the sexual organs have long been recognized as both predisposing and exciting causes of hysteria in children. Masturbation is undoubtedly very common in young boys, and, while it seems unless long continued to have little appreciable effect upon those who are robust physically and mentally, on the weak and sensitive it produces various forms of nervous break-down which may show themselves as major or minor hysterical affections. By some too much and by others too little stress is laid upon this cause. The constant irritation kept up by an adherent prepuce is a cause of hysteria which has attracted abundant attention; it is one of the peripheral sources of hysterical symptoms or paroxysms, and perhaps a more frequent one than many other local irritations, because the sexual organs and acts are the object of abnormal interest to both children and adults of hysterical tendencies. Sexual irritation sometimes acts directly through the mind, children from lewd conversation or reading, or the observation of indecent performances, becoming erotic and hysterical. While parents and guardians should pay strict attention to the protection of children from causes of this kind, they should be careful, on the other hand, not to lead those innocent of any knowledge or thought of such matters to their untimely consideration. Many children in the second year of life, or even earlier, practise masturbation; and this, while it may not be the special cause of the disease, by over-exciting the nervous system may prepare the way for its development.

In children, as in adults, imitation, mimicry, or nervous contagion often plays an important *rôle* in the production of hysteria. In this way have originated many of the epidemics and endemics of various ages and countries, some of which particularly affected children, as the child pilgrimages and dancing manias of the Middle Ages, with their wanderings and sufferings, their revelations and ecstatic seizures. These pilgrimages took place for more than two centuries, and nothing to be compared to them in the numbers taking part and the wide diffusion of the affection has occurred in modern times. On a smaller scale, however, endemics of hysteria from imitation have frequently occurred, and some of them have been put on record. One of these was an outbreak in a church home for children in Philadelphia, which has been described by Mitchell (*op. cit.*). A more recent outbreak, which attracted considerable popular attention, was in February, 1889, in a school for soldiers' orphans at McAllisterville, Pennsylvania. The newspapers contained sensational accounts of this disorder, which they attributed to diverse causes. The patients, who were all boys, indulged in striking and kicking, running, and damaging furniture. One common manifestation was the calling of everything by a single name. One boy was attacked after another, and the effect of imitation was often distinctly traceable. It was supposed that many of the cases were instances of deliberate shamming or feigning, and some of the boys were persuaded into confessing that this was a fact; and doubtless in every endemic or epidemic of this kind some cases will be due to deliberate shamming, and others to involuntary imitation or neuro-mimicry. The affection gradually disappeared.

Roberts[1] relates an interesting experience illustrating this tendency to imitation or nervous mimicry. A boy thirteen years old had an hysterical bark which degenerated into a hoarse sound resembling the bleating of a goat, this continuing, with some variations as to time of day, for fifteen months and then gradually subsiding. The boy was separated as much as possible from his brothers and sisters, but on one occasion passed some days with his elder brother, and four months afterwards this brother also had an attack of hysterical barking, which lasted a fortnight. He had a sister nine years old, who four years after her brother's recovery also began to bark and to show other signs of hysteria. The hysterical bias was inherited from the mother, who in her youth had displayed severe hysterical symptoms.

As Roberts says of this family, it is important to note how hysteria moulds its manifestations, by unconscious mimicry, on a contiguous model, in all three cases the disorder affecting almost exclusively the diaphragm and larynx, and almost exactly in the same fashion. "This is quite in harmony with the history of this great neurosis. When hysteria breaks out—epidemically, as it were—in a school or nunnery, all the cases develop the same type of manifestations as those exhibited by the individual first attacked."

Mitchell relates the case of a lad eleven years of age, whose sister nine years old, an epileptic, had an hysterical attack as the result of running a nail into her foot, which seemed to have impressed him with the idea that he was afflicted in the same manner. After this he frequently had spasms which were lacking in the diagnostic marks of epilepsy, and which were cured by the cold douche and the threat of the application of the hot iron. Another patient after an attack of ague began to limp and complain of pain in the right knee; hip-joint disease was diagnosticated, and the child was taken to a surgical institute, where she grew worse and developed pain, hyperæsthesias, pseudo-palsies, and contractures, with at times attacks of hysterical spasm. She was removed to quiet lodgings and got well.

Hysterical symptoms sometimes develop in children apparently as the result of their being in the company of those older than themselves. They see, hear, imitate, what is done by their elders, without being able properly to understand and correlate such actions. Their minds cannot digest and make good use of the observations and experience of a companionship not fitted for their years.

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[1] Practitioner, London, November, 1879.

The lack of moral training received by the children of hysterical mothers is another cause of hysteria. That any ill treatment, moral or physical, may cause hysteria in children goes without saying; but the chief illustration of the truth of this assertion is to be seen in the children of hysterical parents or of those who are diseased by alcohol or are the victims of some form of mental or moral perversion. In schools, asylums, almshouses, and hospitals hysteria sometimes results from ill treatment.

Hysteria may show itself not only in the course of an acute disorder, but also during the time of depression and weakness when the child is convalescing. Every practitioner has seen cases of this kind, and, indeed, in its minor forms the affection under these circumstances is so common as scarcely to be considered worthy of more than passing attention. Some of these cases, however, assume grave appearances. Roberts (*loc. cit.*) mentions the case of a boy between eight and nine years of age who, when recovering from a fever, was seized with paroxysms of loud, passionate, and tearless crying, with incoherent ravings of the most distressing character. In the intervals between the paroxysms he appeared quite well. He was sent from home, an interrupted galvanic current was employed, and in about six weeks the paroxysms ceased altogether. Riegel[1] narrates five cases of hysterical affections in children, three of which were in boys from ten to fifteen years of age and were sequelæ of other diseases. The difficulty in these cases was in respect to the use of the lower extremities. The patients could not walk, but bent their knees together as soon as they were placed upon their feet, and yet no disease was discoverable. In one of them there were regular recurring clonic convulsions.

Traumatism is undoubtedly a cause of hysteria both in children and in adults. Injuries may give rise to a great variety of hysterical manifestations, from an isolated ache or numbness to the grotesque train of grave phenomena which occur in hystero-epileptics.

A young girl seen in consultation had a clear traumatic history. Two years before coming under observation she had fallen on the ice and struck her head upon a marble step, cutting it severely, after which she suffered with headache and occasionally was light-headed and had great pain near the scar. Later she began to have spasms, the first occurring in a field where she had hold of a rope with a dog. The only thing she remembered was a sharp pain through the scar. Her eyes hurt her, and she could not see to read. During five months she had many spasms, sometimes every day, sometimes not for a week. The scar was removed, and she had no spasm for five weeks, but after this the spasms recurred. She eventually recovered under treatment directed to her hysterical condition.

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[1] Arch. f. Kinderh., from Zeitschr. f. Klin. Med., Bd. vi. H. 5, Bd. v. H. 7 u. 8. Quoted in Archives of Pediatrics, July, 1884, vol. i. p. 486.

In not a few cases hysteria is developed secondarily to traumatism, particularly in children. The child receives an injury,—a blow, a fall, or a twist,—and this results in some real affection, usually pain, heat, swelling, contracture, or interference with motion. In time and under treatment the real disease disappears, but through the impressionable nervous system of the child there is immediately developed a case of neuro-mimesis; or it may be weeks or months after recovery from the effects of the accident that an hysterical affection which bears the impress of the former organic disorder is first observed. This mode of origin is somewhat frequent in affections of the spine and joints. The physician should inquire not only as to recent injury directly connected with the attack, but also as to accident and inflammatory symptoms at some more remote period.

Frequently the beginning of a train of hysterical phenomena is attributable to some sudden fright, either by day or by night. The more recent studies of hypnotic phenomena in connection with hysterical subjects have thrown some light upon the effect of fright in establishing the grave hysterical condition. "Who knows," says a recent writer,[1] "how many pathological states (not simply nervous and functional ones, but organic ones too) may be due to the existence of some perverse, buried fragment of consciousness obstinately nourishing its narrow memory or delusion, and thereby inhibiting the normal flow of life?" This writer gives an illustration from a book by Pierre Janet on "Psychological Automatism." A girl of nineteen suffered with a series of the gravest hysterical symptoms. Janet threw her into a deep trance, and succeeded in calling up her early memories. In the deep somnambulism she explained three things: "Her periodical chill, fever, and delirium were due to a foolish immersion of herself in cold water at the age of thirteen. The chill, fever, etc., were consequences which then ensued; and now, years later, the experience then stamped in upon the brain for the first time was repeating itself at regular intervals in the form of an hallucination undergone by the sub-conscious self, and of which the primary personality only experienced the outer results. The attacks of terror were accounted for by another shocking experience. At the age of sixteen she had seen an old woman killed by falling from a height; and the sub-conscious self, for reasons best known to itself, saw fit to believe itself present at this experience also whenever the other crises came on. The hysterical blindness of her left eye had the same sort of origin, dating back to her sixth year, when she had been forced, in spite of her cries, to sleep in the same bed with another child, the left half of whose face bore a disgusting eruption. The result was an eruption on the same parts of her own face, which came back for several years before it disappeared entirely, and left behind it an anæsthesia of the skin and the blindness of the eye."

Through studies of this character we may eventually get clearer light upon the influence of fright, chagrin, grief, regret, and all other painful moral impressions in producing a temporary or even a persistent hysterical condition. In some impressionable children—those with plastic, ductile nervous systems—in whom hysteria is likely to develop, a tremendous impression made on consciousness may result in that distortion of the nervous system which is called hysteria.

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[1] Prof. William James, Scribner's Magazine, March, 1890.

PATHOLOGY.

Cases of chronic hysteria have been reported in which autopsies and the microscope have shown degeneration of either nerve centres or tracts, but these have not been strictly or not solely hysterical. Hysteria and organic disease may have been complicated, or, in some of the grave forms, chronic hysteria may have developed real pathological changes in the cerebro-spinal system ; but such cases, from their nature and long continuance, have been in adults. Hysteria in young children has therefore no morbid anatomy that can be discriminated either by scalpel or by microscope, although temporary changes in the nervous system must be present. A summary of views elsewhere given[1] as to the pathology of hysteria is as applicable for the disease in children as in adults,—namely : the anatomical changes in hysteria are temporary ; they may be at any level of the cerebro-spinal axis, but are most commonly and extensively cerebral ; they are both dynamic and vascular, and the psychical element must enter into the explanation of nearly all cases.

Great differences have prevailed as to the proper method of interpreting certain grave hysterical phenomena which sometimes are observed in young children,—although not so frequently as in youths and adults,—such as paralysis, contracture, convulsions, anæsthesia, and disorders of the special senses. The question has been frequently discussed, and antagonistic opinions have been given by capable observers. Can such phenomena as anæsthesia and contracture be fully explained on the theory of deception, expectant attention, or imagination in the sense in which the word is ordinarily used? Can such phenomena as stigmatization and ischæmia be wilfully produced? While some of the symptoms shown by hysterical patients may be thus produced or be grossly exaggerated involuntary phenomena, it is impossible to explain all in this way. It is impossible that a patient shall, with eyes blindfolded, by a mere effort of will repeatedly designate the same boundary for an anæsthetic area, as, for instance, the median line of the body. Ischæmia,—in which we have an arrest or partial arrest of circulation, with pallor, coldness, and inability to produce bleeding by puncture,—while it may be occasionally emotional, is not in any true sense a controllable phenomenon. We must therefore, with Coupland, take the ground that many, if not most, of the grave phenomena exhibited by hysterical subjects should be regarded as facts, not as malingered or simulated symptoms.

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[1] American System of Practical Medicine, vol. v.

"In spite, however, of the mystery that hangs over the causation of hysteria and of its precise pathology," says Coupland,[1] "there are certain facts of observation connected with it about which there can be no manner of dispute. Observed and recorded in numberless instances, these facts surely are real enough, and probably have a real pathological change at their root; and to dismiss the disease as unworthy of consideration or to question the reality of its manifestations is surely to abrogate that spirit of scientific inquiry which should enter into all investigations into morbid conditions."

In children, perhaps more than in adults, subjective hysterical symptoms may, however, be exaggerated, simulated, imitated, or induced, to gain sympathy or gratification, or, it may be, merely from a general morbid tendency; but it is both unscientific and misleading to regard all hysterical phenomena in children as of this character.

The question of hypnotism in young children is one of some importance in the consideration of the pathology of hysteria. Many hysterical conditions are practically states of hypnosis. It is said to be easier to hypnotize young persons, especially from seven to twenty-one years, and it was upon this account that the ancient Egyptian, Greek, and Roman priests and the Indian fakirs preferred to employ young children in their initial ceremonies. Out of seven hundred and forty-four persons of different ages who were tested by Liébault in one year, he succeeded in throwing six hundred and eighty-two into a more or less deep hypnotic state, and of the sixty-two who proved non-hypnotizable none were under fourteen years of age.[2] Children are then hypnotizable as they are hysterical: the one fact presumes the other.

As Blocq (*op. cit.*) has put the matter, the explanation of many hysterical manifestations in children is to be found in the imperfect development of the cerebral centres. The inhibitory action of the cortex on the lower centres is then at a minimum, and the child's nervous system therefore responds with too great readiness to all excitants.

SYMPTOMATOLOGY.

A study of reported cases and of personal observations shows that certain phenomena are likely to predominate in the hysteria of early life; for according to the degree of development of certain mental faculties, particularly of the will and intellect, must be the diversity and permanence of nervous manifestations in children.

Hysterical symptoms can be classified as psychical, motor, sensory,—including perversions of the special senses,—vaso-motor, secretory, and miscellaneous,—such as vomiting, phantom tumors, and the mimicry of acute diseases. In children, as well as in those older, these symptoms may be involuntary, artificially induced, acted or simulated through irresistible impulse, or acts of deception or imitation. Probably in children hysterical phenomena most frequently are either involuntary or clear cases of deception or imitation; although I do not believe with Roger that every neurosis by imitation or mimicry is a neurosis by simulation. Direct imitation undoubtedly plays a greater part with children than with those of more mature years.

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[1] Lancet, November 3, 1877.

[2] Björnström, Hypnotism, 1889.

Before taking up symptomatology in detail, it will be well briefly to compare and contrast hysteria in children with the disorder at puberty and adolescence and in adult life.

The psychical phenomena of hysteria in childhood are not so intense, persistent, or multiplex as in older patients, and therefore a true continuous hysterical insanity lasting for weeks or months is not likely to be observed in early years. The tremendous emotional and motor excitement shown in such an affection as acute hysterical mania of severe type cannot be sustained by the weak physical and mental powers of childhood, as no functional disease can exceed the potentiality of the individual. The excited speech, the violent gesticulation, the calculating deception, and the exhausting dramatism—whether such phenomena be voluntary, involuntary, or induced—cannot be long maintained by the child. While hysterical mania in the young child may occur, it can only be a passing storm, less destructive and less ostentatious than in the adult. Some of the more chronic forms of hysterical insanity are somewhat frequently observed in children. Certain impulsive and particularly imitative tendencies are highly developed in children, and hence we have many accounts of hysterical epidemics or endemics among children; but the comparatively slight endurance of juvenile hysterical phenomena is evinced by the fact that, under well-directed physical and moral treatment, even such outbreaks have been quickly subdued.

The motor phenomena of hysteria include general convulsions, local spasmodic disorders, chorea, tremor, ataxia, paralysis, and paresis. Hysterical general convulsions occur before puberty, but they are not so frequent as at and after that period, and, when they occur, are not so likely to be typical hystero-epileptic attacks. Gowers says that hysteroid attacks are common in girls at ten and twelve years of age, but such is not my experience in this country. Local spasmodic affections, such as twitching of the nose, of the eyelids, and of the mouth, general facial twitchings, and spasm of the glottis or even of the larynx, occur with comparative frequency in children. Contracture associated with neuro-mimetic joint- and spine-disease is of common occurrence. Some of the forms of chorea in children are properly designated as hysterical, but most of them are not of this character. Chorea is sometimes simply one of the features of a general hysterical condition. Hysterical tremor is an affection of childhood as well as of puberty, adolescence, and adult life, although it is more common after than before puberty. I have never known hysterical tremor to last for months in childhood. Some cases of paralysis in children have been reported in which tremor also has been present, and occasionally tremors more or less persistent are seen in children who have been subjected to sudden and extreme fright. Hysterical ataxia severe in type is rare, but slight forms of ataxia, so slight as to be scarcely deserving of the name, are oftener observed. In some of these cases it is difficult to say whether they should be classified as ataxia or chorea.

Paralysis occurs in children, in rare cases in very young children, but it is by no means so common as in later life. Hysterical hemiplegia in boys and girls has been reported by competent observers, but cases are not numerous under thirteen years of age. Slight forms of weakness or loss of power in the limbs, which can hardly be called palsy, are sometimes seen in hysterical children, passing away in a few minutes, or in some hours at the most. The graver forms of motor paralysis in children are usually associated with hemianæsthesia and other hystero-epileptic phenomena. Few such cases, however, have been reported in this country. Sensory hysterical phenomena are probably more common in children than any other manifestations,—hyperæsthesia particularly; and this may be either local or general. Headache, neuralgias, and localized subjective pains are also frequent. Hysterical coxalgia has been reported. Anæsthesia is not so common, but Charcot, Thompson, Barlow, Goodhart, and others have recorded cases. Hysterical achromatopsia, blindness, and deafness, usually unilateral, are comparatively frequent. Vaso-motor affections are rare, while some disorders of secretion are common, as, for instance, disturbances of the urinary secretions. Hysterical vomiting and phantom tumors rarely occur. Goodhart, however, has seen functional vomiting and an extreme case of functional hiccough in girls of ten and twelve years respectively. In one case detailed by Henoch, a girl eleven years old had violent attacks of retching, with hæmatemesis, during which half a cupful of blackish-red blood was brought up. Nothing wrong could be detected by examination of the lungs, teeth, throat, or tongue. Henoch believed that it was not an attack of simulation, but one of hysteria occurring after mental excitement. She was cured apparently by the psychical, or perhaps by the painful, effects of ergotine injections.

For the purposes of study we separate the symptoms of hysteria, but, while they may sometimes, they do not generally present themselves as isolated phenomena, but rather as a syndrome, including several of the important sensory, motor, psychical, or other phenomena. When, for example, decided anæsthesia, either for touch, pain, or temperature, is present, it is usually in association with motor paresis and with perversions of the special senses and of the psychical sphere. Contracture is usually in association with neuro-mimetic joint-affections, and sometimes also with a whole train of motor, sensory, and other manifestations. Often the diagnosis of hysteria can be made by a careful consideration of the fact that, while one symptom in a given case may be in doubt, another or others are hysterical, and therefore probably the doubtful should also be thus classed. On the whole, isolated phenomena are more frequent in early life than later. This is particularly true of hysterical pain or hypersensibility.

Let us now take up some of these symptoms or symptom-groups in greater detail. Of the psychical manifestations little more need be said than has been said already; but it cannot be too often repeated that the psychical element is a part of every hysterical manifestation.

A form of hysterical insanity frequently observed in young children is that in which the wits of the child are perversely used for the purpose of attracting attention and creating excitement by some outrageous or extraordinary series of events, as the ringing of bells, rappings and knockings, destruction of furniture, etc. Children are found concocting an elaborate system of fraud and deception, spurred on by the hysterical tendency.

Great mental excitement, sufficiently marked to be classed as mania, is observed in hysterical children. In a school-girl eleven years old, after an attack of typhoid fever, spells of crying and of great excitement and violence began. She would strike other children and threaten to commit suicide, became morose, and complained of headache. She recovered on a roborant treatment. West mentions the case of a girl seven years old, ambitious to learn, in whom the first signs of an overtaxed brain appeared in extreme irritability and causeless attacks of fury. She suffered also from chorea on both sides of the body, but not severely; she would sometimes stumble and fall. She developed severe headaches which lasted a short time. She was morbidly solicitous about her own health and disposed to exaggerate the slightest ailment. Fortunately, her mother was a sensible woman, and by judicious treatment, taking her to the country, and interesting her in botany and pet animals instead of books, accomplished her recovery.

Therio-mimicry, or the mimicry of animals, is sometimes a striking feature of the endemic or epidemic form of hysteria in children. "The noises and actions of animals," says Gowers,[1] "are strangely imitated. The patient mews like a cat, or, much more commonly, barks like a dog. Still more frequent is a tendency to bite. Patients sometimes, but rarely, bite themselves. I have known the lip to be bitten, and two patients bit their own fingers, one so severely as to leave a permanent scar an inch long. Very commonly, however, the patients try to bite other persons, and the tendency renders considerable care necessary on the part of the attendants. Not only do they bite, but do so in a curiously animal manner. A lad of sixteen years, after failing in an attempt to bite an attendant's hand, seized the corner of the pillow between his teeth, and, throwing his head backward, shook the pillow just as a dog shakes a rat, or as a lion shook Livingstone. This therio-mimicry may be in part truly mimetic, but it seems to be the part of a manifestation of some strange animal instinct which we possess in a latent or modified condition, like our canine teeth."

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[1] Manual of Diseases of the Nervous System.

Blocq,[1] under the title of juvenile hysterical mania, considers the question of the mental disorders of grave hysteria in the young, recording an interesting case; but the report is, after all, simply another example of grave hysteria, like the cases so much discussed by Charcot. The boy presented paresis, twitchings of the limbs, hyperæsthesia, dilatation of the pupil, and double concentric contraction of the field of vision, more marked on the left. Nervous ill health began after an attack of typhoid fever which occurred at two and a half years of age. The child began to suffer from headaches, and was never in perfect health; but he was not attacked until thirteen years old with hysterical paraplegia, pains in the left knee and in the lumbar spine, and later with contractures and hystero-epileptic attacks with periods of violent mental excitement. In one of the latter attacks he was stricken with delirium and unconsciousness, recognizing no one, babbling constantly in incoherent fashion, and having spells of fury in which he wished to strangle his nurse, committing also absurd acts, trying to drink his urine, etc. This delirious or maniacal state continued for twelve days, after which he gradually recovered, but had for a time a wandering air, some trouble in ideation, was indifferent, miserable, wished to commit suicide, and had lost completely the memory of recent facts. His mind after a time cleared entirely, but he had no remembrance of the events which occurred during the attack. This case presents the features of a form of mental disorder occurring in hystero-epileptics; and a similar attack or series of attacks may occasionally occur in a case without any other history of grave hysterical symptoms.

According to Blocq, this form of hysterical mania often attacks children, and particularly boys; but here it must be remembered that he is speaking of French children: it is certainly rare in this country, although I have seen a few cases similar to the one described. Blocq refers to a memoir of M. Clopatt on infantile hysteria. This author collected two hundred and seventy-two observations on hysteria in children, ninety-six of these being boys. Of this number one-third showed mental disorder. According to Blocq, the mental perversion is somewhat uniform. It appears as attacks in which the convulsive element is sometimes reduced to a minimum, but at other times much prolonged. Often the child presents at first incoherence of words and gestures; he behaves like a madman, constantly babbling and acting absurdly. More frequently his agitation is violent, and he is furious, striking, shouting, menacing, seeking to bite, pinch, beat, or even strangle. Sometimes his monomania takes the form of zoanthropy. Commonly the excitement ceases abruptly, the patient seeming to emerge from a bad dream. Such mental attacks may either succeed or take the place of other hysterical manifestations, and they may or may not coexist with nervous stigmata.

HYSTERO-EPILEPSY, CATALEPSY, ECSTASY, TRANCE, AND ALLIED

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[1] Rev. gén. de Clin. et de Thérap., Paris, 1889, pp. 768–771.

AFFECTIONS.—Hystero-epilepsy, catalepsy, trance, etc., might be considered in this work, as they have been by me elsewhere, as separate affections, owing to the peculiar and uniform array of characteristics which they usually present, but I will refer to these papers[1] for the fuller consideration of these subjects, briefly considering them at this point under the symptomatology of convulsive phenomena.

Hystero-epilepsy is certainly not so common in children before the age of puberty as it is at and after this period. In this country it is comparatively uncommon among children, or at least but few cases have been put on record, but it occurs occasionally, and has been reported even, in the forms described by foreign authors. In the last twenty years I have seen few cases of this classical grave type in children under thirteen years of age, although hysteroid convulsions of irregular type have been a not uncommon experience.

Hysterical or hysteroid and epileptic or epileptoid manifestations are found commingled in various ways, so that it is difficult to classify cases under practical heads, irregularity of phenomena being one of the most striking features of hystero-epileptic or epilepto-hysterical spasmodic disease as observed in this country. For convenience of study, however, the affection might be fairly well arranged in four classes,—namely, (1) hystero-epilepsy with most or all of the phases of the classic Gallic type; (2) hystero-epilepsy in which the hysterical element predominates and in which the manifestations are irregular both in form and in frequency; (3) epilepto-hysteria, a spasmodic disorder distinctly intermediate between hysteria and epilepsy, but in which the epileptic element predominates, and which may pass into incurable epilepsy; (4) a class in which attacks distinctly hysterical and others distinctly epileptic occur as separate events.

While, on the whole, hystero-epilepsy in children under puberty is more common in girls than in boys, it would seem, from a study of recorded cases, that the grave regular type of the affection occurs more frequently in boys. I will summarize a few of these reported cases, not only to illustrate the manner of the occurrence of this disease in boys, but also because the details may serve to some extent as a picture of the symptomatology of the affection.[2]

D'Abundo, an Italian observer, records in great detail the case of a boy ten years old. Some of the most important phenomena were the following. Painful sensations in the abdomen of the boy increased and became intense; he had pain and choking in his throat; he ceased to speak, his eyes were fixed on space, and he replied to no one. This period was characterized by great increase in the number of his respirations; eighty movements a minute were counted, and with each expiration and inspiration came three short interrupted movements. He did not froth at the mouth. Analgesia was present. After three or four minutes the fast breathing ceased and a period of disordered movements ensued. The muscles everywhere seemed to be convulsed; at first rigidity, and then the limbs seemed to fly in every direction and his body assumed an arched position on the bed, his head and feet nearly touching. It seemed as if he wanted to speak, and his hands were placed on the sacro-lumbar region. The opisthotonus decreased gradually, and the boy began to make frightful movements; his face showed variable spasmodic phenomena; he, for instance, contracted the muscles of mastication and elevated and shut the eyelids. Compression of the abdomen at this time caused the phenomena to cease. At other times the attacks were different. The muscles were not so rigid, and no disordered movements were present. His face assumed a great variety of expressions; he appeared to have hallucinations of sight and hearing, and assumed positions of defence and of extreme fright, sometimes difficult and awkward positions as if trying to hear something. All the movements and manifestations were in very quick succession. He would rise from bed, pirouette round, and place all his limbs, his trunk, and even his head in most difficult positions. The seizure usually lasted an hour, or an hour and a half; then he would get up immediately, talk and laugh, and relate all the details of the attack, explaining his illusions and hallucinations.

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[1] American System of Practical Medicine, vol. v.

[2] References to these writers are given in the beginning of the article.

Bourneville and D'Olier, quoted by Charcot, report a case in a boy of thirteen years, who came of a family in which were several idiots and epileptics. In the intervals of his attacks he had left hemianæsthesia with amblyopia, and he showed hysterogenic zones at the bregma, the left iliac fossa, and in the lumbar region. The bregmatic point was the most sensitive. The least shock provoked an attack, and even being thrown to the ground by his comrades would cause one; strong pressure would arrest the attack with the same facility. The attack was regular,—the epileptoid period, the period of great movements, with the arched position, then passionate attitudes, with violent cries. He had from November, 1879, to December, 1880, no fewer than five hundred and eighty-two attacks, without true epilepsy and without any trouble of the intellect, in spite of the frequency of the seizures.

Bourneville and E. Bonnaire have reported observations on a similar case of hystero-epilepsy in another young boy, who was cured by hydrotherapy associated with gymnastics and internal treatment.

Gowers describes the case of a lad twelve years of age, in whom contracture of one limb occurred during the attack, which was always followed by transient paralyses.

Carreau reports a case in a boy eight years old, whose mother was hysterical and whose father had had chorea. The boy was slender, weak, had an uncontrollable temper, never played with other children, was deceitful, would feign sickness, and was vicious. The spasms were alternately tonic and clonic, and he foamed at the mouth. He was anæmic, and had enlargement of the cervical glands, nasal catarrh, and dyspepsia. According to his mother, he had convulsions at irregular intervals, always preceded for a day or two by malaise, constriction at the epigastric region, nervous irritation, and a tendency to cry at the slightest provocation. They always happened in the day, and he had premonitory symptoms. He fell on some selected spot where he was not hurt, and remembered to a certain extent what he did during the paroxysm, though he would not readily admit this. The inter-paroxysmal symptom most complained of was pain in various parts of the body.

Other cases might be given, but no description of a hystero-epileptic attack in child or adult will answer to identify every case. The typical grave attacks in children, as in adults, are usually preceded by prodromes, psychical, organic, motor, sensory, or mixed; and when once the train is started the different periods may succeed,—epileptoid contortions, great movements, emotional attitudes, and delirium,—the last two, however, being more likely to be wanting than any of the others. The case may assume almost any irregularity, especially as regards the motor and sensory symptoms; but usually in all cases certain phenomena are likely to be present, although any one or several of them may be absent; for example, certain sensory phenomena, such as painful sensations, hysterogenic zones, and anæsthesia; perverted respiratory conditions, such as rapid breathing or dyspnœa; spasmodic phenomena, which may vary, but in which certain features predominate, as the occurrence of opisthotonus. The condition as to consciousness is peculiar, although varied. The patients do not exhibit the profound unconsciousness, with turgid face and stertorous breathing, which accompanies true epilepsy; as a rule, they do not bite the tongue nor froth at the mouth. They show all phases, varieties, and degrees of consciousness,—slight dazing which rapidly passes away; hallucinatory states, with or without recognition of surrounding persons and objects; somnambulic or hypnotic states in which they are capable of being impressed and controlled. Sometimes they remember what has occurred during an attack, sometimes they are oblivious of it; sometimes they can relate with great precision all its details, or, it may be, some of its phases; they can even, in some cases, give vivid accounts of their hallucinations and illusions.

Gowers has described some of the most interesting cases of the second class, in which the hysterical element predominates and the manifestations are irregular.

In one of the most severe cases of hysteroid attacks met with by him, in which the manifestations of hysteria were not only intense, but varied and prolonged, the patient was a girl aged ten. He describes the case at length.

The child belonged to a talented but highly nervous family. She had a sympathetic mother, who was convinced that her daughter was suffering from tumor of the brain. The illness had commenced after a severe mental shock (the death of her father). She had at first attacks of intense "shaking of the limbs," succeeded by violent delirium, and she screamed with pain in the head. After this there was persistent mental disturbance, "rambling in talk, associating surrounding objects with incidents she had been reading, and fancying every one would harm her. From time to time she had attacks of rapid flexion and extension of the legs, throwing about the arms, and catching at the bedclothes. On some days she would speak to no one."

Gowers proceeds with great detail to describe the mental and physical condition of this patient, and the various phases of her attacks, which continued for about a month. She complained of pain in the left parietal region, with also at this point intense superficial hyperæsthesia. In the intervals between the attacks sometimes she did not recognize her relatives, sometimes she would scream as if in agony, sometimes suddenly become rational. As the attacks ceased she complained that she could not see with the left eye. Vision in this eye was found to be one-twentieth, with concentric limitation of the field; color-vision was not tested; ophthalmoscopic appearances were normal. She was not hemianæsthetic. At a subsequent period she had curious recurring or alternating mental states, in some of which she was spiteful, passionate, and mischievous, and in others listless and apathetic. After a time her fits recurred, with a series of events well described by the physicians who had her in charge, which I will give, as it is descriptive of the plan and order in which these attacks sometimes proceed:

"(1) The eyelids were widely separated, the pupils being dilated and sensitive to light. (2) The eyes were gradually closed. (3) The head rotated from side to side on the pillow, the movement to the left being always more forcible. (4) The left forearm was alternately flexed and extended on the arm, the fingers of the hand being extended, and the left leg drawn up and kicked down, the toes being kept extended. Each of the above stages lasted only three or four seconds. (5) Sudden relaxation and immobility for ten or fifteen seconds, broken by a few moans during respiration. (6) She turned on the right side, and the fingers and toes became strongly extended back. This tonic spasm gradually passed on to the wrists and forearms, when suddenly violent clonic spasm occurred all over the body except the face and hands, which continued extended and stiff. There was no cry, no tongue-biting, no foaming at the mouth. After a few seconds there was sudden cessation of the spasms."

An association of hysterical phenomena with those of true epilepsy—a true epilepto-hysteria, the third class above given—is sometimes observed. By epilepsy the brain has become so deteriorated and degraded that the patient is liable not only to true epileptic attacks, but also at intervals to hysterical seizures, because of the functional disturbance of unstable, badly-nourished tissue. We can thus see how the hysterical seizures accompany, follow, or replace epileptic attacks of a grave character, and, with Trousseau and Gowers, believe that certain forms of convulsive seizure are in the strictest sense intermediate or indeterminate. In such cases the hysteria may disappear and the epilepsy continue, or the tendency to the hysterical attack may remain, and the brain become more deteriorated.

Hysteroid seizures occur in children suffering from post-hemiplegic epilepsy, but here the recognition of the two diseases is easy. "Hysteroid seizures," says Gowers, "are not at all rare in these patients. They may succeed epileptoid attacks, major or minor, or may occur alone. Some of the most severe hysteroid fits I have seen were in a girl the subject of infantile hemiplegia, whose arm was permanently paralyzed. The attacks came on without any indication of initial epileptoid seizure, and I have known them to continue, unless interfered with, for several hours,—violent opisthotonus, bounding movements, biting, tearing of the hair, etc. The same patient had also other hysterical manifestations,—attacks of violent laryngeal spasm, rapid breathing, and a phantom tumor. But she had also severe epileptic fits, beginning on the paralyzed side, one of which I saw."

As a fourth class I have mentioned cases in which at one time attacks distinctly hysterical occur, and at another those which are as clearly epileptic. The possibility of the coexistence of separate well-marked hysterical and epileptic convulsions in the same child should not be overlooked. In this class of cases the phenomena of the two diseases do not blend in the same attack, nor do the symptoms of one disorder immediately follow or alternate with those of the other. The patient has separate crises. The so-called hystero-epilepsy with separate crises in adults has long been known, and was first studied in France by Landouzy. Almost any form of hysterical attack may be exhibited at one time, and almost any form of epileptic at another, as petit mal and convulsive hysteria, grave epilepsy with minor or major non-convulsive hysteria. The practical point for the physician to remember is to inquire closely into the character not of one but of several attacks.

The following case, seen both by Dr. Weir Mitchell and myself, might be classed either as epilepto-hysteria or as the form of hystero-epilepsy with separate crises:

A child eleven years old, with neurotic and phthisical family history, had from her infancy been somewhat backward and peculiar, and had a defect of speech, which exhibited itself at times in a tendency to speak slowly, associated with some twitching and want of control over the muscles of articulation. She had been coddled and somewhat spoiled, and was addicted to masturbation, usually at night. She had scarlet fever, and after this began to have fits or seizures of peculiar character. These were frequent, several occurring sometimes during the course of the day. They commonly followed some injury, excitement, or slight peripheral irritation: thus, striking her toes, pinching her cheek, or pulling a hair would cause the attacks. When they came on, if walking or standing, she would bend forward and slip down to the ground, but soon after would come to her senses. During the attacks her face was pale. Two weeks before she was seen by me the attacks changed, so that she fell precipitately forward, and several times severely bruised her head. On questioning her closely, she said she could sometimes tell when an attack was coming on, and could sometimes control them and often did. She did not bring them on herself, and could hardly tell how they came. She said that she sometimes knew what was going on around her: she seemed to like to dwell on the excitement caused by the spells. She did not bite her tongue, or sleep after the attack, or scream at the beginning. The above description answers for the usual character of the attack. At times, however, she had seizures of a different kind, often of convulsive tremor and clearly hysterical. She was inclined to be untruthful and disobedient. She improved considerably in a few months under the careful attention of a good nurse.

Those phases of hysteria which are known as catalepsy, ecstasy, and trance are of moderately frequent occurrence in children. In the "American System of Practical Medicine" I have detailed a case of catalepsy or automatism in a child two years old, notes of which case were furnished me by Dr. De Schweinitz of Philadelphia,—so far as I know, the youngest patient of this kind on record. The child exhibited all the phenomena of hypnotic catalepsy and suggestion. Either by manipulation or command she could be placed in any position, in which she would remain until changed by others. Many experiments were tried with her successfully. Other cases of catalepsy at an early age have been reported, the youngest by Jacobi in a child three years old. Hemi-catalepsy has also been observed in children as well as in adults. Several of these cases have been reported by me.

A case with rotatory phenomena and other hysterical manifestations came recently to the Philadelphia Polyclinic service for Nervous Diseases. This patient was a school-boy, aged eleven years. At nine years he had begun to have "seizures" without apparent cause. At first these were rotatory in character; they would commence while he was standing, and he would go round and round for two or three minutes at a time. He appeared to be conscious during the attacks, of which he had fifteen or sixteen during the twenty-four hours. These lasted three months, and then ceased for nine months, when he had seizures of a different character. His legs and arms were drawn up spasmodically, and while rigid he was affected with a general tremor. At the onset he would scream for some time,—not a sudden epileptic scream, but a frightened cry. His face was suffused, and he appeared to be unconscious, but he had never fallen nor bitten his tongue.

Springthorpe[1] reports a case of trance, ecstasy, and hystero-epilepsy in a child ten years old. The patient was a sensitive, emotional girl, with paralytic, epileptic, alcoholic, and tubercular ancestry. When twenty months old she had an attack of sunstroke followed by a convulsion, and at seven years of age she had a severe attack of whooping-cough. One night in bed she was heard breathing heavily, and for an hour could not be aroused, but the next morning was all right. Other similar seizures occurred at irregular intervals, and after more than a year the character of the attacks changed. She began to show signs of mild religious ecstasy, which was to some extent attributable to an extensive acquaintance with the Salvation Army. She had seizures with heavy breathing, quivering lip, tearful face, crying, sobbing, and sighing. She said, "Oh, dear!" "How pretty!" etc. She saw "angels." After a time she held conversations with her heavenly visitors or appearances. Special and general sensation were in abeyance, and hysteroid convulsions in the muscles of the neck were sometimes present. Later the ministrations of the Salvation Army were changed for those of a clairvoyant or mesmerist. She began to talk gibberish, and after a time went off in severe hysteroid convulsions; opisthotonus was extreme, and the convulsions were general. Her attacks, in short, in their different phases and varieties were hystero-epileptic.

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[1] Australasian Medical Gazette, January, 1885, p. 106.

Minor convulsive attacks are not uncommon in hysterical children, and these exhibit, but in very imperfect form, the phenomena of hystero-epilepsy. The attacks seem to be partly purposive and partly unconscious. Ashby and Wright describe a case of this kind in a girl seven years old. When the attack came on, she would jump up in bed, turn round once or twice, sit down again, and arrange the bedclothes, smoothing them carefully down, and yet be unconscious during the fit, and have no remembrance of it afterwards. A sharp word or a prick of a pin would often arrest these fits.

Local spasm of almost any form or seat may occur in hysterical children. Twitchings of the facial and ocular muscles are perhaps the commonest types.

Henoch describes cases of local spasm which took various forms, as violent fits of hiccough, voice-spasm, chorea, asthmatic attacks, spasms of the muscles, face, eyes, and extremities. What he calls voice-spasm or vocal spasm has been described by other authors under such names as hysterical cough, laryngeal spasm, etc. In one case extreme hyperæsthesia of the front of the chest was replaced by violent spasmodic coughing resembling whooping-cough; in another the child after every expiration uttered a half-whimpering or half-squeaking sound; in still another a cooing or croaking sound accompanied the spasm.

Jacobi gives several instances of hysterical cough in young children: one was a boy six years old; two others were brothers, one six and the other four. Local treatment did not relieve. One of the children was addicted to excessive masturbation, and after suppressing this habit the child recovered.

Peculiar rhythmical movements are sometimes present in hysteroid or hystero-epileptic cases. Gowers says of one of these cases, "This peculiar rhythmical movement in the legs was a feature of the hysteroid attacks in one patient whose case, although published elsewhere, presents many instructive points deserving narration at length. Both epileptic and hysteroid attacks occurred; organic heart-disease was present; the disease succeeded an attack of chorea, and the patient presented a strong inherited tendency to neurotic disease. It is probable that the hysteroid convulsion really succeeded a slight epileptoid seizure."

The recognition of lyssophobia, or hysterical hydrophobia, in children, may be of the utmost importance. A child who has been bitten by a dog is seized with convulsions, which may come on spontaneously, or accidentally as the result of other disease, or because of conversation with regard to hydrophobia, mingled with expressions of fear because of the biting. Immediately a diagnosis of hydrophobia is made by the horror-stricken relatives and friends, and this is adopted by the thoughtless or badly-trained physician. Through suggestion many of the phenomena of hysterical attacks are produced in children, just as a train of symptoms can be started by hypnotic suggestion. In the pseudo-hydrophobic cases barking, biting, etc., may be thus produced. Many of these cases are purely hysterical. Not infrequently we see in children as well as in adults hysterical seizures unconnected with the bite of an animal, in which many of the so-called hydrophobic symptoms are present. Gowers relates an interesting case of a boy of thirteen, in whom hysterical attacks commenced after he had been bitten and frightened by a dog.

Any variety of paralysis may occur in hysterical children, as hemiplegia, monoplegia, paraplegia, local palsies in the face, trunk, or limbs, paralysis of the bladder, and vocal paralysis or aphonia.

Gillette,[1] at a meeting of the New York Obstetrical Society, reported a case, already referred to, which, on account of the extreme youth of the patient, it might be well to detail. A girl only eighteen months old had a habit of walking and plunging and falling on the floor for amusement, and one evening when she was about to do so her mother caught her by the arm and stopped her. The arm fell helpless to her side; the child moaned and desired to be held and caressed. No signs of injury could be found. The next morning the arm was still hanging helplessly by her side, and she was moaning and crying as if in considerable pain. The physician remembered that the child had always been very sympathetic, wonderfully so for her age, even weeping when sad pictures or doleful stories were presented to her, and therefore believed the case to be one of hysteria. In the evening the arm was still apparently paralyzed. The next morning the doctor was sent for in haste, and the other arm also hung helplessly by her side. The parents and friends were greatly alarmed. The father was requested to get a ball of variegated colors and offer to play with the child, which he did after all had left the room but Dr. Gillette, the patient, and the parents. The experiment was instantly successful: the child played ball at once as if nothing was wrong with her arms. She tried the hysterical attack the next morning, but it did not work, and she did not attempt it again.

Some of the forms of paralysis in children set down as hysterical are not certainly of this character, or have in them only a strong hysterical element. A form of paralysis mentioned by Jacobi, for example, resembles spinal infantile paralysis, and appears to result from a change in the circulation in the spinal cord, bearing a great similarity to what is known to take place after infectious diseases. He gives the case of a girl who first began complaining of stomach-ache and later was troubled with nausea. She was taken with typhoid fever, and when convalescing the nausea returned. Shortly after this she had general convulsions, being constantly nauseated and very weak. When seen by Jacobi her weakness was almost complete paraplegia. She had varying conditions of hyperæsthesia and anæsthesia, sometimes with coldness and pallor, sometimes with circumscribed red spots; frequently perspiration alternated with dry coolness. Of diagnostic importance, according to Jacobi, was the small number of tangible changes in proportion to the large number of symptoms. He attached particular importance to the absence of temperature-elevations, making many observations. The case was regarded as one of vaso-motor neurosis. Ergot was given, and the galvanic current to the spine was used. Later the patient was taken to Europe and completely recovered.

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[1] New York Medical Journal and Obstetrical Review, 1882, vol. xxxvi. p. 66.

The same writer gives other illustrations of hysterical paralytic and paretic conditions in the young, although he believes they are not frequent. A girl of nine years, for instance, fell in convulsions, which were followed by ptosis and deviation of the right eye outward, the pupil somewhat dilated, on the left the pupillary branch being also a little affected. The tongue deviated to the right. The child improved, but after another attack of convulsions had the same paralytic symptoms. Jacobi cured her by firmly impressing her with the idea of success and then pressing his thumb firmly on the supraorbital nerve and commanding her to open her eye. Her ptosis was instantly relieved, and she was well for several weeks, when she exhibited the same symptoms and was relieved in the same manner. It might be questioned whether this was not an exhaustion paralysis after an epileptic cerebral discharge, rather than an hysterical palsy.

Lee[1] has reported a case of aphonia, in a girl four years old, seen at the Pennsylvania Hospital under Dr. Addinell Hewson. It developed suddenly. She was supposed to be hysterical, and was watched carefully while at play among her companions to see if she did not speak at unguarded moments. She was examined by several physicians, some of them laryngologists. As a final test, she was placed under ether, and as she came out of its influence cried lustily, and talked as patients usually do before recovering fully from the effects of the anæsthetic. Her surroundings were changed, and she was cured. Goodhart attended a girl of twelve with functional paralysis of the abductors of the vocal cords. She had also a croupy cough; there was entire absence of any morbid appearance of the larynx, except in the position of the vocal cords. These played somewhat close together during expiration, and during inspiration the anterior parts completely closed, the left overlapping the right, and leaving only a chink behind for the entrance of air into the lungs. In the course of half an hour the paresis had almost disappeared. This patient had been in the hospital under Dr. Taylor for cataleptic attacks, and in one of her fits her eyes were first turned strongly to one side, and then she squinted.

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[1] New York Medical Journal and Obstetrical Review, 1882, vol. xxxvi. p. 67.

Sensory hysterical phenomena are probably more common in children than any other manifestations, a fact which does not seem to be generally appreciated by the profession at large, and, in consequence, cases clearly hysterical in character are frequently supposed to be organic and of serious import.

The most common forms of hysterical sensory disorder may be classified as hyperæsthesia, anæsthesia, pains referred to special parts or organs, and pain irregularly distributed over the body.

The meaning of hyperæsthesia in children should always be fully considered. The tendency of the physician, and more strongly of the parents, is usually towards a grave diagnosis; and many cases simply hysterical have been set down, because of hyperæsthesia and the complaint of deep-seated pain, as neuritis, meningitis, myelitis, brain-tumor, reflex neuralgia, etc. Every one who has been much with young children knows how impressionable, imitative, sympathy-loving, and observant of suffering they are. Pain appeals especially to the imaginative faculties of sensitively-organized young children. According to Ashby and Wright, probably the most common form of hysteria in girls is hyperæsthesia, a complaint of tenderness or pain which cannot be accounted for except by a neurosis,—local tenderness about the spine or one of the joints, especially the hip; hypersensitiveness of the thyroid gland or front of the larynx; headaches, either frontal or occipital, or of the form of "clavus." "Hysteria," say the same authors, "is apt to mimic various diseases which are normally accompanied by severe pain, such as peritonitis, pleurisy, rheumatism; it must, however, be borne in mind that there may be some actual disease present, and the sensory disturbance is only an exaggerated condition of what would normally exist."

Hysterical loss or diminution of sensation—either tactile anæsthesia, analgesia, or loss of temperature-sense—occurs in children with considerable frequency, usually in connection with the other phenomena of hystero-epilepsy, but sometimes as an isolated symptom. Peugniez has recorded a series of cases of anæsthesia of the skin and special senses in children from ten to fifteen years old.

Barlow also has collected a series of cases in children between ten and fifteen years old. A boy two years old, when the skin between his fingers was pricked with a needle, did not wince or withdraw his hand, and when one side of his face was pricked showed the slightest possible play of the features, while upon pricking the other side he cried. In another case strong faradism was tolerated with perfect indifference when applied to either leg; in another faradism was felt, although a needle had been pushed into the skin without the child withdrawing the limb; in a fourth the needle was tolerated for several minutes, and then the limb was slowly withdrawn, but without a cry. In still another analgesic paralysis without wasting, which had lasted nine months, was also present. A girl five years old had her arm scorched over a tract about three inches long without crying; she also had her thigh deeply cut by a bit of broken pottery without showing any distress. A girl of eleven had complete analgesia on the right side of the body, and also on the left cheek. She could not localize the spot where she was pinched on the right side, but she had no difficulty on the left. This child's field of vision was considerably limited, and she had what the mother termed "blind fits." In a girl nine and a half years old both arms and legs were analgesic. A boy nine years old, subject to fits, was found to be tolerant of the application of a needle down one side of his body and almost so on the other side, but he could localize the position of the needle perfectly, although it did not give him any trouble. A girl nine years old had paralysis of both legs, without any affection of the sphincters or wasting or rigidity of the limbs, and was absolutely tolerant of every kind of stimulus applied to the limbs. A girl of eight years, with a history of convulsive and other hysterical manifestations, was found to be distinctly analgesic in her arms after a violent attack. A girl three years old, with a history of convulsive and screaming fits, was analgesic all over for two weeks after an attack.

At a meeting of the Clinical Society of London, October, 1877, Thompson reported a case of hysteria in a boy, with anæsthesia, ischæmia, and contractures of the lower limbs. It is perhaps significant that while this boy had an English mother his father was French. The boy had always been delicate, but in the spring of 1877 he began to show hysterical phenomena. He became unable to walk, owing to paresis, rigidity, and distortion of the lower limbs, and his voice was almost inaudible. The legs were firmly flexed upon the thighs, and the feet extended as in talipes equinus. The genital organs were ill developed; anæsthesia and ischæmia were prominent symptoms from first to last. Under the application of gold coins the anæsthesia and ischæmia disappeared. Other metals were tried, but the effect could be produced only by the gold.

Goodhart has twice seen hemianæsthesia with hemiplegia in boys of eleven and twelve. In a case under his own care he was at first disposed to believe in the existence of some actual lesion. This boy was twelve years old, with a neurotic family history. Paralysis came on in the night. "His face was paralyzed on the right side, and the tongue deviated to the right. The right arm was paralyzed, the extensors of the forearm most markedly so, and the wrist dropped as in lead-poisoning. He made evident efforts to move it when told, but was obliged to call in the aid of the opposite hand. There was less decided failure in the leg, but, when he walked, his toes caught the ground in putting the foot forward, the knee was flexed, the heel drawn up, and the limb moved clumsily as from want of harmony between the coacting muscles rather than from actual paralysis, but the extensors obviously had the worst of it. The loss of sensation was complete, and thoroughly distributed to the right half of the body, mucous membrane as well as skin. The knee-reflex on the paralyzed side was markedly exaggerated. . . . The paralysis both of sensation and motion—but the former far more than the latter—varied much from day to day; and sometimes the special senses suffered."

Headaches which may be properly classed as hysterical occur sometimes, although rarely, in children; they are much less frequent than in the young or middle-aged adult. In children in the majority of cases they are imitative, at least in part. The child of a mother who suffers from migraine or any of the forms of recurring headache will, if precocious or imitative, sometimes really or apparently suffer from severe head-pains. Such cases are very deceptive. The fact that true migraine occurs in very young children must not be overlooked in making a diagnosis. Sinkler has written an interesting paper on the subject, giving a number of cases in children under puberty. In one case under my own observation, a child between two and three years of age began to have mild attacks of migraine, both the mother and the grandfather of this child having suffered with typical migraine for many years.

West mentions a boy of thirteen who had been ill with headache and vague cerebral symptoms. His sister had died with disease of the brain, and he had shown extravagant grief at her death. His worst symptoms were headache, intense sensitiveness to sounds, and dislike of the light. Sometimes he would not allow his head to be brushed or even touched. He was feeble and ill nourished, and often walked with a limping gait. In spite of the long continuance of his symptoms, he did not grow worse. When not observed he walked and spoke better, and forgot his headache and other ailments if interested in anything. West also mentions the case of a girl ten years old, who had a spasmodic cough when younger, and at eight years of age began to suffer with excruciating headaches. These were arbitrary in their onset, and, except a capricious appetite and a constipated habit, the child had no other bad symptoms. Sometimes she would lie for hours with her hands to her head, moaning and crying, and would cling to her mother with vehement protestations of affection. If the doctor went into the room unexpectedly, even if she were cheerfully at play, she would immediately put her hands to her head and commence to moan. She afterwards developed a train of grave hysterical symptoms, making the diagnosis clear. Eventually she recovered.

Dessau reports a case of hysteria in a boy thirteen years of age, in which the affection began with a pain in the right ear. Soon after the pain in the ear ceased he complained of pain in the right hypochondriac region, which gradually extended over the left side, and about the same time he had a dry, empty, barking cough, with muco-purulent sputa. In a few weeks pain was complained of in the left ankle-joint, later in the knee-joint. These pains were accompanied by firm contractures, but he had no swelling about the joints. His general condition was bad. He recovered under the use of cod-liver oil, the hypophosphites, and syrup of the iodide of iron. This case presents the features which should help the physician to discover that he is dealing with a case of hysterical pain, although on first being called the diagnosis would be difficult. The pain was severe, and no organic condition could be discovered in any of the locations to account for its presence; it passed from one region to others remote and disconnected; it attacked tissues and organs so different in character as to exclude such causes as rheumatism and gout, which affect particular structures.

Bemiss[1] reports an instance of hysteria in a boy six years old, who two weeks before coming under the care of the physician had begun to complain of pain in his abdomen and around his waist. He was treated for worms, but without result. He was pale, reduced in flesh, and peevish; his tongue was pale and slightly coated; his temperature was 99.5° F. The pain which had been at first in the abdomen had gradually descended to the thighs, knees and feet; it was rather an excessive hyperæsthesia than a true pain; swelling and local heat were absent. He was treated with quinine and bromide of potassium, and in three days he was able to walk in a limping manner; but two days later he was found on his back with both feet lifted from the mattress and the thighs lifted from the abdomen; he would scream if any one even offered to touch his feet. These symptoms had come suddenly. He got better and worse under treatment. In a few days he complained not only of his feet, but also of his left hand, which he insisted upon having wrapped up, especially the middle finger.

Henoch relates the case of a boy, six and a half years old, previously healthy, who had had measles, and two weeks after the beginning of the disease another boy fell upon his abdomen. He began to complain of great pain, crying and tossing about continually. The pain came in paroxysms of increasing frequency and intensity; temperature about 101° F.; pulse frequent, tongue coated, with fetor of the breath, and thirst. The central symptom was intense hyperæsthesia of the skin of the abdomen and chest, so that severe pains were produced by picking up a fold of the skin. He made a rapid recovery.

Cases similar to those of Henoch and Bemiss are not infrequent. I have several times been called in to decide upon their character. The question of the existence of a neuritis, dermal or truncal, is one to be considered. Possibly a mild form of a neuritis or other organic disorder is present sometimes, the pain and suffering proper to the disease being exaggerated by the cerebral condition of the hysterical patient. Starr,[2] in the Middleton Goldsmith Lectures, says that pains sometimes set down as hysterical are due to a neuritis of greater or less severity. While such cases have usually recovered, they have sometimes been tedious, lasting at least for weeks.

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[1] New Orleans Medical and Surgical Journal, October, 1886, p. 255.

[2] Medical News, 1887, vol. i. pp. 141, 189, 197, 225.

A case seen with Dr. Feldstein, of Philadelphia, illustrates this difficulty of diagnosis between hysterical hyperæsthesia and multiple neuritis. This patient, a girl eleven years old, began to complain in the spring of pain in the head and also at the nape of the neck, the latter associated with a feeling of stiffness. She soon also made complaint of pains in the spine and of both the upper and lower extremities, and also of universal tenderness to pressure. The pain and hyperæsthesia did not follow the line of the nerve-trunks, but were general and dermal; she spoke of them as being present all the time; but at intervals she had exacerbations of pain in circumscribed, isolated patches on the extremities, these areas becoming red and remaining so for a short time. Her disposition changed: she became irritable, fretful, and excitable. For three weeks she showed some irregular but not marked elevation of temperature. Various methods of treatment were adopted, including salicylates, iodides, small doses of mercury, tonics, and anodynes; but nothing in the shape of medicine seemed to benefit her much. She was three months in bed. At the end of two months she showed some enlargement of the thyroid gland. At first she lost flesh, but at the end of three months was as fleshy as before her sickness.

What are known as hysterogenic or epilepto-hysterogenic zones are sometimes present in hysterical children. These are sensitive spots or areas of the skin, and on touching, pressing, pinching, or otherwise exciting them active hysterical or epileptoid manifestations, in some cases even convulsions, occur. In one case, in a child, pressure between the scapulæ would produce either hysterical convulsions or a state of excitement like a mild mania. In another case, pressure or pinching of almost any portion of the body would cause spasms, although usually these hysterogenic zones are in particular regions.

Hysterical affections of the eye are seen among children, and relatively with considerable frequency, and these may be of any of the forms described as occurring in the hysteria of adolescence and adult life,—ptosis, strabismus, pupillary alterations, amblyopia and achromatopsia of either one or both eyes, and total blindness. A fair number of cases of hysterical amblyopia in children have been reported by observers in various countries, the reporters giving different opinions or expressing doubts as to the proper interpretation of the phenomena. The chief point in doubt has usually been whether or not the loss or diminution of sight has been the result of pure fraud, of unconscious deception, or of some temporary impairment of the functioning in the cerebral visual centres, either primary or cortical. This problem is by no means so easy of solution as might at first appear. A child asserting loss of vision in one eye is surprised or tricked into the use of the other by placing a strong convex or concave lens, or a prism, before the unaffected organ, and the inference is made that deception has been practised by the patient or culprit; that, in other words, the individual can see with the amblyopic or blind eye, but will not, owing to mental perversion or obliquity. Such a conclusion, however, is not necessarily correct. It is possible that the visual centres of one side of the brain may in such cases be in abeyance as long as the other side is free to act, the former, however, being stimulated into functional activity by incapacitating the latter. Probably pure fraud is the best explanation for some, but certainly it is not for all.

Dr. De Schweinitz,[1] in reporting a case of hysterical amblyopia in a colored girl nine years of age, who denied even light-perception in one eye, speaks of an interesting experiment bearing on this point. "In one case of simulated blindness (malingering) the subject was made to stand the test by having it explained to her that those present at the examination fully understood that her statement in regard to sight in the eye under examination was totally false. She was sufficiently intelligent to appreciate that she had been detected in her attempt at deceit, and readily admitted the truth of the charge. On the other hand, a perfectly intelligent woman subject to hysterical amblyopia, in whom the test demonstrated the fact that the eye claimed for blindness had full visual acuity, utterly denied the possibility of sight, in spite of the fact of her apparent perfect appreciation of the sufficiency of the tests which demonstrated that she could see." Both hysterical amblyopia and achromatopsia in children, as in adults, have usually been found in patients in whom the other phenomena of grave hysteria are present, as, for example, hemianæsthesia, hemiparesis, contractures, convulsions, and psychical episodes, usually of the maniacal or hallucinatory character.

Years ago I reported a case of simulated monocular blindness occurring in the service of Dr. Harlan in the Wills Ophthalmic Hospital, Philadelphia. Harlan has reported other similar cases, and has contributed several articles on this subject.

Achromatopsia is a "condition in which there is a failure to appreciate colors. In Daltonism, or true color-blindness, one color may be taken for another ; in achromatopsia the notion of color may be entirely lost. These colors are found by the patient to disappear in regular order, and return in reverse order as the patient recovers. Some remarkable cases of this kind have been reported as occurring among French hysterics. A few examples of the same affection have been reported in America. Sometimes the patient has lost perception of one or several colors. When only one color is lost, it is usually violet ; if two, violet and green ; then in regular succession follow the colors of the spectrum."[2]

Of one form of hysterical blindness in children I have seen two examples,—that in which the patient complains of sudden blindness in one or both eyes, this lasting only for a very brief time, as for a few minutes.

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[1] Journal of Nervous and Mental Diseases, April, 1890, p. 275.

[2] American System of Practical Medicine, vol. v. p. 247.

Mendel[1] has reported a case of aggravated hysteria in a boy ten years old, with amaurosis of first one eye and later of the other. This patient's maternal grandmother was epileptic. He suffered painful sensations in the course of the trigeminus and other nerves, and after a time appeared irregular action of the muscles, unsteadiness of the intellect, incontinence of urine during the day, and within a year and a half pains in the head, vomiting, amblyopia in the right eye, and emaciation. These symptoms vanished, with the exception of irritability and peevishness, and were followed by drumming of the feet, dizzy sensations, emprosthotonus, with rapid forward movements of the body, without loss of consciousness, these symptoms appearing in paroxysms for twelve days. On the thirteenth, in place of the tonic spasm appeared spasmodic shaking, spasms of laughter, hallucinations, and loss of consciousness, amaurosis of the right eye, and paresis of the right arm. Six days later the blindness and paralysis had disappeared, and a general hyperæsthesia was developed; then amaurosis of the left eye, lasting twenty-four hours, was added. The boy was removed to a public institution, and in three days was well, nothing remaining of his old trouble save a decided excitability.

Among the views that have been suggested to explain these hysterical defects of vision is that which has also been resorted to for the explanation of many other hysterical phenomena,—namely, hypnotic suggestion, in some cases auto-suggestion, perhaps the result of the observation or discussion of blindness in others. The patient believes that he cannot see, and therefore does not see. But this explanation, seemingly lucid enough, is, after all, a begging of the question. The brain, or a portion of it, must for a time be in a changed condition in order that the self-deception may be accomplished.

Hysterical deafness or diminution of hearing has been recorded as occurring either as an independent symptom or in association with grave hysterical phenomena: I do not recall a single case in my own experience in which this has been present as an isolated symptom in a child under thirteen years of age. Loss of smell on the same side as deafness and blindness has been recorded.

Among the most important hysterical affections occurring in children and among those in which pain is a leading element are those simulating organic affections of the joints. To Shaffer we are indebted for the most valuable contribution on this subject, one which, although by no means neglected, has not attracted all the attention which it deserves. A large percentage of his cases were children under the age of puberty, and therefore strictly within the limitations of this paper. His study, while designated "The Hysterical Element in Orthopedic Surgery," affords in the cases which he admirably details a study of almost all the phenomena presented by hysteria in children,—hyperæsthesia and subjective pain, rigidity and contractures, spasm and paralysis, malingering and neuro-mimesis, lying and limping, precocity and perversity. The observations to which he refers of Brodie, Skey, Paget, Esmarch, and others are familiar to the profession. The false often so closely resembles the real disease that, as Shaffer shows, even the most experienced are at times at a loss to decide whether a given articulation is in a progressive state of chronic disease or simply in a neuro-mimetic state. He gives many instances of neuro-mimesis both of the limb-articulations and of the spine.

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[1] Deutsche Med. Wochenschr., No. 16, 1884.

Jacobi has also given a number of interesting illustrations of neuroses of the joints in children from five to twelve years of age. The majority of his cases had been observed about the knee-joint, but the ankle-joint and hip-joint were by no means free. The internal condyle of the femur, the styloid process of the ulna, and the vertebral column were the favorite places. The same affection, however, had been observed in the sciatic, obturator, crural, peroneal, saphenous, and tibial nerves, in the cervical, lumbar, hypogastric, and sacral plexuses, and in the peripheric nerve branches distributed about the integuments, joints, periosteum, and bones. A girl eleven years of age applied for surgical treatment for coxalgia. The pain was intense, and the symptoms closely simulated those of organic hip-disease; but the child was not emaciated, and no fever could be detected. Under chloroform the spasmodic contracture relaxed, no crepitation was felt, and mobility was complete. The patient recovered under general roborant treatment with an occasional sedative, and compulsory exercise. Similar treatment succeeded in a girl nine years old, who suffered from intense pain in the knee-joints, but more frequently in the ankle-joints and in the tarsus and metatarsus. In another case the difficulty of diagnosis between osteitis and hysteria was very great, and both were probably present. A girl of eight years, under treatment for ulnar neuralgia, developed a moderate swelling at the wrist and shoulder-joint, which disappeared to be replaced by severe pain in the toes. Her sufferings were mitigated when œdema of the right foot made its appearance. Another girl, of five years, was treated for acute rheumatism of the shoulder-joint. A boy of eight years, with a slight mitral incompetency, had rheumatism of both wrists and both ankle-joints, with fever. After his fever had disappeared he still complained of excessive pain, yelling and screaming at times.

Varieties of hysterical club-foot have been described by Shaffer, Laycock, Shaw, Little, Skey, Mitchell, Sir Charles Bell, Charcot, Adams, and others. Talipes varus seems to be one of the most common forms, if not the most frequent one, in young girls. In extreme cases of this kind the outer margin of the foot alone touches the ground, the sole presenting vertically backward, as in a case described by Little. The anterior and posterior tibial and gastrocnemius muscles may all be tensely contracted. In inversion of the foot in children the probability of the hysterical nature of the affection must always be borne in mind. Such cases are usually unilateral; they are always acquired, often suddenly, and the ordinary indications of disease and disorganization, such as pain, heat, and swelling, are absent. Shaffer records four personal cases. One of these was in a precocious and emotional girl of eleven years; another was in a boy of ten, this boy illustrating many phases which hysteria may assume in boys of tender years,—paralysis, spinal pain and tenderness, anæsthesia, contracture, hyperæsthesia, talipes, etc. Among other cases of hysterical contracture which have been recorded in children as well as in adults are those in which the spasm affected the thumb and the masseters.

Weak backs, and backs which may be bent and twisted into various positions which often closely counterfeit organic curvatures, are observed in hysterical children, and have been considered by Laycock, Noble Smith,[1] Shaffer, and others. Laycock speaks of lateral curvature as due to hysterical paralysis, holding to the opinion of Stromeyer, that the serratus magnus is involved in the production of the curvature. Noble Smith [1] records a case of curvature which, although in an adolescent patient, illustrates the peculiarities of hysterical curvature in the young. She had "suffered with her spine" for many years, and had been treated with plaster-of-Paris jackets for two and a half years. She carried her head and shoulders considerably out of the perpendicular and bent to the right side. In sitting, the right axilla was two inches or more outside a vertical line from the outer edge of the pelvis. She had been cured, it was stated, a few months before by "faith," but had soon relapsed. By moving her body into various positions he found that there was a fixed curve, moderate in degree, in the lumbar region to the left, but the bending of the body over to the right was a voluntary position, or governed by her hysterical condition. He insisted that if ever the patient came to see him holding herself in that position he would decline to deal further with the case. After this she held herself perfectly straight, with the exception of a moderate degree of fixed curvature which existed in the lumbar region, to the cure of which subsequent treatment was directed.

"In young children," says Smith, "great weakness of the back may be present, which, in the various positions of sitting or standing, allows the spine to bend in various directions, forming at one time posterior curvature and at another one or more lateral curves. Although this condition would probably lead eventually to the formation of lateral curvature, yet in diagnosis a distinction is to be drawn; for when this state of weakness exists, support to the back and rest are more important at first than exercise. In fact, in severe cases the strength of the child is more rapidly and safely restored by absolute rest at first than by attempt to exercise the muscles. These curves are readily movable in any direction, the spine being easily straightened or bent."

Hysterical children are subject to various forms of night-attacks, so that the term "nocturnal hysteria" is almost justified as descriptive of certain common affections. Under the name of night-terrors varieties of these attacks have been frequently described, and these are very properly regarded as, at least in many cases, forms of hysteria. Ollivier speaks of them as "une forme larvée de l'hystérie." They may, however, occur in children who are epileptic, or who are suffering from some exhausting disease, but even then may be regarded as hysterical manifestations. They are most frequently observed in neurotic children, before and during the time of second dentition. Almost any child, however healthy or fortunate in its heredity, may under special causes, as indigestion, fright, or acute pain, become the temporary victim of such seizures. Handfield Jones records a case in which the cause was probably inflammatory pain in the ear. A weak, excitable nervous system affords the predisposition, while many causes may directly excite the attacks, among which may be mentioned pain, hunger, indigestion, injudicious feeding, worms, masturbation, difficult dentition, insufficient or too much bed-clothing, over-study, sensational reading in the evening, and ghost or other terrifying stories. These attacks vary considerably, but have some tolerably constant features. The child may have gone to sleep well, but during the night, and usually within an hour or two after retiring, suddenly awakes with every sign of fright and alarm, often screaming and gesticulating with terrified expression, as if it saw and feared some frightful object. Consciousness is usually perverted, —as in one of the various hypnotic or hysterical conditions,—the child seeming to recognize imperfectly those around it, although sometimes it will for a time be apparently unconscious of everything but the source of its terror. In some cases, doubtless, the little patient is the victim of illusions and hallucinations, transforming objects within sight into terrifying appearances, or having hallucinations of objects, probably usually animals.

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[1] Curvatures of the Spine, 3d ed., London, 1889.

J. K., aged seven, a school-boy, began to have nervous spells in his sleep. He jumps up suddenly from a sound sleep and walks round the house, crying and trying to say something, but cannot make himself understood. His mother says that at these times he shakes all over, with his hands close shut, and, she thinks, with his teeth closed also. He never falls down, but is wakeful afterwards, and tells what he has been dreaming about. He got out of bed one night, screaming and crying, and was found trying to creep over the bureau. His father and mother took hold of him, but could hardly restrain him: his face had a scared expression.

A boy, seven years old, had always a tendency to start at nights, and began to have spells about the same hour, in which he would suddenly stand straight up in bed, scream, and call for his mother, who would find him in a dazed and frightened condition. He had these attacks as often as once in a week or ten days, and if annoyed or crossed during the day was most likely to have them. He was retiring and solitary in his habits, not caring to engage in play with other boys. About a year before coming under observation he had an attack which lasted about a month, which seemed to be malarial in character, as he had periodical attacks of chill, fever, and sweat.

After this he began to complain of dizziness and twitching in the eyes, and also of partial loss of vision. Under tonics and systematized exercise he recovered.

Nocturnal incontinence of urine is frequent among hysterical children, although it should be regarded only in special cases as an hysterical symptom. According to Trousseau, the first cause of incontinence is a neuropathic predisposition. In those cases in which periodicity is a decided element, the nervous character of the disorder is most probable. That incontinence may be the accompaniment of severe nocturnal epilepsy should always be borne in mind, and this probability should be excluded. The manner in which hysteria and epilepsy sometimes merge into each other is apparent in some of these cases. The children suffer from nocturnal, periodical neuroses. Of course in coming to a decision for therapeutic purposes with reference to nocturnal incontinence, all special exciting causes, digestive, renal, sexual, genital, and others, should be taken into consideration. Even when such reflex causes of excitement are present, the neuropathic tendency may have something to do with the continuance of the disorder. As bearing upon the question of the hysterical or, at least, neurotic character of night incontinence in children, may be mentioned the fact that Liébault has in a number of cases cured children of this bad habit by hypnotic suggestion.

Somnambulism in its ordinary form might also be classed with nocturnal hysteria. It certainly occurs almost invariably among the hysterical and epileptic, at least when it is a persisting affection. Night-terrors are indeed regarded by some as a special form of somnambulism. "The victim of night-terrors," says Lyman,[1] "not only moves his body, but gives vocal evidence of his feelings of apprehension and alarm. In like manner, projecting his dream into action, a sleep-walker may arise from his bed; he climbs out of the window and descends to the ground, executing all manner of complicated and dangerous movements; he walks long distances, and finally returns to his couch without waking. In the morning no recollection of the events of the night survives. Again, the movement may be less locomotive in character. The intellectual faculties chiefly may be aroused, and then only such movements are executed as may be necessary to give expression to mental powers." This hysterical somnambulism or noctambulism has a striking tendency to recur at about the same time of the night in many cases. This is usually a comparatively early period of the night, soon after the earliest deep sleep. A number of cases of this kind have come under my observation. A child six years old, whose aunt had had chorea in childhood, and whose father walked in his sleep, every night for two weeks went to bed at seven, and got out of bed and went downstairs in a dazed state at 8.20; her attack usually lasted about fifteen minutes. She was subject to attacks of hysterical crying. A girl, four years old, who had chorea and stammered, had attacks of hallucination and sleep-walking at night, usually occurring within two hours after retiring.

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[1] American System of Practical Medicine, vol. v. p. 371.

Occasionally, although more rarely than in adults, children suffer from nocturnal attacks of paresis, numbness, hyperæsthesia, hemeralopia or night-blindness, delirium, or fever. These symptoms sometimes show themselves just at the time of waking, particularly sensory phenomena, such as painful numbness. Féré,[1] in an interesting contribution to the "Pathology of Night," has considered many of these forms of nocturnal functional disease, and shows that they are not infrequently connected with the hysterical diathesis. Darkness exerts not only a purely psychic influence, but also a distinctly physical influence, on the vital functions. The various nervous phenomena peculiar to night are well described by Féré as the result of a deficiency of physiological excitation, being in fact paralyses caused by non-irritation. So far as reported experiences go, these nervous nocturnal affections are more likely to occur in adults than in children; but the possibility of their occurring in the latter should not be overlooked.

It is now generally admitted that, even excluding cases of simulation or fraud, a fever which may be truly termed hysterical must be recognized, although there are few records of such cases in young children. Hysterical fever was probably a correct diagnosis in two personal cases. One of these was a boy of ten, with hysteriform choreic movements, accompanied by pain in the head and limbs, and by irregular but widely-distributed hyperæsthesia. The diagnosis of meningitis had been made. The temperature ranged for several days between 100° and 104° F. The patient recovered under a practically negative treatment. The other was a girl of nine, with symptoms which closely simulated those of an aberrant form of typhoid fever, but in which some of the most positive signs of the disorder, such as diarrhœa, the eruption, etc., were absent. From the time of Briquet hysterical fever has been discussed, and during the last few years it has received considerable attention. Dr. Mary Putnam Jacobi,[2] in an excellent article, records a case and reviews at some length the literature of the subject to date, citing Pinard, Henri Fabre, Bressler, Hale White, Donkin, Creig Smith, Meade, Debove, Barié, and various physiological investigators, as Wood, Tscheschin, Charles Richet, Schreiber, Aaronson and Sachs, Ott, and Girard. She omits to mention Briand, Gubler, Rigel, and Dieulafoy, referred to in the writer's papers on hysteria. The clinical observers in this list record cases occurring after puberty, but *a priori* hysterical fever is perhaps more likely to be present in early life because of the greater vulnerability of the thermic centres. In cases of high temperature in young children we should always bear in mind the possibility of hysteria, but we should also remember that in many affections not hysterical a high temperature is observed in children of neurotic temperament.

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[1] Brain, October, 1889, vol. xii. p. 308.

[2] Journal of Nervous and Mental Diseases, June, 1890, vol. xvii. No. 6, p. 373.

DIAGNOSIS.

While the diagnosis of hysteria in children is perhaps not much more difficult than in adults, the disease is more likely to be overlooked. Many physicians do not anticipate its occurrence in children, and, not expecting it, they attribute hysterical affections to more serious causes. They forget, what has been demonstrated by the facts reported in this paper, that hysterical manifestations are possible as soon as the child has developed the capacity to receive impressions and form concepts. Hysteria in children must therefore be distinguished from a large number of functional and organic diseases, most of them belonging to the nervous system, although the number of affections thus to be differentiated is not so great before as after puberty. Great care should be taken in examining children when hysteria is suspected: their intelligence and shrewdness should not be underestimated. The doctor will often discuss in the presence of the child questions of diagnosis, prognosis, and treatment with much greater freedom than before an adult, but most children are observant, and the neurotic and precocious who are the victims of hysteria are particularly keen and watchful, so that the physician may deceive himself in diagnosis and thwart himself in treatment, especially in his attempts at moral treatment, by unwittingly making the child a confidant of his views.

It should be remembered that diagnosis in nervous disease, as in psychiatry, is sometimes a relative matter. A child or an adult is sane or insane according to the individual, social, national, or even racial standard; so it might be said that a child or an adult is hysterical or not hysterical according to the standards by which it is judged.

The complication of hysteria with organic disease is observed in children as well as in adults, although probably not so frequently in the former as in the latter. Gowers directs attention to a number of special instances of this complication, and very truly says that the symptoms which result from a union of these disorders may be most perplexing. Hyperæsthesia, anæsthesia, contractures, and other phenomena are present sometimes in typhoid fever in young girls. Hysteria simulates or masks tubercular meningitis; rheumatic pains persist with hysterical neuralgia; arthritis may set up in an hysterical joint; a laryngeal catarrh may excite hysterical aphonia, and bronchitis hysterical dyspnœa and rapid breathing. Hysterical symptoms not only complicate general diseases, but also frequently accompany other affections, functional and organic. Infantile hemiplegics are sometimes hysterical in high degree, and real diphtheritic paralysis may pass into hysterical palsy and anæsthesia. These are a few of the complications and accompaniments of hysteria which are given by Gowers, and which have been observed by all neurologists in children as well as in youths and adults.

Shaffer calls attention to cases in which symptoms of actual hip-joint disease—and of other articular affections—exist associated with undoubted hysterical manifestations. A boy twelve years old came into his office limping and complaining greatly of his left knee. He had fallen upon the ice, striking upon his knee-joint, which accident was followed by a severe arthritis, but in about ten weeks after the injury he walked as well as ever. Some months later, however, he complained of his knee paining him very much, and was obliged to stop walking, and after this he hopped around, always holding the leg flexed on the thigh in one position. Any sudden motion produced sharp pain, and considerable atrophy of the thigh and leg muscles was present. The boy was thin and anæmic, and evidently very apprehensive about his knee, and was what would be called a nervous boy. The case was evidently one of hysteria imposed upon an original organic pathological condition. He wholly recovered in a few weeks.

"It may be remarked," says Roberts, speaking of a particular case, "of hysteria that, although it imitates every neurotic disorder, the imitation is never perfect. There is always wanting, either in the history or in the symptoms, some feature which is essential to the imitated disease." The case which called forth this opinion was an imitation club-foot; and no one, as he says, ever saw true club-foot come on, as in this patient, in an abrupt way, without pain, convulsion, paralysis, atrophy, or associated symptoms of any sort. These remarks are full of suggestion to one trying to make a diagnosis of hysteria, especially in children. However close the resemblance, scrutiny and analysis of a case will show something, either in the onset, the symptoms, or the course, lacking in the genuine organic disease.

According to Blocq, the diagnosis of a case of hysterical mania may be singularly difficult, the two affections with which it is most likely to be confounded being moral insanity and meningitis. Of course the diagnosis is assisted by a consideration of other hysterical phenomena, such as pain, hyperæsthesia, contractures, and visual changes. The mental disorder proper must be differentiated by the manner of onset, the intermittency of the phenomena, and above all by the presence of these hysterical stigmata.

Forms of ordinary idiocy or imbecility may be for a time supposed to be cases of hysteria, but the progress of such cases will soon reveal their true nature, although the idiot and the imbecile, like their better-furnished brother, may be at times hysterical. The affection described by Maudsley as moral imbecility is sometimes, in its incipiency at least, regarded as hysteria. Maudsley's description of these cases will serve to make clear the diagnosis between them and cases of real juvenile hysteria, although in the latter moral perversion is often prominent.

"There are children," says Maudsley,[1] "of defective mental capacity not reaching the degree of idiocy, or even of positive imbecility, whom it is very difficult to know what to do with sometimes. They are dull, heavy, stupid, appear careless, indifferent, and as if they will not try to learn anything, and display low or vicious tastes; when sent to a respect-able school, they are commonly after some time sent home again as impracticable. Their inability to learn looks very much like stupidity and obstinacy, when it is really the result of disease and marks a certain measure of imbecility. It is sometimes the misfortune of boys of this sort to be sent, after failing at the usual schools, to some one who advertises for unruly pupils and who represents himself as possessed of some specific for managing or training them. A few years since, a boy of this kind was said to have been flogged to death by his master, who was put upon his trial for manslaughter, was found guilty, and received a severe sentence. Without doubt the poor boy was harshly and cruelly used; but there are medical reasons for thinking that the case was not quite so bad as it was represented in the public papers. Dr. Wilks has expressed an opinion to this effect, grounding it upon the fact that in some of these cases of half imbecility there is an abnormal quantity of serum in the ventricles of the brain, and that death may sometimes take place suddenly in consequence of the increase of the fluid beyond a certain extent. In the case referred to, an unusual quantity of serum was found in the ventricles of the brain after death. In reality, the condition of things may have been the cause of the youth's stupidity, and so his death have been occasioned by a punishment which would not have seriously injured a healthy child. Although this would not have been a justification of the punishment, it would still absolve the school-master from some portion of his culpability. When we reflect on the possible state of things in the brain, it will be obvious that no good, but much mischief, will be done by harsh measures; kindness and encouragement, good diet and regular habits, proper bodily exercise, and the regular control of some judicious persons, will be the best means to employ. Above all things it is necessary to forego all attempts to make such defectively-organized beings attain to a mental degree of development which they are by nature incapable of; they should be put to some humble occupation for which they are fitted.

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[1] The Physiology and Pathology of the Mind, 2d ed., London, 1868, p. 328.

"There is another class of boys who cause great trouble and anxiety to their parents and to all who have to do with them. Afflicted with a positive moral imbecility, they are inherently vicious; they are instinctive liars and thieves, stealing and deceiving with a cunning and skill which could never be acquired; they display no trace of affection for their parents or of feeling for others; the only care which they evince is to contrive the means of indulging their passions and vicious propensities. Intellectually they are defective also, for they usually read no better at sixteen than a healthy child of six years, and yet they are very acute in deception and in gratifying the desires of their vicious natures."

The curious affection variously known as convulsive tic, echolalia, coprolalia, and Gilles de la Tourette's disease is sometimes regarded at first as a form of hysteria, and in fact does occur in children with a neurotic family history who may present hysterical symptoms. This affection is, however, rather an hereditary psychosis or monomania, differing from hysteria, among other things, in its persistence and in its narrow range of phenomena. It begins, as a rule, in children between the ages of six and sixteen years, and therefore is properly considered in this connection. It affects boys by preference. It was first described by Bouteille, in 1818, in a treatise on chorea. In 1884 and in 1885 Gilles de la Tourette[1] collected, analyzed, and discussed previous cases, adding others of his own and giving the affection a place in nosology. Dana,[2] in this country, has contributed to the subject. The main features of the disease are incoördinate movements, local or general, sometimes of great violence, and often associated with explosive disturbances of speech. The patient bursts out into some profane or obscene expression, at the same time making a peculiar grimace or contortion. Instead of a spontaneous blasphemy, he may repeat or echo words that he overhears. He often imperatively and explosively utters things desirable to be kept secret. Physicians should not set down these cases as simply hysterical; but they should recognize their seriousness and hereditary nature.

Sometimes in children even more than in adults it is difficult to determine whether a seizure should be termed epileptic or hysterical. It is at times practically impossible to come to any decision. Omitting those cases which are clearly purposive or instances of shamming, the study of other forms of hysterical and epileptic fits must impress us with the kinship between these so-called distinct diseases. As already shown, various combinations of epilepsy with hysteria are often observed: thus, hysterical seizures, more or less severe, may follow slight and brief epileptic seizures; the two forms of attacks may alternate in the same patient; or intermediate cases may be found in which the phenomena of the two diseases are so blended that it seems almost impossible to separate them.

Henoch discusses the cases in which hysterical and epileptic conditions blend or alternate, and in which the diagnosis is correspondingly difficult. He believes that some of the cases which he classes with hysteria may develop into true epilepsy, the tendency of the case to end in this way being greatest when epilepsy is hereditary or when the patient has had true epileptic fits. The description by Henoch of his cases shows the difficulty in sometimes separating hystero-epilepsy from true epilepsy. He speaks of consciousness as lost or weakened, of the patient going about in a dazed or dreamy state, etc. Such patients occasionally hurt themselves, as in the case of one who fell through a window of a cellar. Some are aware they have an attack, others are not. The physician can never be absolutely sure that these cases will not degenerate into true epileptic attacks, although this is not the rule.

The more one's experience increases, the more he becomes convinced that the line of demarcation between some hysterical and epileptic fits is not so sharp and deep as his reading and his first experiences would lead him to suppose.

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[1] Archives de Neurologie, July, 1884, and January and March, 1885.

[2] Journal of Nervous and Mental Diseases, July, 1886, vol. xiii. p. 407.

Cases of the classical type are the most readily differentiated. Charcot's diagnostic points in favor of hysteria, often cited, are: (1) The existence of hemianæsthesia. (2) Affections of sight (dyschromatopsia and achromatopsia). (3) The existence of hysterogenic zones, and the method of provoking or arresting an attack by pressure on these zones. (4) The action of æsthesiogenic agents on the affections of sensibility and of motility.

While the occurrence of nocturnal attacks is rather in favor of the existence of epilepsy, this is by no means a sure diagnostic mark. Not only do night-terrors, which are largely hysterical, occur, but convulsive hysterical attacks are sometimes observed during the night. According to Roberts, these night-attacks find their explanation in the fact that even then somebody is around to note the performance. He gives an illustration of a boy who had paroxysmal attacks of a curious character at night in bed. He had irregular twitchings of the arm and blinking of the eyelids, which developed into general convulsive fits of short duration. He never bit his tongue nor foamed at the mouth, and the question of unconsciousness was doubtful. The mother slept with the boy. The attacks invariably occurred early in the night or after seven in the morning, never in the middle of the night, when sleep is most profound. Roberts holds, and probably with correctness, that the boy would not have had the night-attacks if he had not slept with his mother. During the day he had many hysterical manifestations at different times. After a few months he recovered entirely.

The nocturnal attack, if epileptic, usually comes on with a scream or noise, the convulsion is often general, or at least severe, the tongue is often bitten, the bed is sometimes wetted, and the patient afterwards passes into a deep sleep, and the next day suffers considerably from soreness of the muscles and sometimes from headache.

The diagnosis of hystero-epilepsy from Jacksonian epilepsy due to a lesion causing cortical irritation is sometimes very difficult, and some authorities, as Victor Horsley, hold that hystero-epilepsy is often, if not always, a true cortical disease,—that the spasms are due to cortical discharge. This point has been referred to by me in a paper on "Cerebral Localization in its Practical Relations:"[1] "Some cases which seem to be clearly cases of hystero-epilepsy closely resemble organic epilepsy of the Jacksonian type. Hystero-epileptic attacks, it is well known, can be produced by irritation of the hystero-epileptic zones, described by Charcot, Richer, and others, which are evidently analogous to the epileptogenic zones of Brown-Séquard. Almost every form of spasm in localization and extent can be found in descriptions of hystero-epilepsy. Features of distinction are, however, present. Undoubtedly one reason for the similarity between spasmodic affections, reflex, hysterical, toxic, or cerebral, lies in the fact that in these cases, whatever may be the starting-point, central areas are discharged and give definite character to the convulsions. Horsley speaks of hystero-epilepsy as a cortical disease, but this view cannot be upheld for all cases, if he means by this that the spasms are usually the result of cortical discharge. They are rather sometimes bulbar or spinal, cortical inhibition being removed."

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[1] Transactions of the Congress of American Physicians and Surgeons, 1888.

The following diagnostic characters of epileptic and hysterical fits, as given by Gowers, will answer almost as well for children as for adults :

	"Epileptic.	Hysteroid.
Apparent cause,	Absent.	Emotional disturbance.
Warning,	Any, but especially unilateral or epigastric, aura.	Palpitation, malaise, choking, bilateral foot-aura.
Onset,	Commonly sudden.	Often gradual.
Scream,	At onset.	During course.
Convulsion,	Rigidity followed by "jerking," rarely rigidity alone.	Rigidity or "struggling," throwing limbs and head about.
Biting,	Tongue.	Lips, hands, or more often people and things.
Micturition,	Frequent.	Never.
Defecation,	Occasional.	Never.
Talking,	Never.	Frequent.
Duration,	A few minutes.	Often half an hour or several hours.
Restraint,	To prevent accident.	To control violence.
Termination,	Spontaneous.	Spontaneous or artificial (water, etc.)."

One point which will sometimes assist in the diagnosis of hysterical paralysis is brought out by examining a child in a sitting or lying posture. The child will tell you that she cannot walk, that she cannot even stand, but when sitting or lying all the movements of the limbs can be elicited by adroit persuasion and manipulation.

Almost any form of paresis or paralysis occurring among children might, under some circumstances, be mistaken for an hysterical affection ; for example, any of the paralyses which follow the acute contagious and infectious diseases of childhood, as diphtheria, scarlet fever, measles, small-pox, and typhoid fever. These palsies, however, nearly always have clear marks of their organic origin, the most important of which are trophic and electrical changes, positive alteration in knee-jerk, and peculiarly-distributed sensory disorder. When, therefore, with wasting, lost farado-contractility or the reactions of degeneration are present, the case may be set down as not hysterical. Lost knee-jerk, or a very decided difference in the jerk of the two sides, may usually be regarded as proof of organic disease. When anæsthesia, analgesia, or hyperæsthesia is limited to the anatomical distribution of certain nerves, the disease is likely to be organic. Pathologically the paralyses are, as a rule, due to neuritis or myelitis or a combination of these two affections, and therefore distinct, trophic, electrical, reflex, and sensory phenomena will be present, although, it may be, in varying degree.

For the palsies which follow cerebro-spinal fever or other forms of meningitis, cerebral and spinal, the same rules of diagnosis will suffice, and in these affections the history of the case will usually afford valuable aid. Practically the same is true of acute atrophic paralysis or anterior poliomyelitis,—the infantile paralysis of most writers,—and of the paralysis of multiple neuritis. In infantile cerebral hemiplegia or monoplegia,—paralytic, spastic, or epileptic,—general arrested development, persistent contracture, and in many cases convulsions, more or less localized and generally with unconsciousness, will be present. In these cases, however, the electrical changes will either not be present or will be quantitative only. Knee-jerk may be increased on one side, and sensation in rare instances is permanently impaired.

Rachitis in children is often misleading, simulating not only hysteria, but sometimes very closely serious organic affections incurable in character, as progressive muscular atrophy or pseudo-hypertrophy, poliomyelitis, and also the forms of paralysis following or accompanying diphtheria, scarlet fever, measles, cerebro-spinal meningitis, and Pott's disease. Berg,[1] in describing these cases of rachitic pseudo-paraplegia, says, "The little patient of two, four, or even five years of age is unable to walk, and younger children to sit or stand. Efforts to walk are not made without aid, as a general thing, but when such efforts are made the feet and legs are spread wide apart,—for better basis of support,—the body bent forward to maintain equilibrium. Stumbles and falls are frequent, and the little patient walks like a partial paraplegic. Examination shows a rachitic body; costal cartilages marked by the so-called rachitic rosary; tenderness in the body of the muscles, and over bony prominences at muscular insertions. Demonstration proves that every muscle retains its powers intact."

The points for the diagnosis of disorders of common and special sensibility are given sufficiently in the section on symptomatology.

As appears from the discussion already had upon the symptomatology of hysteria in children, the various painful affections of the joints of the limbs and of the spine are particularly liable to puzzle and confound the diagnostician. Some orthopædic surgeons, and above all Shaffer of New York, have carefully studied these affections from the diagnostic standpoint. Sifting the symptomatology of such cases, it will be found that the cases of neuro-mimetic joint-disease involving such joints as the hip, knee, elbow, etc., will present certain tolerably uniform characteristics, usually well-marked emotional tendencies, with highly-developed self-consciousness, suspicion, and watchfulness; the muscular rigidity or contracture present being generally variable in character and capable of being modified by diverting attention and by various schemes. Shaffer gives the following as the points on which an exclusive diagnosis was made in a certain case:

"1. The emotional element in the child: easily affected to tears without pain. She seemed also very conscious of observation and suspicious of it.

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[1] Medical Record, November 16, 1889.

"2. The rigidity of muscles of thigh variable, as when attention is directed thereto or diverted therefrom, etc.

"3. The flexion of the thigh which occurs when the patient sits in a chair cannot be obtained when she lies down.

"4. Patient can put on her own shoes and stockings.

"5. She attributes disease to and dates back her trouble from an injury eight years ago.

"6. There is more or less hyperæsthetic pain at almost any point of opposite leg, or any part of the body, when pinched or touched.

"7. Uniform temperature; no evidence of suppuration; good appetite; good general condition.

"8. Psoas muscles are not involved in the contraction."

"In this case the extremely variable character of the muscular contraction was the turning-point in the diagnosis; a wide difference existed between the symptoms developed at the formal examination and those shown by the patient when she thought herself unobserved."

Shaffer has shown the great value of the faradic current as a means of precision in the diagnosis of hysterical joint-affections, either in children or in adults. Often in these cases some atrophy from disuse and pressure is present. In organic joint-diseases marked muscular atrophy is present, and is accompanied by loss of electro-contractility. Inflammation of a joint is attended very constantly with wasting of the muscles that move the articulation. According to Shaffer and Seguin, in the organic case the response to the faradic current is lost, or at least depressed.

Powerful narcotic and hypnotic drugs like opium and chloral, and powerful anæsthetics like ether and chloroform, may be able to assist in making a differential diagnosis of rigidity and contracture, but their value must not be overrated. Under ether and chloroform the muscular spasm or contracture, both organic and hysterical, will disappear; it will not, however, so readily yield in the organic case to opium or chloral. As pointed out by Charcot, if under chloroform the rigidity of the muscles of the members gives way slowly, or even persists to any marked extent, a spinal organic lesion is placed almost beyond doubt. Hypnotic suggestion might assist in a differential diagnosis, as during hypnosis spasm or contracture may be readily made to disappear entirely. In some organic cases, also, spasm or contracture might be modified. In natural sleep hysterical rigidity and contracture disappear.

Shaffer gives some striking examples of simulated disease of the spine. From a study of these cases and of his arguments and conclusions, the most important points of differential diagnosis would seem to be that in chronic spondylitis, before the appearance of deformity, rigidity of the vertebral column is usually present and due to reflex muscular spasm, while in neuromimesis, under proper manipulation, normal mobility may be determined;

pain, severe, localized to nerve-lines, and increased by motion or jars, is present in the organic affection, but in neuro-mimesis the pain is superficial, usually located over or near the spinous processes, is sometimes transient, and frequently changes its location; a nocturnal cry and an apprehensive facial expression are usual in Pott's disease and absent in neuro-mimicry; and, finally, the gait and attitude of the organic case are characteristic.

A study of many cases of hysterical or neuro-mimetic spine-disease shows a not infrequent history of the patient's having observed or studied other cases. Here mimicry is easily explained; but in many other cases it is difficult to understand how an uninformed patient can so closely counterfeit a real affection.

"There is this difference," says Shaffer,[1] "between the lateral curvature of hysterical origin and true scoliosis. The former partakes of the character of functional weakness, especially of those muscles which act upon the spinal column extrinsically, while the latter is due to a progressive muscular contraction, dependent upon causes yet to be pathologically ascertained, but which appear to primarily affect those muscles which act intrinsically. The hysterical form does not become a true scoliosis, in my own experience, unless the specific pathological cause be added, and we may perhaps infer that this specific cause is more apt to be developed in the hysterical diathesis, just as we say that chronic joint-disease is more apt to occur in the strumous diathesis. Whatever the pathological condition may be that induces the peculiar condition known as rotary lateral curvature, we at least know that the muscular contraction is both a painless and a progressive one, and that it resembles in character that found in true torticollis, in congenital club-foot, and in many instances in infantile paralysis. The conditions found in true torticollis especially resemble those which are apparent in true lateral curvature; and that this condition is one of contracture, rather than simple contraction, is confirmed by Paget, who says, in speaking of neuro-mimesis of lateral curvature, 'If these signs of distinction are not enough, ether or chloroform will help. You can straighten out the mimic curvature when the muscles cannot act; you cannot so straighten a real curvature.'

"Recognizing, then, the character and persistency of this contracture, it is always a matter of difficulty—if it is not an impossibility, in the absence of the symptoms in the earliest stage—to determine just when the efficient cause of the progressive scoliosis commences to operate. When the spine is markedly curved, and rotation is apparent, the diagnosis is not difficult, and while the tendency of true scoliosis is to become very slowly worse, and to result in irremediable deformity, the hysterical curvature, if properly treated, sooner or later recovers, just as do the emotional contractions of the hip and knee.

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[1] The Hysterical Element in Orthopædic Surgery, Archives of Medicine, April, 1880, vol. iii. p. 186.

"The early stage of hysterical lateral curvature, and the first (apparent) stage of true scoliosis, however, present many features in common, and, as before mentioned, the emotional element is almost uniformly present in each. This adds to the difficulty of diagnosis, and has led to many errors."

The diagnosis of hysterical fever must be made by a careful consideration both of the temperature and the previous history of the child, and of all the circumstances surrounding the case. In many such cases close examination will show, on the one hand, the absence of positive evidence of either local or constitutional disorders, or, on the other hand, the presence of varying and vacillating hysterical phenomena, such as aphonia, transient paresis, wandering paresis, hyperæsthesia, spasms, or morbid psychological manifestations. Pulse and respiration as well as temperature may be exaggerated in cases of hysterical fever, but not uncommonly a feature of such a case is the want of correspondence of these great phenomena of fever, or even the existence of any one of the three as an isolated symptom.

PROGNOSIS.

The prognosis of hysteria in children is, as a rule, good, but the tendency to relapse should not be forgotten. That a child has had one or more hysterical attacks before the age of puberty should lead parents to be on their guard for the return of the disorder in the same or some other form during puberty or adolescence. While difficult to eradicate, the effects of an hysterical inheritance may often be modified to the lasting benefit of the child. Most reported cases of hysteria in boys and girls, even the severe forms with convulsions, anæsthesia, paralysis, contractures, and disorders of the special senses, have eventually recovered. The occurrence of hysteria in young boys and girls may have a prognostic or prophetic importance with reference to the future life and career of the individual. It should sometimes influence the choice of an occupation or a profession; or the child, by particular care and attention to its physical and mental training, should be especially prepared for the profession of its choice.

Hysteria in children, as in adults, is rarely, if ever, fatal, although a few cases apparently hysterical in which death resulted have been reported. Such patients may, of course, perish from accident or intercurrent disease. Gowers says that as a rule, to which exceptions are infinitely rare, hysteroid attacks, however severe and alarming in aspect, are devoid of danger, the laryngeal spasm presenting the greatest apparent risk to life. The same author says that a case mentioned by Reynaud is the only recorded instance of death in an attack of this description. Gowers also speaks of another source of danger,—the tendency to turn on the face which is sometimes seen in the post-epileptic state, which is, strictly speaking, rather an automatic than a hysteroid phenomenon; but the two conditions merge into each other. A patient in whom this symptom was present was a little girl aged twelve, who had never suffered from epileptoid seizures, and had had an attack of hysterical paraplegia immediately cured by strong faradization. "Her fits always occurred on waking out of sleep, before she was quite awake. They commenced with a half-moaning, half-singing noise, and the louder this was the worse was the subsequent attack. Then she gave a sudden spring, and always turned on her face, and presently began to scratch her pillow. She would sometimes rise in bed suddenly and then dash her head back on the pillow. The attacks lasted only a few minutes, and always ended suddenly." He had also met with one instance in a little girl, in which death occurred in consequence of attacks which appeared to be hysteroid, but which perhaps were of intermediate form between hysteria and epilepsy.

TREATMENT.

The prevention of hysteria in very young children is in large part simply the hygiene of the nursery and the removal of bad example. When a mother is hysterical, epileptic, or insane, it is a prophylactic against hysteria, as against other diseases, to have the infant nourished by another, a strong healthy nurse, or, if this cannot be accomplished, the child had better be raised on artificial food. The example offered by the mother, brothers and sisters, and playmates is often bad. The plastic mind of a young, neurotic, precocious child is wonderfully receptive of impressions which may lead to the disturbance of the nervous equilibrium. It is a crime against young children to do what I have seen done more than once, —namely, to allow them to be for weeks in company of a mother who is afflicted with the graver forms of hysteria or insanity. I recall two recent instances of patients suffering from hysterical insanity, both mothers of intelligent and interesting young children. These patients passed through many of the most distressing and violent phases of the gravest hysteria,—at times maniacal, swearing, gesticulating, striking and destroying; at times in a condition of trance, hypnosis, or stupor; at times in cataleptic or hystero-epileptic attacks,—passing from stage to stage through the whole train of neural phenomena which curse a patient of this type. Through it all, and in sight and hearing of it all, delicate, impressionable children were interested and often astonished and frightened observers. Such association can seldom fail to plant the seeds of mischief and misery, perhaps for all time. The drunken or passionate father is, both by example and by the development of fear, the cause of hysteria in children; and the presence of imbeciles and epileptics in a family of young children may lead to the same dire result. Another form of injurious example which should be removed when possible is that of certain local affections; for instance, choreic movements, such as snapping the eyes and other grimaces, twitching of the limbs and twisting of the body, may be developed by imitation, as has been shown under Etiology. These are practically habit-choreas.

"The family physician who discovers a child to be neurotic, and who, from his knowledge of parents, ancestors, and collateral relatives, knows that a predisposition to hysteria or some other neurosis is likely to be present, should exercise all the moral influence which he possesses to have a healthy, robust training provided. It is not within the scope of an article of this kind to describe in great detail in what such education should consist. Reynolds is correct when he says that 'self-control should be developed, the bodily health should be carefully regarded, and some motive or purpose should be supplied which may give force, persistence, unity, and success to the endeavors of the patient.' In children who have a tendency to the development of hysteria the inclinations should not always or altogether be regarded in choosing a method or pursuing a plan of education. It is not always to what such a child takes that its mind should be constantly directed; but, on the contrary, it is often well to educate it away from its inclination. 'The worst thing that can be done is that which makes the patient know or feel that she is thought to be peculiar. Sometimes such treatment is gratifying to her, and she likes it,—it is easy, and it seems kind to give it; but it is radically wrong.' "[1]

The curative treatment of hysteria in children includes measures for the restoration and maintenance of the general health, and remedies for special phases of the disease, such, for example, as convulsion, contracture, aphonia, etc. Tonics are often useful and sometimes necessary, but they should not be resorted to and depended upon blindly. Every case should be carefully studied: one may demand nutrients, such as malt and cod-liver oil with tonics, while another may do better on some reduction of diet, with sedatives or even opiates. Arsenic among mineral remedies, and sumbul, cimicifuga, valerian, and asafetida among vegetable substances, are valuable. Where conditions of excitement are prominent, the sedatives may be carefully used; but their employment should not be long continued. The most valuable of these are sulphonal, chloral, monobromide of camphor, the other bromides, conium, and codeia.

Massage, of much value in adult hysterical cases, has not so great a range with children, although it may be used with advantage, but Swedish movements, and particularly systematized gymnastics and calisthenics, are of the utmost value. Almost any form of juvenile hysteria will improve under carefully-regulated gymnastics.

Hydrotherapy, either general baths, shower-baths, or douches, will often prove of great service, particularly when combined with isolation, electricity, and careful dietetic treatment. One or two baths daily or every other day should be used. Care should be taken not to frighten children by the rough use of water-treatment, which is not resorted to as a punishment, but as a means of invigorating and giving tone to the system. Salt-water baths are often especially serviceable.

Isolation of the patient is often of great importance in the grave forms of hysteria in children. This applies to both the endemic and epidemic forms of the disease, and also sometimes to sporadic cases. Charcot strongly directs attention to this matter. A young Russian, whose case is carefully detailed by him, was cured when he was rigidly isolated from all relatives. The same author refers to an epidemic of hysteria which broke out in a college under the charge of monks near Belfort, and in which the young boys were cured by sending them home to their families.

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[1] The writer, in American System of Practical Medicine, vol. v. p. 275.

Isolation is chiefly a moral measure in the treatment of hysteria; and, after all, the main curative treatment of the disease is moral. Great attention must be paid in some instances to arousing, and in others to controlling, the will. The child should be kept under firm but kind control; it should be encouraged to do for itself, and should be deprived of morbid sympathy.

"As soon as a child is old enough to develop a will of its own, the first thing it does is to try and get its own way, and one of the earliest lessons it has to learn is that it can only have its own way when it is compatible with the comfort and rights of others; and even a mere baby will soon find out how far it may encroach on the kindness or weakness of those around it.

"As we are none of us born models of virtuous behavior, some kind of punishment must necessarily be used now and then in the nursery; but, as far as possible, the child should be made to feel that the punishment is the natural result of his bad action, and not the mere venting of anger and annoyance on the part of the parent or nurse. If a child once finds out that certain actions always entail unpleasant consequences, he will no more think of committing them than he would think of putting his hands in the fire, which, he has early learned, has an unpleasant habit of burning. There are no better philosophers than children, who always resign themselves to the inevitable. Let the child find out that bad behavior in the drawing-room means instant banishment to the nursery; that if he knocks his brother with a stick the result is 'no stick;' that if he refuses to put away his toys one night, he must manage without toys the next night; and so on.

"If the mother merely talks at the child, and says, 'How often must I tell you not to do so?' or, 'I shall send you up-stairs,' the child soon perceives that, after all, this entails no consequences, and he very wisely acts accordingly. On the other hand, nothing should be denied a child without some reason. A great many mothers, and most nurses, act on the principle contained in *Punch's* remark, so delightfully illustrated by Du Maurier, 'Maude, go and see what Baby is doing, and tell him he mustn't.'"[1]

For convulsive attacks such measures may be used as compression of the hysterogenic point,—as, for example, the testicles in boys, the ovaries, spine, or infra-mammary region in girls. Observations have been reported by Dreyfus and Aussillaux where the hystero-epileptic attacks have been stopped by compresses of cold water to the genitals. Inhalations of ether, chloroform, nitrite of amyl, and bromide of methyl, and douches of cold water, have all been successfully employed.

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[1] Jesse Oriana Waller, in the Nineteenth Century, 1889.

In a case of hystero-epilepsy described by Gowers, after several of the attacks had occurred, Dr. Wilson, the attendant physician, passed a line band around the arm near the axilla, with a knot over the nerve-trunks, and as soon as the head began to roll the knot was pulled tight: the attack was immediately arrested, and no other occurred during the night. The patient slept well, but the attacks came on in the morning before she was thoroughly awake, and she had forty before eleven A.M., all being the same in character. Again they were arrested by the ligature, and ceased.

Contractures, although hysterical, are not always readily cured. Some pass away quickly, others remain for years; and Charcot has described the latter as permanent contractures. In the cases treated successfully various measures have been employed, as faradism, galvanism, Swedish movements, hypnotic suggestion, counter-irritation, and many medicines. Shaffer believes that the use of mechanical force in hysterical contracture is positively contra-indicated. Hypodermic medication will sometimes prove of great value, because of both its physical and its mental effects. The existence of atrophy is, of course, of some importance in determining, in a doubtful case, both its nature and its treatment. Functional atrophy from disuse occurs; but when in addition to atrophy there are reactions of degeneration to the galvanic current, the diagnosis of organic disease may be considered certain.

In the treatment of hysterical joint-affections it is important to bear in mind several practical matters. These have been well presented by Shaffer in his series of papers already quoted. It is usually well to stop all local treatment, except, perhaps, massage and manipulation of the joint. Excessive sympathy and attention must be withheld. The child's disease should not be discussed in his or her presence. Introspection must be carefully combated; the patient's mind must be diverted to objects outside itself, and an open-air life, with as much exercise as possible, must be secured. By ingenuity and adroitness, by persuasion, promise, and an occasional show of assistance, both the limp and the contracture can often be either slowly or rapidly overcome.

THE DISORDERS OF SLEEP.

By CHARLES P. PUTNAM, M.D.

It would be outside of the scope of this article to discuss the nature and causation of sleep. Though many theories have been proposed, none of them have obtained general recognition.

Whatever be its nature, sleep brings a time for rest and repair of the whole body, but especially of the brain. To a certain extent the tissues are always wasting and always being repaired, but in sleep the repair goes on at a more rapid rate than the waste. Pettenkofer and Voit found that of the total amount of carbonic acid eliminated during twenty-four hours fifty-eight per cent. is given off during the day and forty-two per cent. during the night, while the amount of oxygen taken in during twelve hours of the night far exceeds that taken in during twelve hours of the day.

Especially for children is a sufficient amount of sleep necessary. Want of sleep quickly interferes with the proper performance of all their functions and seriously interferes with their health.

Hilton says, "In infancy the child who sleeps much mostly thrives. *Mutatis mutandis,* the observation is equally true that the wakeful, restless child seldom displays the evidence of active nutrition. . . . Growth—the renewal of some parts, and the fresh development of others—seems thus to claim sleep and rest as its helpmates."

The amount of sleep required varies at different ages. New-born infants often do nothing but sleep and eat, except when they are being dressed. From fifteen to eighteen hours may be considered the necessary amount at this age. At one year thirteen hours is not too much. Children two, three, and four years old often sleep eleven or twelve hours or even more, and sometimes take a nap in the middle of the day besides. A child that took less than twelve hours of sleep at this age could not be considered a good sleeper. Nor should the sleeping hours of a child up to ten years of age fall below ten hours.

The most common disorders of sleep are night-terrors, somnambulism, nightmare, dreams, and insomnia. Of these, night-terrors are the most common in childhood, and will therefore receive the most attention in this article. What is said about night-terrors will apply also, with certain modifications, to the other affections above mentioned.

In order to understand these disorders, it is necessary to consider certain points in the physiology of the cerebral function in relation to sleep. When observations are made on a person going to sleep (and the converse is true of waking up), it is evident that one function of the brain, then another, is suspended, until enough of them are at rest to constitute what we call sound sleep.

Dreams, somnambulistic attacks, and night-terrors find their explanation in a loss of the higher and an exaltation of some of the lower cerebral functions. The conscious life of the individual is in the realm of the higher functions, and these constantly exercise a kind of inhibition on the lower ones. When by the suspension of the higher functions, those concerned in consciousness, this inhibitory influence is lost, the lower functions, no longer under control, run wild in various ways. When, therefore, these higher functions are suspended, two sets of symptoms are observed,—first, those constituted by the loss of the higher functions; second, those which arise from the unnatural prominence of the lower functions, which manifest themselves in response to various excitations, just as the higher functions would do if they were in working order. These manifestations represent the best that the crippled brain can do at the moment. (Hughlings Jackson.)

The excitation which gives rise to these manifestations may be now a disturbance in the digestive, respiratory, or other organs, now a recollection of events of the day, now something else not to be recognized; and such excitation, from whatever source, may give rise in one case to a dream, in another to an attack of somnambulism, in another to one of night-terror.

W. Bevan Lewis says,[1] speaking of nocturnal crises of the insane, "In sleep, object-consciousness quickly, even *suddenly*, succumbs; subject-consciousness goes *more slowly*, and the more profound depths are not usually reached for *an hour*, or even longer; the reflex excitability of all the nervous centres (spinal also) is reduced,—the organic functions are lessened. In these cases of nocturnal excitement, however, the effect of this periodic lapse of consciousness is to call up more turmoil at lower levels; all those subjective states arising from epi- and ento-peripheral stimuli, or centrally initiated, become the subject-matter of the mental view; all those disconnected and simultaneously originating ideas which crowd the mind, and which in healthy waking states are reduced to serial, orderly thought, now run riot,—and beyond this hallucinations of the special senses prevail."

Lyman gives a similar description of this process in the excellent articles on night-terrors in his book on "Insomnia" and in Pepper's "System of Medicine."

Soltmann, in his article on night-terrors in the "Handbuch für Kinderkrankheiten," refers to Langendorff's investigations on the centres of inhibition of the reflexes, which showed that frogs that had been blinded began to croak when they were touched on the back with a wet finger, although this did not happen in the case of an uninjured frog where the inhibition had not been destroyed; and he thinks that in the case of night-terrors where the inhibitive power of the senses, mainly of sight, is removed,—that is, in sleep or when the eyes are closed,—the reflexes make themselves manifest because no longer under control, an explanation which Schmidt-Rimpler had given for the delirium which had been induced in certain conditions by shutting the eyes or darkening the room.

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[1] Text-Book of Mental Diseases, 1890.

NIGHT-TERRORS.

Synonymes.—Pavor nocturnus; Nächtliches Aufschrecken; Terreurs nocturnes.

A child that has gone to bed apparently well and has slept soundly for an hour or two, or perhaps been slightly restless, suddenly starts with a piercing cry which resounds through the house. He is found sitting up in bed, or standing in the middle of the room, or perhaps cowering in a corner, trembling, screaming with terror, and staring out as if at some definite object. The skin is covered with sweat; the hands clutch each other and anything within reach, or are moved about as if for purposes of defence against some attack; when spoken to, the child evidently does not understand. He calls for his mother or his nurse, but does not know her when she comes, and perhaps alternately clings to and repulses her. He may make it evident that he is afraid of some particular object, some terrible form, human or animal; but much more frequently he exhibits only general, undefined terror. After a time, from a few minutes to an hour, or even longer, he gradually comes to himself, though still frightened, and sometimes to a degree still under the same delusion as before; recognizes persons around him, and seeks and accepts consolation from them. When asked to tell what frightened him, he generally can give no idea, but soon wishes to be put to bed, and usually goes off into a sleep from which he may not awake until morning.

After such an attack children sometimes pass a large quantity of urine or have a movement of the bowels.

Occasionally these "terrors" are reported as occurring twice or oftener during the same night (in one case, according to Dr. West, seven or eight times), but usually there is only one.

Such attacks come on at various intervals,—every night, every few nights, or at longer periods, quite irregularly.

In the morning the child seems about the same as usual, and when questioned about the attack generally denies any knowledge of it, or sometimes any mention of the subject brings a certain puzzled look to the face, and the patient evidently wishes to avoid talking about it. Occasionally older children tell of certain monsters or dangers which had terrified them.

Such attacks occur in all degrees of severity, but they have certain peculiar characteristics. They occur almost always an hour or two after going to sleep, or, if there is more than one attack, the first one is apt to occur at this time. During an attack the patient is for a time quite unconscious of the outside world. The attack is always, as it is called, a "terror," and never of an agreeable character, as is often the case with dreams. Complete or nearly complete forgetfulness of the attack is always observed, only a dim consciousness of something disagreeable being sometimes left behind. The attacks usually occur between the second and the sixth year,—that is, between the first and the second dentition,—though this is not always the case. Attacks of screaming that later develop into well-marked night-terrors occur long before the first dentition is over, and they sometimes continue up to the age of fourteen, or even, it is said, to the age of puberty.

Atkinson says that nocturnal incontinence of urine is frequently associated with night-terrors.

In a very few cases attacks closely resembling night-terrors have occurred in the day (Henoch and others), and occasionally, too, they occur at a later hour in the night than that mentioned above.

Children who have these attacks are generally delicate or excitable. Thus, Steiner voices the general opinion of writers when he says they are "almost never perfectly healthy and robust, but, as a rule, delicate, weakly, anæmic, rachitic, and apt to show besides night-terrors other signs of nervous irritability, especially if they come from weakly or nervous parents." Yet it is noticeable that almost every one uses qualifying expressions to show that good health is not altogether inconsistent with this disturbance.

When a child has an attack of night-terrors, his sleep, though apparently profound, has really only suspended the higher functions of his brain. Then, for some cause not always explained, there occurs a stimulation of the brain, or, rather, of such parts of it as are still active, and there is an explosion of nervous force, which would not have occurred if he had been fully awake or fully asleep. The child's behavior is no longer the result of sober thought. Only imperfectly does he see through his eyes or hear through his ears. That which most impresses him as if seen has no material existence, but is a distorted recollection, or bundle of recollections, which to him are present realities. Sooner or later he regains, generally only by degrees, his full consciousness. He sees his own bedroom, his parents, and his nurse. The images which terrified him just before fade away into comparative insignificance or are altogether extinguished. His desire for accustomed sleep comes back, all the more forcibly because a part of the brain is tired with its exertion, and presently he is sound asleep. While fully awake the brain does not know, or knows only dimly, what happened when only its lower functions were at work, but when the child is again in a similar condition on another night the same recollection may come up, and then he has another night-terror of the same character.

Immediate causes for some attacks are satisfactorily made out; for others they are not easily found or are not found at all. Often there is a disturbance of the digestive organs, after the removal of which the attacks cease. Such a condition may be due either to a weak digestion or to improper food. Less frequently attacks are attributed to catarrhs of the respiratory tract, to enlarged tonsils, to trouble in the ear, to morbid excitement of the mind during the day, to fever, to worms,[1] to teething, to alcohol, stramonium, belladonna, or quinine, to irritation of the skin, and to ill-ventilated bedrooms. (P. Niemeyer.)

A large number of the reported cases are attributed to alarming occurrences during the day. They are frequently reported, for the very reason that they are comparatively of infrequent occurrence.

Thus, Wertheimber reports the case of a boy who on one occasion had been scolded and threatened by a gardener for helping himself to flowers. An hour and a half after going to bed he started up trembling and with open eyes riveted on the corner of the room, screaming, as if in great fear, "Go away, go away, for God's sake!" Another day he heard his father read of some boys who had been ill treated, and he again awoke an hour and a quarter after going to bed, started up with a cry, and was found with his hands folded, trembling and begging for mercy.

Meigs and Pepper report the case of a child who had been bitten by a parrot, and that of another who had been frightened by a white dog.

But in some cases excellent observers have been unable to find any immediate cause that could fairly account for the attacks.

Thus, Steiner says, "I have observed night-terrors in children with quite normal digestion and daily movements of the bowels. Their appetite was just as good on the days when they had attacks as at times when they had none. The majority of my patients were children between the ages of three and six years,—of an age, therefore, when no teething is going on,—and I am not able to cite a single case where the night-terrors could be brought into any causal connection with worms: in a word, all the circumstances which are commonly designated as the regular excitants of night-terrors played in my cases a very subordinate *rôle* or none at all. By this I would by no means let it be understood that certain immediate causes (the predisposition being already there) cannot and do not have a partial influence on the number and severity of the paroxysms, but the important factor in this condition must always be found in a disturbance of the nutrition of the brain. Of these immediate causes the most important are a bad mental training, the telling of ghost-stories to children before bedtime, and going to sleep in the dark. All these things are well calculated to stimulate the lively imagination of children already timid and excitable."

Nevertheless, it is difficult to believe that all of Steiner's cases occurred independently of any external stimulus, so strong is the evidence of others to the contrary. Indeed, some of Steiner's patients were affected with scrofulous ophthalmia and other ailments, that seem quite sufficient to call out night-terrors.

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[1] Debacker reports a case cured by removal of a tape-worm.

That in the complex condition of an apparently healthy child certain physical disturbances might easily be overlooked is not unnatural.

The writer has seen children with night-terrors who were considered by their parents to be perfectly well, but who were apparently cured by taking only the mildest food for their supper,—say, bread and milk,—although they had not shown any signs of digestive disturbance.

Still, there is no question that in some cases every attempt to find an immediate excitant of the attack has been fruitless, and we may well consider whether, as with epilepsy, there may not be cyclic explosions in the form of night-terrors for which the brain is preparing all the time between the attacks, so that the attacks take place not as the result of any disturbance of other organs, nor even under the stimulus of distorted recollections, but purely or mainly as the result of a certain degree of tension acquired by the brain in this interval.

We should also remember that more than one cause may be at work.

Soltmann seems to have something like this idea in his mind when he says, "If we are convinced that the attacks occur without any outside influence,—that is, spontaneously,—and always with terrifying visions, phantasms of sight, with reproduction or fantastic distortion of previous experiences, it then becomes evident that we are dealing with periodical manifestation of irritation resulting from abnormal excitement of the prolongation of optic fibres in the brain,—that is, of central sensation areas (pulvinar of the thalamus opticus, corpora geniculata, quadrigemina, and cortex of the occipital lobe), which, however, in accordance with the law of eccentricity are projected onto the field of vision. It is, therefore, a cerebral neurosis (cerebral hyperæsthesia of the optic tract).

"It seems to me not improbable that a morbid misinterpretation of peripheral sensual excitation can give rise to the attacks, when the source of the irritation is in the optic media,—namely, in the retinal vessels (movement of blood-corpuscles)."

It is well to mention here that, though most observers have recognized only one class of attacks, Silbermann, and after him also Baginsky and perhaps others, believe that night-terrors should be divided into the idiopathic and the symptomatic. Silbermann holds that these classes differ from each other in their symptoms, in the quality of terror, and in the kind of children affected. He says, "In the idiopathic form the patient gazes in horror at an imaginary object; in the symptomatic he is distressed for breath, wrings his hands, and makes motions for defence. In the idiopathic he cries out, 'The black cat!' 'The black dog!' etc. In the symptomatic he makes only short ejaculations, 'Ach!' 'Ach!' 'Weh!' 'Weh!' The idiopathic attacks are expressions of objective terror; the symptomatic, of subjective terror. The idiopathic is a transitory hallucination of the sight; the symptomatic, a sensation of distress arising from digestive disturbance which through the agency of the vagus nerve gives rise to dyspnœa."

He calls the idiopathic form "a transitory hallucination of sight, an expression of imagery of terror (objective terror) caused by abnormally increased irritation of the brain (cortex)," while the symptomatic form he regards as "a reflex neurosis of the pulmonary vagus resulting in dyspnœa and thereby in a sensation of terror (subjective terror)." Idiopathic attacks occur, he thinks, always in children that are delicate and excitable, and without any exciting cause in any other vagus; while symptomatic attacks occur only in strong and robust children, always as the result of a digestive disturbance.

It is hard to convince one's self that there are two classes so definitely separated from each other. It is true that between two individual cases there may be a vast difference in all the particulars mentioned by Silbermann, but, taking all cases together, the degrees of difference are so slight that it is almost or quite impossible to draw a line of demarcation. At least it is often observed that nervous and excitable children have an attack brought on by a definite cause, and equally that children apparently perfectly well have attacks for which no cause can be assigned.

When an attack of night-terror occurs, the question naturally comes up, how likely is it that this attack is connected with any serious cerebral or other disease? There is no doubt that attacks which cannot be distinguished from harmless night-terrors occur as a result of febrile affections, and as symptoms of epilepsy, of tubercular meningitis, and perhaps of other diseases.

The statement that night-terrors pure and simple sometimes turn into epilepsy should also be considered.

Wood, in his "Nervous Diseases," says, "In a large majority of cases night-terrors are not connected with any organic disease of the brain, or with epilepsy." "Night-terrors which are the outcome of serious brain-disorder are rare, and not to be positively distinguished by their symptoms from those of less serious import. They, however, frequently recur several times a night, and continue for many weeks, whilst the night-terror of irritation usually happens only once, and extremely rarely more than twice, in a single night, and does not continue to recur for weeks, except it be at considerable intervals. Moreover, the serious night-terror is almost invariably accompanied by the other manifestations of disorder of the brain's action, which point out its true meaning."

In the *Medical and Surgical Reporter*, December, 1889, Wood says (if correctly reported), "Night-terrors are not rarely the precursors of epilepsy." He was describing the case of a boy who screamed at night from the age of three to nearly eleven. The boy was wakened with difficulty, and sometimes had a condition of perverted consciousness in which he recognized his parents but believed himself beyond their help and sympathy. Then for six months he had attacks of running in a circle and falling unconscious. After that he had a tremor, and a little froth was noticed at his mouth. Meanwhile the night-terrors had ceased. Wood believes the trouble in this case to have been epileptic.

Soltmann says, "Attacks [of night-terrors] are observed to go over into epilepsy, . . . and there are cases where it is impossible to say whether a given attack is night-terror or epilepsy."

Moizard quotes Jules Simon as having seen several cases where epilepsy occurred in the form of hallucinations and night-terrors, and as having reported a case where epilepsy followed night-terrors. Moizard also quotes a case of Debacker where the following succession was noted: night-terrors at the outset, then hallucinations, somnambulism, and epilepsy. One of Moizard's own cases had a similar course.

Henoch reports a case where night-terrors took the place of epilepsy. A ten-year-old girl had had a great many epileptic attacks at intervals three years before, then after an interval she had several more, which were soon after associated with hallucinations and screaming, and a month later these epileptic attacks entirely disappeared, and their place was taken by night-terrors, which, however, sometimes occurred twice in one night.

Lyman says, "Insanity, hysteria, neurasthenia, epilepsy, chorea, and nervous dyspepsia are often discovered among their near relatives." West says that long-continued attacks may issue in serious disease, and Bouchut almost the same.

Money, also, says, "Night-terrors are a species of reflex cortical epilepsy."

This is the evidence, such as it is, on the connection between night-terrors and epilepsy. But it is noticeable that of these authors Wood cites but one case, Moizard three, and Henoch one, the rest of the statements being quite general. Although Soltmann speaks of epilepsy in the article on night-terrors, he does not speak of night-terrors in the article on epilepsy, which immediately precedes it and which treats of epilepsy at great length. At least eight writers in treating of night-terrors do not mention epilepsy at all. Nor am I aware of any article on epilepsy which mentions this connection between the two affections. Epilepsy occurs early in the morning more often than at night, and the hour usually varies in a series of attacks. Altogether, the connection between night-terrors and epilepsy, in so far as they are separate diseases, is no clearer than that between any two of the neuroses; and yet, inasmuch as attacks closely resembling night-terrors are occasionally only symptoms of epilepsy, it is well to watch carefully for a time before deciding that epilepsy is not present.

Prognosis.—The prognosis, so far as the night-terrors themselves are concerned, must be regarded as good, even though the attacks last for a great many years. It must, however, not be forgotten that in a great majority of cases they are symptoms of a neurotic condition, and that the same instability which gave rise to them is likely to show itself in other forms of nervous disease and in general lack of vigor.

Persons thus affected are likely to be candidates for hysteria, neurasthenia, and analogous diseases. On the other hand, it must be said that many children who are subjects of night-terrors are at the time in good health and remain so during their whole lives, and that many who are in delicate health at the time that they have the attacks grow up to have average strength.

When the attacks are only symptoms of epilepsy, rabies, or some other disease, of course the prognosis depends on that disease.

The possibility that night-terrors will turn out to be the precursors of epilepsy and other diseases has been spoken of already. Certainly the proportion of cases that turn out in this way is very small.

Treatment.—The treatment of the affection must be directed to the source of peripheral irritation, if one can be found, and to the general health, including the acquirement of a stable brain. Disturbances arising from indigestion, from catarrh of the mucous membranes, from enlargement of the tonsils, from worms, from teething, or from other analogous disturbances, naturally call for their appropriate treatment. My experience has been that sweets, jellies, jams, etc., taken at supper are more likely to cause such disturbances than foods of any other one kind usually found in an ordinary diet, even in cases where there is no special evidence that the digestion is out of order. Cases have been reported where the removal of tonsils, the relief from worms, or the development of teeth was followed by relief from night-terrors.

No doubt, as has been said in regard to frights, greater emphasis is laid on these disturbances as causes of the disease than on the more common ones, for the very reason that they are uncommon.

Various remedies have been used with success for the general health,—iron, phosphorus, cod-liver oil, salt-water bathing, fresh air in the bedroom, light gymnastics, etc.

When the attacks occur frequently, it is well to give bromide of sodium or of potassium, in doses of from two to ten grains, either at night or three times a day, in order to break up the habit. Chloral also may be used in small doses for a similar purpose, especially when the early part of the night is disturbed and restless, and it may be combined advantageously with the bromides; but this drug should be given with the greatest care, as children readily become accustomed to it.

It seems almost unnecessary to suggest the avoidance of ghost-stories and other exciting tales at bedtime; on the other hand, going to bed in the dark, which has been regarded by many as a serious evil, will hardly be so considered in this country.

Wertheimber praises the use of quinine, and thinks it has a direct sedative action on the cerebral cells,—an explanation which seems to be contrary to the general reputation of quinine. Jacobi[1] also reports a case of cure by quinine (or, more strictly speaking, by sulphate of cinchonine) when he thought the affection was due to intermittent fever.

A digestible and nutritious but not stimulating diet should be recommended.

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[1] American Medical Monthly, 1861.

DREAMS, NIGHTMARE, SOMNAMBULISM.

As regards their nature and causation, much that has been said of night-terrors applies also to dreams, nightmare, and somnambulism. They are not pre-eminently affections of childhood.

Dreams are not at all uncommon among children : even in quite young infants a smile or a frown is often seen to pass over the features in sleep, showing that part of the brain is active ; and from the time that children are able to distinguish the experiences of the night from those of the day, they often relate their dreams. Dreams are known only as they are reported by the dreamer, who recollects certain occurrences that seem to have taken place in the night. There is, strictly speaking, no outward manifestation of them, for when dreams give rise to any motor action they are properly to be classed under the head of nightmare, somnambulistic attacks, or night-terrors.

Nightmare may be considered a peculiar kind of dream, in which sensations of distress are coupled with a feeling of inability to move or save one's self from some terrible situation, or similar symptoms.

Somnambulism, though literally meaning sleep-walking, should properly include all motor action in sleep. In somnambulistic attacks the patient while still asleep walks about, or performs various other acts, such as he might have performed in the day,—sometimes, also, acts which he could not have performed in the day, such as climbing on dangerous ledges, hanging out of windows, etc.

Although adult somnambulists have done intellectual work which they could not have performed in the day, this does not seem to have been observed in children.

The patient when awake has no knowledge, or only a dim recollection, of what has happened in a somnambulistic attack.

If we regard as somnambulism everything in which cerebral action results in motor action, we must include under it night-terrors, which are distinguished from somnambulism mainly by the fact that it implies also emotional distress in a condition of partial sleep.

These affections are closely related to each other and to night-terrors, inasmuch as they too depend on suspension of certain cerebral functions and undue activity of others.

They seldom, perhaps never, give rise to serious disturbances, but, on the other hand, they often indicate that the general health is more or less impaired. In the rare cases where they require any special interference they can be treated on the same general principles as night-terrors. The warning that they give of some possible impairment of the health must not be overlooked.

INSOMNIA.

Insomnia occurs in children, though much less often than in adults. It is, however, for the child a more serious affection than it is for the adult.

No child can lie awake for several nights, or even for only a considerable part of each night, without suffering seriously from it.

The causes of insomnia are often the same as are found in adults, but the most common of them are indigestion, over-fatigue, and nervous excitement. Parents and nurses are more responsible for the sleeplessness of the child than they are likely to be aware of. It may be observed that from earliest infancy certain nurses, without any apparent effort, bring about a great deal of sleep for a child, while other nurses unwittingly keep a child awake and on the *qui vive.* Infants that are habitually put into the cradle asleep and snatched out of it as soon as they wake do not feel as much at home there as those that are put in awake and left to lie awhile after waking. When, therefore, infants thus trained awake, they are startled at their unexpected surroundings, and sometimes not readily put to sleep again.

The treatment of insomnia in infants and children is similar to that in adults, but, while the number of ordinary causes for insomnia is fewer in children, the influences that bring it about are often slight and difficult to discover. I have already spoken of the influence which a nurse may have, without knowing it, upon a child's sleep. Insomnia may be caused by hunger or by an overloaded stomach. It is due sometimes to cold, but much oftener to heat and want of ventilation. Children require even more ventilation in the night than in the day, because the doors are not opening and shutting in the night.

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IDIOCY AND IMBECILITY.

By EDWARD N. BRUSH, M.D.

THE various grades of mental deficiency observed in children are usually grouped under the two heads idiocy and imbecility, with the occasional addition of a third term,—feeble-mindedness. These terms represent varieties of mental power and activity in an ascending scale, from the idiot, the representative of the lowest expression of human intelligence, to the child whose mental powers are not markedly deficient, but in whom there are nevertheless evidences of mental enfeeblement.

Idiocy may be defined as mental deficiency of varying grades down to extreme stupidity, resulting from imperfect development or disease of the nervous centres, either pre-natal or occurring before the evolution of the mental faculties in childhood.

While idiocy and insanity are usually regarded as distinct conditions,—the former due to faulty development, the latter an acquired condition,—a moment's consideration will show that the contrary proposition is frequently true. The moral imbecile has his counterpart in the case of so-called moral insanity, and the erratic conduct and one-sidedness of the feeble-minded child are but earlier manifestations of the "partial insanity," "paranoia," "primäre verrücktheit," or "folie héréditaire," of later development.

It seems within the bounds of modern teaching to say that the mental disturbances which may occur at the critical periods of life, puberty and the climacteric, and which are so commonly found to be associated with a family history of some form of neurosis, are in many instances the result of an imperfect nervous organization which has given way under the general physiological perturbations of these periods. Had this defective nervous organization been more distinctly marked, idiocy in some of its grades would have resulted; under the conditions which did exist, the nerve-centres performed their functions with sufficient correctness until an unusual strain added a new factor to the problem.

Etiology.—In a broad sense etiologically idiocy may be said to be due to defective brain-development. The etiology of the defective development must be sought for in the life-history of the preceding generation or of preceding generations, paternal or maternal, or both. The hysterical, the neurotic, the cachectic (syphilitic, tubercular, etc.), the insane, and the drunken ancestry of idiots are, in many instances, the sources to which we must look for etiological information. In the variety of idiocy known as cretinism[1] heredity and environment are almost the only factors which need to be dealt with in a consideration of its causation.

Statistics gathered in Europe show that male idiots are more numerous than female. This disparity has been explained by the greater liability to injury of the male head at delivery from forceps, prolonged pressure in the maternal parts, etc., owing to its greater size.

Injuries to the fœtal head during the period of utero-gestation play a not unimportant part in the production of idiocy, and are to be considered etiologically in conjunction with possible head-injuries during prolonged labor or from forceps delivery.

Shuttleworth, superintendent of the Royal Albert Asylum for Idiots, England, states[2] that prolonged labor, without instrumental interference, is the assigned cause of idiocy in twenty-nine per cent. of the cases admitted to that asylum. He is of the opinion that judicious instrumental interference will in many cases prevent the evils of too protracted compression.

Down[3] states that in twenty per cent. of two thousand idiots examined by him there were marked symptoms of suspended animation at birth.

Crichton Browne[4] has contributed two valuable papers upon the agency of protracted and abnormal labors in the production of idiocy and other mental diseases. He is of the opinion that with advancing civilization there has been an increase in the size of the human head without a proportionate increase in the pelvic diameters. To this he attributes in some degree the preponderance of idiocy in civilized over savage nations.

The health of the mother during the period of pregnancy and the accidents incident to that period have more or less influence in the production of mentally deficient offspring. Down (*op. cit.*) states that in twenty per cent. of the cases which he observed there was a history of disturbance of the mother's physical health during pregnancy. In many other cases there were histories of falls, hemorrhages, etc. The nervous state of the mother at this period doubtless has some influence. The same observer found in thirty-two per cent. of his cases a history of great anxiety, emotional excitement, or fright on the part of the mother during pregnancy. It may be questioned how much of this nervous and emotional disturbance was but the expression of an unstable nervous system in the mother, which, being transmitted to the child, resulted in idiocy.

Carpenter[5] records some remarkable results of fright and anxiety in pregnant women as observed in ninety-two children born in Landau shortly after its siege. Sixteen were still-born; thirty-three died within ten months;

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[1] *Ante*, vol. ii. p. 279.

[2] British Medical Journal, January 30, 1886.

[3] Transactions of the Obstetrical Society of London, December, 1876, vol. xviii.

[4] West Riding Lunatic Asylum Medical Reports, vols. i. and ii.

[5] Human Physiology, Philadelphia, 1876, p. 921.

eight were idiotic, and died before the age of five years; and two had numerous fractures of the bones of the limbs.

Griesinger[1] says, "Violent shock and grief during pregnancy appear not to be without influence" as a cause of idiocy. This author also lays some stress upon great anæmia of the mother as a causal element.

Intemperance in one or both parents, and especially intoxication at the time of conception, is regarded by many as a fruitful source of mental weakness in the offspring.

The influence of consanguinity is a point so much disputed that the subject has become a difficult one to discuss. According to Mr. Alfred Huth,[2] the data are misleading and defective. However, the consensus of popular opinion is undoubtedly against consanguineous marriages. Mr. Huth is of the opinion that mere consanguinity has little or nothing to do with the question, and that such marriages, where there is no family predisposition, are harmless. It seems certain that when there is a family defect, neurotic or otherwise, a union of two elements of the same stock would be more liable to result in a defective offspring than in cases where one of the parents came of a distinct and possibly stronger stock. Between perfectly healthy persons of good family history, such marriages are probably harmless.

Dr. F. Norton Manning presents some valuable data in an article entitled "A Contribution to the Study of Heredity,"[3] based upon a study of fifty idiot and imbecile children under care. These fifty children, twenty-nine males and twenty-one females, belonged to twenty-one families, in which there were eighty-two children in all, forty-eight males and thirty-four females. He divides these twenty-one families into four groups. In the first group, including three families, in which there were no consanguinity and no known insane relatives, there were six children, four males and two females, all imbecile. The second group consisted of only one family, in which there were eight children, of whom three were most unmistakably imbecile, as the result, it is asserted, of accidents to the mother during pregnancy. In the third group, in which twelve families are represented, forty-four children are included, twenty-six males and eighteen females. In this group there was no consanguinity of parents, but there was marked insane and imbecile heredity. In five of the families there was insanity on both sides, and of the eighteen children of these families twelve, four males and eight females (all the females), were imbecile or idiotic. In the remaining seven families insanity was present on one side only. Of the twenty-six children of this group, sixteen males and ten females, fourteen, six males and eight females, were idiotic or imbecile. The fourth group contained five families. In three of these families the parents were brother and sister, in the remaining two they were first-cousins. Of the twenty-four children, fifteen males and nine females, fifteen, thirteen males and two females, were idiotic or imbecile. Of the seventeen children whose parents were brothers and sisters, nine only were idiots, but they were profoundly so; while of the seven children whose parents were cousins, six were imbeciles and idiots. In these instances, however, there was a family history of insanity, several cousins of the parents being insane.

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[1] Mental Pathology.

[2] The Marriage of Near Kin.

[3] Australian Medical Gazette, August, 1885.

The cases above recorded exhibit in a striking way the influence of heredity and in a measure the results of consanguineous marriages, although deductions drawn from these cases alone might be misleading.

An example of the misapplication of facts is shown by the frequent quotation of the statistics of Dr. Howe by writers who wish to demonstrate the harmfulness of consanguineous marriages. Dr. Howe says,[1] "The statistics of the seventeen families, the heads of which, being blood-relatives, intermarried, tell a fearful tale. Most of the parents were intemperate or scrofulous; some were both the one and the other; of course there were other causes to increase chances of infirm offspring besides that of the intermarriage. There were born unto them ninety-five children, of whom forty-four were idiotic, twelve others were scrofulous and puny, one was deaf, and one was a dwarf. In some cases all the children were either idiotic or very scrofulous and puny. In one family of eight children five were idiotic." Writers who quote this passage lose sight often of the qualifying clause which Dr. Howe introduces.

The elements of drunkenness and scrofula would alone account in a large measure for the degenerate offspring. Indeed, it has long been observed that the parentage of idiocy is, as a rule, of a degenerate and weakly order, to which the scrofulous and tubercular belong.

When all the etiological factors of which we have any knowledge have been considered,—the degeneracy of families, consanguinity, ill health or accidents during pregnancy, heredity, etc.,—we are still met by cases that cannot be explained by any of these. Parents with excellent family history, not even remotely related, with other children mentally and physically sound, occasionally give birth to idiotic children. Such accidents are often wholly inexplicable: we can only conjecture that in some way the cerebral development of the child *in utero* was arrested, with the natural result, deficient mental power.

A consideration of the etiology of idiocy would be incomplete which made no reference to those forms of mental defect resulting from arrested development during infancy and early childhood. Doubtless many of these cases in their remote origin are due to causes above enumerated, which, however, do not become fully operative until extra-uterine life has advanced to some degree. Other cases are purely accidental in origin, arising from the diseases and accidents of infancy and childhood. Such are the cases of idiocy arising from convulsions, epileptic or otherwise, from trauma, meningitis, acquired hydrocephalus, rachitis, etc. These various causes will be more fully enumerated and discussed in referring to the various forms of idiocy. As has been said, some causes are purely accidental, preventing the development of a perfectly healthy brain in a child of unexceptionable heredity, while other apparent causes are but exciting, setting in motion latent elements or producing the strain which overthrows a weak nervous organization.

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[1] On the Causes of Idiocy, p. 35.

Statistics as to the relative frequency of idiocy are unsatisfactory and often misleading. Those collected in foreign countries are inapplicable to the conditions of life in America.

The census of the United States for 1880 returns 76,895 idiots—45,309 males and 31,586 females—in a population of 50,155,783. Of these, 63,311 were native whites, 4007 were foreign whites, and 9577 belonged to the colored races. The ratio to population is 1 to 652. These statistics probably approximate the truth; but the natural hesitancy of parents to report idiotic children, and the confusion, even in professional minds, between dementia and idiocy, have doubtless been the cause of many errors and omissions.

The mental and physical condition of idiots varies with the individual. Idiocy of the lower grades is usually associated with some physical malformation. Congenital idiots very frequently exhibit a deficient physical as well as mental organization. This is not so often true of those cases which are occasioned by an arrest of the mental growth during infancy or early childhood. A reference to some of the more marked physical defects will be made when considering the forms of idiocy.

In many cases muscular co-ordination is impaired; the bodily movements are awkward, irregular, and occasionally choreiform. Speech is disturbed in some instances, in some cases being wholly unintelligible, in others the voice being harsh and ill modulated.

Among imbeciles are found occasionally the so-called *idiot-savants.* Gottfried Mind, an imbecile cretin, was one of this class. So skilful was he as a delineator on canvas of cats, singly and in groups, that he came to be known as the Katzen-Raphael,—the "cats' Raphael." The writer saw in the Royal Albert Asylum, in England, an imbecile youth, who had only sufficient mental capacity to do simple errands, yet who could without hesitation tell the day of the week upon which any date for years past or to come would fall. I have now under observation an imbecile of rather low grade who has a remarkable memory for dates. He is able to tell the anniversaries of the birth or death of innumerable poets, authors, and statesmen, and can give at once the dates of many of the great events of the world's history. He has also some ability in acquiring languages, knowing something of Greek, Latin, French, German, and Scandinavian. An idea of his general intelligence is given by his estimate of the cost of a building which he had watched during its construction for some months, the cost of which was several thousand dollars. When asked its probable cost, he replied, "One hundred and fifty dollars," and, seeing a look of incredulity in his questioner's face, quickly lowered his estimate by twenty-five dollars. Blind Tom also belonged to this order.

Diagnosis.—In many instances diagnosis is not difficult, even in the earliest stages of infancy, but in others it is no easy matter to decide, until considerable time has elapsed, whether we have to deal with a case of slow development, a backward child, or a case of mental enfeeblement.

The development of normal children is by no means in accordance with any fixed rule, nor does it proceed by regular gradations. Impaired or deficient nutrition, acute diseases, and other causes hinder and even at times set back the physical and mental development of infants, and should be borne in mind in forming an opinion. Defects in the sense-organs should be looked for, especially deafness, as these may lead to a diagnosis of deficient brain-power which does not exist.

If there are other children in the family, the history of their mental development and the progress of their physical attainments will form the best standard for comparison that can be obtained. At what age did they appear first to notice mother or nurse? when did they first attempt to talk? when did they begin to creep or stand or walk?

There are children who are backward, who develop slowly, both bodily and mentally. Dentition is delayed, they do not attempt to stand or walk until long after the usual period, speech is slowly developed, and in every way they retain the appearance and manners of infancy until well advanced. Such children often cause great anxiety to parents lest this mental and physical hebetude be continued through life and idiocy or imbecility result.

Of such children Seguin long ago said, "The idiot even in the slightest degree presents an arrest of development both of body and of mind; the backward child does not remain stationary, but his development goes on more slowly than that of other children at his age."

The backward child at four or six may represent in general intelligence the ordinary child of two or three, but repeated examinations and the statements of parents will show, if it be a case merely of retarded development and not one of idiocy, that in many ways it is in advance of the child of younger years. There will be a history of both intellectual growth and physical growth, slow, to be sure, but regular and consistent the one with the other, which cannot be shown in the idiot or imbecile.

Many cases of idiocy, where there is but slight defect, do not show it until the age of two or three years. Deficient physical powers, incoördinate movements, and an ungainly aspect may excite fears, which, as the period at which greater mental and physical ability is expected is reached, are confirmed. Where but slight degrees of mental deficiency—imbecility or feeble-minded states—exist, the diagnosis, especially in the early years of childhood, is difficult. The physical powers may be normal, the body well formed, the expression pleasing and intelligent. In these cases it is only as time progresses that the mental defect becomes manifest. Some show it at the school-age, others at puberty, and still others later in life. In these latter the defect is usually in but one or two directions, a one-sidedness of character which attracts attention. Ireland[1] says, "I have seen individuals who had sufficient mental power to pass college examinations, take degrees, and even gain prizes, who were so manifestly unfit to conduct themselves in the ordinary affairs of life that they were the laughing-stock of the most ignorant people around them."

To this class belong the moral imbeciles,[2] children who early show, amid the best surroundings and training, a perverted moral sense, exhibited by wilful disobedience, phenomenal cruelty, or systematic mendacity.

In some of these cases it is difficult, as Spitzka says, to distinguish between states of inherent mental weakness which may be properly called imbecility, and similar states which are more correctly classed as insanity. The clinical and etiological resemblance of the various states of arrested mental development to the degenerative insanities has already been referred to at the beginning of this article.

Classification.—Various classifications of idiocy and imbecility have been proposed. As will readily be conjectured, an ideal classification is impossible. If it is based upon etiology, the same case may belong in two or more subdivisions; if upon mental capacity, it is difficult to fix a standard of comparison.

The classification of Dr. Kerlin, the able medical superintendent of the Pennsylvania Institution for Feeble-Minded Children, is as follows: I. Idiocy: (*a*) apathetic; (*b*) excitable. II. Idio-imbecility. III. Imbecility: (*a*) lower grade; (*b*) middle grade; (*c*) high grade. IV. Juvenile insanity.

Dr. Shuttleworth, a well-recognized English authority in this field, and the medical superintendent of the Royal Albert Asylum for Idiots, Lancaster, England, proposes[3] the following classification:

CLASS A.—CONGENITAL.

1. Microcephalic.
2. Hydrocephalic (also non-congenital).
3. Scrofulous—"Mongol type."
4. Sensorial (also non-congenital).
5. Primarily neurotic.
6. Paralytic (also non-congenital).
7. Choreic (also non-congenital).
8. Cretinoid: (*a*) sporadic, (*b*) endemic.

CLASS B.—NON-CONGENITAL.

a. Developmental.

9. Eclamptic.
10. Epileptic.
11. Syphilitic.
12. Post-febrile (also *accidental*).

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[1] Idiocy and Imbecility.

[2] Kerlin, Medical News, March 19, 1887; Blandford, Insanity and its Treatment, p. 365; Savage, Insanity and Allied Neuroses, p. 272; Spitzka, Insanity, its Classification, etc., p. 281.

[3] British Medical Journal, January 30, 1886.

b. Accidental or Acquired.

13. Toxic.
14. Traumatic.
15. Emotional.
16. From mixed causes.

Dr. Shuttleworth confirms by this classification what has been said as to cases belonging in more than one subdivision of the classification. It will be observed that he makes a broad general division into congenital and non-congenital. He remarks, in the course of the lecture in which this classification is proposed, that the subdivision "developmental," which he places under the head "non-congenital," might with some propriety be retained in the congenital group.

While the idiocy in these cases may not be developed until childhood is somewhat advanced,—the first or second dentition, for example,—there can be no doubt that in many of them the cause of the mental catastrophe is innate.

Dr. Langdon Down[1] has, owing to a resemblance, observed by himself and others, of certain idiots to the various races of men, proposed a grouping upon an ethnological basis into Caucasian, Ethiopian, Malay, and Mongolian types.

Dr. Ireland, the most recent English author on the subject, proposes, in his work,[2] the following classification, which has been adopted in this article: I. Genetous; II. Microcephalic; III. Eclamptic; IV. Epileptic; V. Hydrocephalic; VI. Paralytic; VII. Cretinism; VIII. Traumatic; IX. Inflammatory; X. Idiocy by deprivation.

Genetous Idiocy.—As the majority of cases of idiocy are congenital, the use of this term by Dr. Ireland is perhaps unfortunate; but he distinctly excludes from this class those cases, though congenital, whose pathology is apparent, as microcephalic, hydrocephalic, etc., and includes all those of pre-natal origin whose cause is not well made out. These are the cases in which the force of heredity is most marked, and among them are included those occasional instances of idiocy which seem to mark a commencing diminution of vital force in the parents, the idiot being the youngest of a large family, or the child of parents of whom one or both are of advanced years.

Phthisis, scrofula, and rachitis are found in these children, and many of them eventually succumb to these diseases. The general bodily condition is usually below par, the skin is cold, the circulation feeble, and sensibility impaired. These patients seem to have received, with the heritage of a defective brain, a feeble, dwarfed, and crippled body. The fingers and toes are short and wad-shaped, the feet or hands may be deformed, and vision or hearing defective. Not infrequently there is some defect in the ocular apparatus: the eyelashes may be absent or inverted, or the lids may be everted, and coloboma iridis is occasionally present. Nystagmus and strabismus are not uncommon. The ears are occasionally misshapen and shrivelled. The teeth appear late, are defective, and decay early. The lips are thickened and often fissured, and the saliva dribbles away. Owing to the imperfect muscular control over the lips and tongue, there is often defective and stammering speech in those cases in which the speech-faculty is not wholly wanting. Hare-lip and cleft palate are occasionally observed.

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[1] Observations on an Ethnic Classification of Idiots, Reports of London Hospital, 1866, vol. iii.

[2] Idiocy and Imbecility, by William W. Ireland, M.D. Edin., Medical Superintendent of the Scottish National Institution for the Education of Imbecile Children.

In many of these cases, as in some other forms of idiocy, a high and narrow arch of the palate is present. The arch is usually narrower behind than in front, and in some instances there is a narrow furrow running along the middle of the arch. Dr. Ireland says of the high narrow arch, "It might be compared to the inside of a saddle viewed from below, the pommel being turned backward, for the arch is sharper behind than in front."

In five hundred and seventeen cases examined by Dr. Howe, twenty-one were blind, twelve were deaf, twenty-three had some defect about the mouth or nose, fifty-four had deformed hands or feet, and in ninety-six paralysis of one or more muscles was observed.

It is among idiots of this class that the Mongolian and other ethnic forms are found. Of these the Mongolian type seems to be the most common. The following is Dr. Down's description of the Mongolian idiot: "The hair not black as in the real Mongol, but of a brownish color, straight, and scanty; the face flat and broad, and destitute of prominence; the cheeks rounded and extended laterally; the eyes obliquely placed, and the internal canthi more than normally distant from one another (the epicanthic fold often abnormally large); the palpebral fissure very narrow; the forehead wrinkled transversely, from the constant assistance which the levatores palpebrarum derive from the occipito-frontalis muscle in the opening of the eye; the lips large and thick, with transverse fissures; the tongue long, thick, and much roughened; the nose small; the skin has a slightly dirty, yellowish tinge, and is deficient in elasticity, giving the appearance of being too large for the body.

"This type occurs in more than ten per cent. of cases: they are always congenital idiots; they have considerable power of imitation; they are humorous; they are usually able to speak; the co-ordinating faculty is abnormal; the circulation is feeble; the improvement which training effects is greatly in excess of what would be predicated if one did not know the characteristics of this type; the life-expectancy is, however, far below the average, and the tendency is to tuberculosis."

Among genetous idiots are some of the most favorable cases, so far as training is concerned, and, on the other hand, some of the most hopeless.

MICROCEPHALIC IDIOCY.—The impression prevails to a great extent that idiots have, as a rule, small heads, but observation shows that this is not the case, the microcephales forming but a small proportion of the whole number of idiots and imbeciles. The skull in idiocy shows frequently lack of symmetry, but, excluding the two varieties microcephales and hydrocephales, it will be found that the skull of the idiot has upon an average nearly as great a circumference as that of the normal child of the same age or general physical development.

Spurzheim, Gall, Combe, and others attempted to demonstrate in a measure the truth of their phrenological doctrines through observations made among idiots in whom certain mental faculties—as, for example, memory—were prominent;[1] but their views have no influence upon the present opinions as to the causes and pathology of idiocy. A perfectly-formed cranium often contains a brain almost wholly incapable of mental function.

Ireland considers that a cranium below seventeen inches in circumference is incompatible with active mental powers, and as a matter of convenience he fixes this as a standard of microcephaly.

In the majority of instances the skull of the microcephale is oxycephalic. Few cases reach ordinary stature, and not infrequently a dwarfed body is associated with microcephaly. Occasionally these unfortunates are exhibited as relics of a lost race, as, for example, the so-called Aztec children who are figured and described in Dalton's Physiology. They were a boy and a girl aged respectively about seven and five years. The boy weighed twenty pounds, and was two feet nine and three-fourths inches high. The antero-posterior diameter of his head was four and one-half inches, the transverse less than four. The girl was two feet five and one-half inches high, and weighed seventeen pounds. The antero-posterior diameter of her head was four and one-third inches, the transverse only three and three-fourths inches.

Microcephales are active observers of what goes on about them, are frequently restless and active, sometimes irritable and pugnacious. They are not retentive of impressions, and are therefore not capable of more than the simplest training and instruction.

The brain of the microcephale, according to Gratiolet, has "stopped growing too soon," and, while there is some question as to the cause of microcephaly, it seems very evident that the real condition is one of arrested development (pre-natal) of the cerebral hemispheres. Virchow, and others following his dictum, have advanced the theory that microcephaly is due to premature synostosis of the cranial bones; but, while this condition has been found in certain cases (Bailarger reports several instances, three in one family, in which the fontanels were closed at birth), it cannot be regarded as the cause.

Post-mortem examinations show that the smallness of the brain is at the expense of the cerebrum, the cerebellum being much larger in proportion than in the healthy brain. In the cerebrum some of the lobes are usually diminished in size, sometimes being quite rudimentary, leaving the deeper folds and structures of the brain uncovered.

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[1] Combe, Observations on Mental Derangement, p. 277 *et seq.*; Spurzheim, Observations on the Deranged Manifestations of the Mind, p. 119 *et seq.*

ECLAMPTIC IDIOCY.—This form of idiocy is one that occurs during the development of the child after birth, and is due to the convulsive seizures of that period. Epileptic convulsions are not included in the causes of this form of idiocy, as they relate to another variety.

In regarding eclampsia as a cause of idiocy it is well to bear in mind that it may be in some instances merely concomitant, the result, rather than the cause, of a brain-defect which, passing beyond the convulsive stage, results in idiocy.

In proportion to the large number of infants and young children who suffer from convulsions, few, fortunately, become idiotic or imbecile.

Teething forms the most prominent cause of infantile convulsions. Intestinal diseases—constipation, exhaustive diarrhœa, intestinal worms—and anæmia are also enumerated among the exciting causes.

An unstable nervous organization seems to be the heritage of some families, the majority of the children at some period of early life being subjects of eclampsia.

At the Earlswood Asylum, England, fourteen per cent. of the cases were ascribed to convulsions due to teething. Convulsions are sometimes followed by hemiplegia, and a form known as paralytic idiocy may result, as will be shown farther on in this article.

Eclamptic idiots are not hopeful cases in the way of instruction, and in those instances in which the eclampsia is but an early symptom of advancing brain-change of a degenerative type they pass into the lowest order of mental deficiency.

EPILEPTIC IDIOCY.—The natural tendency of epilepsy is towards mental deterioration, and, as Echeverria has said,[1] "the earlier the age at which epilepsy springs up, the deeper it undermines the organic and moral constitution, and the more disastrous are its results."

In eclamptic idiocy the convulsions start, or are the first elements in, a series of changes which result in a cessation of mental development, and not infrequently in a retrograde movement. The convulsions cease, and are not further complications of the case. In epileptic idiocy the cause becomes also a complication, and, as time progresses, an aggravation, of the mental deficiency.

Bearing in mind Esquirol's very graphic distinction between idiocy and dementia,—the dement "was a rich man who has become poor; the idiot, on the contrary, has always been in a state of want and misery,"—it is easy, from the history of the case, to distinguish between those cases of profound dementia which result occasionally from epilepsy, and deficient mental development which it also causes.

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[1] American Journal of Insanity, July, 1873.

Ireland is inclined to fix the age of seven as the period before which if mental impairment results he would call the case one of idiocy. It is apparent that it is impossible to draw any hard-and-fast line which shall separate mental impairment from hindered mental development. I am inclined to place the age-limit considerably in advance of this line. An epileptic idiot is one whose mental development was arrested or prevented by the occurrence of epilepsy in infancy or childhood. From this classification would be excluded all those cases in which epilepsy is developed subsequently to observed mental defect. In these cases the epilepsy is a complication, and may be expected to increase the mental defect.

Petit mal and *grand mal* are both observed. The former, as is the case in epileptic insanity, is as harmful as the positive convulsions in producing idiocy, if it is not, as asserted by some, more active. The earlier the epilepsy begins, the more profound and hopeless the idiocy. If, as stated by Reynolds, hereditary epilepsy begins earlier than non-hereditary, it would be natural to expect among epileptic idiots a family history of epilepsy or other neurosis.

Teething appears in statistical tables as an active cause of infantile epilepsy. West[1] out of forty-one cases records twenty in which epilepsy began at teething. Teething can hardly be considered, however, the sole cause, but rather is to be looked upon in such cases as the spark which explodes the mine of an unstable nervous organization.

Epileptic idiots are irritable, often intractable, and hence not easily managed or trained. They are apt to retrograde rather than improve in mental capacity, and in many cases when the epilepsy appears during early childhood, after there has been some mental development, the intelligence already acquired is in large part or wholly lost.

Owing to the unpromising nature of these cases, they are, as a rule, excluded from public institutions for the training of idiots and feeble-minded. The prognosis is by many authorities considered almost universally unfavorable, and, if the medical attendant can maintain the patient in a fair degree of bodily health, control the frequency of the convulsions, and prevent further rapid mental deterioration, he will accomplish all that can be hoped for.

There are some who dissent from this somewhat sweeping assertion,—among others the late Dr. W. A. F. Browne, a distinguished Scotch alienist,[2] and a no less distinguished authority, Dr. Kerlin, of the Pennsylvania Institution for Feeble-Minded Children at Elwyn. The latter has seen in his own practice several cases in which with an amelioration or cessation of the convulsions there has been an improvement in the mental status.

I have elsewhere reported the case of a child of six and a half years in whom epilepsy was developed at the age of five. When seen, she had forgotten her name, and had lost to some extent her power of speech. Her gait was feeble and uncertain, grasp awkward, and muscular movements in general incoördinate. She was untidy in her habits, and her expression was listless and stupid. She had been saturated with the bromides, which were discontinued, and iron, cod-liver oil, and out-of-door air and exercise substituted. At the end of nearly a year she remembered the names of her physician and friends, spoke plainly and more fluently, and had better control over her muscles. At the age of nine she remained epileptic, but the convulsions were less frequent, and there had been slight progress in mental development. Her parents were of opinion—and their judgment appeared correct to me—that she had not regained the intellectual capacity present at the age of five, when the fits commenced. There had been, however, no systematic mental training.

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[1] Lectures on the Diseases of Infancy.

[2] Epileptics: their Mental Condition, Journal of Mental Science, vol. xi. p. 352.

A case now under my care exhibits the deteriorating influences of epilepsy upon both the mental and the physical expression. The patient, a man aged thirty-eight, has been an epileptic since the age of three. As a child he was regarded as feeble-minded, but went to school, and could read and had an elementary knowledge of arithmetic. As the convulsions continued, he became more dull, was irritable and at times violent towards his teachers and friends, and at the age of twenty-five was for a while in an institution for the insane. He was again taken home, where he remained, sinking gradually into fatuity, until the age of thirty-five, when it again became necessary to place him under charge. When admitted to the hospital there was marked loss of mental power. He could not read, and was able to comprehend only the most simple things. His language and manner were childish. He had hallucinations, especially after convulsive seizures, and was and is at times violent under their influence. He believes that he is assaulted in his fits and that he is called opprobrious names. He is very fleshy, his face has lost much of its human expression, and when excited he appears almost bestial, with his protruding jaws and thick lips.

His cranium is well shaped and fairly symmetrical. It has a circumference of twenty-three inches, with an antero-posterior diameter of seven and five-eighths inches and a transverse diameter of five and six-eighths inches.

The medical treatment of epileptic idiots is the treatment of epilepsy in general, plus the endeavors to train and develop the mental faculties. If there is any one thing upon which stress should be laid, it is attention to diet and nutrition. A word of caution may not be out of place against too great and routine reliance upon the bromides. Among the insane I have seen serious and sometimes lasting harm result from prolonged use of the bromide salts.

HYDROCEPHALIC IDIOCY.—The majority of cases of congenital hydrocephalus die early. In those who survive, mental development is very frequently hindered, in some cases almost wholly prevented. In a few instances the hydrocephalic condition does not increase, the brain passes through the normal stages of growth and development, and but little mental impairment—in some none—is observed in after-life.

Hydrocephalus resulting in idiocy may be either congenital or acquired. The acquired form is most frequently observed from the third to the tenth year. According to Steiner,[1] the majority of cases occur before the fourth year. In acute hydrocephalus the termination is so commonly fatal that it can be but an infrequent cause of idiocy. Some few cases pass into a chronic form. As a rule, they are feeble, scrofulous, rachitic, or tubercular, and die early. Chronic, slow-developing hydrocephalus is the form most commonly productive of idiocy. The family history of these cases will usually show the presence in direct or remote progenitors of tubercular, scrofulous, or syphilitic diseases.

The diagnosis of hydrocephalus is ordinarily easy, but it may be confounded with hypertrophy of the brain, and its presence is not always productive of an enlarged cranium. Griesinger[2] points out that hydrocephalus may be present in a cranium considerably below the normal size. Ireland calls attention to the same fact.

In hypertrophy of the brain there is enlargement of the skull, but the enlargement is most prominent on each side, above the superciliary region, while in hydrocephalus the increase is most prominent at the temples, and there is commonly a greater distance between the eyes. In cases of hydrocephalus without cranial enlargement, the expectation would be that the brain, being wholly subjected to the pressure, would suffer greater disturbance of function than in cases where the bones of the skull yielded. Deafness is not infrequently associated with hydrocephalus, and in some instances blindness.

Hydrocephalic idiots are gentle and trusting in their manners, and are of the more favorable class as regards the results to be looked for from training and education. The complications to be expected are the onset of general tubercular disease, epileptic convulsions, which usually overthrow the effects of training, and scrofula.

Paralytic Idiocy.—Infantile paralysis, whether of congenital origin or appearing in childhood, is in a certain proportion of cases followed by idiocy or imbecility.

Osler[3] says, "A lesion so serious and extensive as that which is associated with infantile hemiplegia may seriously interfere with cerebral development, and among the most common sequences we find various anomalies of intelligence."

Dr. Osler's book is based upon one hundred and fifty-one cases, twenty-three being from the Pennsylvania Institution for Feeble-Minded Children at Elwyn. Of the remaining cases, mental defects were observed in but twelve, but the author calls attention to the fact that the majority were seen early, before mental development would be retarded. The greater number showed paralytic symptoms before the fourth year of life.

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[1] Compendium of Children's Diseases.

[2] Mental Pathology and Therapeutics.

[3] The Cerebral Palsies of Children, p. 40.

The influence of abnormal labors, instrumental delivery, etc., in the production of idiocy has already been referred to, and Osler makes mention of these as causative in infantile cerebral palsies. The diseases of childhood are sometimes followed by paralysis of cerebral origin, as measles, scarlet fever, especially cases with renal lesions, diphtheria, and whooping-cough.

The pathology of these cases is yet to be worked out. Osler in his book points out the conditions which have been observed, but calls attention to the lack of observations in cases which have proved fatal soon after the onset of the paralysis. In some instances the paralysis is congenital, and due to faulty development of the motor zones or conducting tracts or to diseases or injuries during intra-uterine life. Cerebral hemorrhage occurs in children, as also embolism or thrombosis of the cerebral vessels. In the sixteen cases reported by Osler in which these conditions were found, but one, excluding three congenital cases, was under three years of age, while ten were over six. Atrophy and cerebral sclerosis have been found in several instances. Wilmarth at the Pennsylvania Institution for Feeble-Minded Children has collected a remarkable and interesting series of brains showing sclerosis. Porencephalus has been found in a few autopsies of paralytic idiots.

Cases of paralytic idiocy usually improve mentally under training, unless the fatuity is profound. The paralysis will require great patience in its treatment, without much hope of favorable results. The leg usually regains much of its lost power, but the arm remains weak, and contractions, choreiform movements, and convulsions are among the sequelæ. The use of supporting apparatus is not infrequently rendered impossible or futile through lack of intelligent co-operation on the part of the patient.

CRETINISM, the seventh in order in this classification, has already been exhaustively treated by Dr. Judson S. Bury.[1]

TRAUMATIC IDIOCY.—Idiots whose condition is due to traumatism may be included in some of the varieties already mentioned,—*e.g.*, eclamptic or paralytic,—or in the class immediately following in Ireland's classification, —inflammatory. There are some cases in which the mental state appears to be wholly due to traumatism, which may occur during intra-uterine life, during delivery, as in the paralytic cases referred to above, or during childhood; but, unless the history of the case is very clear, it will often be extremely difficult distinctly to separate traumatic cases from others.

Savage (*op. cit.*) is of the opinion that in purely traumatic cases it is the sudden injury that produces harmful results, and that injuries occurring in the first three or four years of childhood are more commonly followed by arrested brain-development than those occurring during intra-uterine life or parturition.

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[1] *Ante*, vol. ii. p. 279.

There are no distinctive features, outside the history of the case, to distinguish a case of traumatic idiocy from certain others. The degree of mental deficiency is liable to be of the nature of imbecility rather than of the profounder state of idiocy, unless the traumatism sets up serious inflammatory or destructive brain-changes, in which case, life being spared, the degree of mental impairment may be profound. Some of the cases ascribed by parents to accidental injury may prove upon inquiry to be congenital, the very injury to which the idiocy is ascribed being due to the child's defective ability to walk or otherwise care for itself.

INFLAMMATORY IDIOCY.—Instances of impaired or arrested mental development following inflammatory disturbances of the brain are met with among idiots and imbeciles, and properly belong under this division. Certain cases among congenital idiots are doubtless due to inflammation of the brain or its membranes before birth, but nothing in the condition of the patient will permit us to diagnose the cause during life. Inflammation may extend to the brain from aural or nasal disease, and, while such cases are usually fatal, idiocy may follow in some instances in the few who survive. Under this head belong those cases of hypertrophy of the brain with mental enfeeblement which are occasionally observed. The diagnostic differences between these cases and hydrocephalus have been referred to under Hydrocephalic Idiocy. The hypertrophy is largely due to connective-tissue changes, and the mental deterioration to disturbance of brain-function from pressure. Such cases are usually progressive, are hopeless, and the children generally die young, from paralysis or convulsions. In some cases the brain post mortem has been found to be very heavy. In one case reported by Spitzka the brain weighed sixty-eight ounces.

IDIOCY BY DEPRIVATION.—This is the condition which results when a child, either congenitally or in early life before the mental faculties are developed, is deprived of sight and hearing, and is thus in a sense shut off from the world. The well-known case of Kaspar Hauser, who, though possessed of his senses, was for some mysterious reason deprived of the power of exercising them, belongs to this order.

While these persons differ from true idiots, the difference, until means were devised for educating them, was in kind and not in degree. The idiot, by reason of brain-defect, is deficient mentally because his brain is to a greater or less degree incapable of receiving and recording impressions, while these, whose brains are capable of receiving and storing up impressions, are deprived of two of the most important channels through which the impressions must come. Thus, in early times deaf-mutes, who were considered incapable of education, were in law regarded as idiots, incapable of holding property, executing contracts, or testifying in courts of law. Since deaf-mutes have been educated, these restrictions have been removed. Dr. Howe has shown, in the famous case of Laura Bridgman, what can be done in such cases, demonstrating that, except when neglected and uneducated, they are not true idiots.

Mental Disturbances, etc.—Idiots occasionally have attacks of mental disturbance, which may be of an excited or depressed character, or may be associated with active hallucinations or delusions. They are also subject to delirium from fever, meningeal inflammation, etc., like other children. Carson, of Syracuse, has reported a case of opium-habit in an idiot who at the age of seven took ten grains of solid opium in twenty-four hours.

Growth and Weight in Idiots.—After a careful inquiry and extended examination, Dr. Tarbell, of Boston, announced the following conclusions, at a meeting of the Medical Officers of American Institutions for Idiots.[1] First, idiotic and feeble-minded children are two inches shorter and nine pounds lighter than normal children of their age. Second, the relative *rate* of growth of the two sexes in idiots corresponds very nearly to that of the two sexes in normal children, and is subject to the same variations at the age of puberty.

Dr. Shuttleworth, of the Royal Albert Asylum, Lancaster, England, announces about the same conclusions, in a paper presented to the same body.[2]

Pathological Anatomy.—The pathology of the various forms of idiocy has been briefly referred to under each class, where it seemed necessary. It remains to be said that the statement of Griesinger (*op. cit.*) may be adopted, that "there is scarcely any portion of the brain which has not been found either altogether absent or quite rudimentary in these creatures." The microscopical appearances of the idiot brain have been as varied as has been the gross anatomy.

Education of Idiots.—The practical questions which will be put to physicians by anxious parents and guardians will relate to the probability and possibility of improvement in the cases brought to their attention, and to the best means of bringing this about.

Though St. Vincent de Paul gathered a few idiots about him and strove to improve their condition, the first real attempt to train an idiot may be said to date from the experiments of Itard in 1801 with a being whom he considered a savage, but who was really an idiot. While Itard never continued his attempts in a second case, his pamphlet, "De l'Éducation d'un Homme sauvage," was found by those who followed him to be full of suggestive ideas.

At the present time all civilized nations pay more or less attention to the care and training of these unfortunate beings. After the earlier and irregular attempts at training and education of idiots made in France at the Bicêtre and La Salpêtrière, the first systematic efforts in this direction were made in the United States; and the schools of this country have since deservedly been models for imitation by other countries.

Education and training must, in the nature of things, have small beginnings with some idiots. In many, habits of neatness form the necessary starting-point. In others, the use of the hands, feet, eyes, and tongue must be taught by slow and patient training.

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[1] Transactions, 1884.

[2] Ibid.

The idiot often does not know how to co-ordinate his movements. His motions are awkward and rude. As far as possible these must be corrected, as the very groundwork for future effort.

The leading principle in the education of idiots demands that each case be studied by itself, and the training adapted to its peculiar mental and physical deficiencies. The general health must be improved, convulsions, as far as possible, controlled, and, when operative interference or orthopædic apparatus can be called to the aid of malformed or paralyzed extremities, the relief to be afforded will be found a valuable aid in securing further physical improvement. Operative interference upon these cases must, however, be undertaken with caution. The deformed and paralyzed members are often so defective in innervation and nutrition that reparative processes take place slowly or not at all. It will thus be seen that the work of the teacher must be directed by the knowledge and judgment of the physician.

No rule can be fixed as to the age at which training can be commenced. Much, when these cases are recognized, can be done by judicious parents at home. These unfortunates must often be taught with great patience what normal children will acquire by natural observation and imitation; and if this is impressed upon parents the subsequent work of the teacher will often be much easier.

Removal from home to institution life, even for children of the wealthy, offers the best prospects of improvement. Association with children of similar defects is not harmful, while, on the contrary, if there be other children in the family, the effect of constant association with an idiot may be bad for them, and, unless carefully guarded, the weak one is apt to be imposed upon and tyrannized over by the others.

As a result of education and training, a small proportion may be permanently improved, so as to take care of themselves and earn their own living. Others, in larger numbers, will be able to support themselves under suitable guardianship; while a still larger proportion will never advance beyond a certain point, will always remain liable to retrograde changes, and will demand continuous supervision.

Auguste Voisin,[1] Liébault,[2] and others have made use of hypnotism by suggestion in the treatment of backward and imbecile children, especially cases in which there seemed to be moral perversion,—*mauvais sujets*, as Voisin calls them. They have succeeded in producing a change in the habits and general mental state of several of these cases, which has in a few instances continued for three or four years. The patients are hypnotized, and while in the hypnotic state their bad or perverse traits are enumerated and the "suggestion" of a different course is made. Several *séances* are required in the more perverse cases, according to these authors, while in some the "suggestion" is effective if made but two or three times.

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[1] Revue de l'Hypnotisme, November, 1888, and June, 1889; British Medical Journal, September 21, 1889.

[2] Emploi de la Suggestion hypnotique pour l'Éducation des Enfants et des Adolescents, Revue de l'Hypnotisme, January, 1889.

In some cases of moral imbecility life-detention will be necessary, and no less an authority than Dr. Kerlin believes that the ordinary routine of education should be denied them, as by education they will simply attain greater expertness in crime.[1] Such cases should be trained to useful employment, and thus rendered as far as possible self-supporting.

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[1] Medical News, March 19, 1887.

INSANITY.

By E. C. SPITZKA, M.D.

Infantile mental disease offers some of the most important problems to the pædiatrist and pedagogue. The often unseen or too tardily recognized seeds of insanity in the adult are frequently sown and nurtured during infancy and adolescence. In this regard the subject before us might well merit separate volumes for discussion, covering, as it does, a large share of the general subject of the etiology of insanity. In this article it is not the intention to cover so wide a ground, but to limit ourselves strictly to those forms of mental disorder which are peculiar to childhood. The related subjects of idiocy and imbecility are discussed in another part of this volume, and hence are not considered in their important relations to the inherited forms of mental degeneration here.

Thus limited, infantile insanity is a rare disorder, and, as the material for its study is seldom to be obtained in asylums for the insane, the literature of the subject owes more to the general medical writer than to the psychiatrist. Of 1532 insane individuals statistically studied by Hagen,[1] 27 are recorded as congenitally insane and 32 as having acquired the disease during childhood. Inasmuch as of 500,000 inhabitants of the same district 149,850 ($29\frac{97}{100}$ per cent.) were under the fifteenth year, he arrived at the conclusion that one in 70,684 children annually became insane, excluding those born so. It is difficult to obtain correct figures showing the frequency of infantile insanity, for the reason above stated. Thus, Moeller in 1867 found not a single insane infant in the asylums of Saxony;[2] in 1871 he found seven accumulated in the mean time, to which small number one was added up to 1875. But during the same period there were in the same country, outside of its asylums, 122 cases registered up to 1867, 38 from 1867 to 1871, and 34 from 1871 to 1875: in all 194 cases residing with their families, and but 8 in asylums. The reluctance of parents to submit infants and children at a tender age to asylum treatment, and the lesser liability of insanity in a child to lead to open conflicts with social usages and criminal law, explain this great discrepancy between the proportion of adult and infantile asylum residents as contrasted with the corresponding non-asylum population.[1]

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[1] See also Koster and Tigges, Geschichte und Statistik der Westphälischen Provinzial Anstalt, Marsberg, supplement to Zeitschrift für Psychiatrie, vol. xxiv. p. 255, and J. L. Koch, Zwiefaltens Irrenpflegeanstalt, zur Statistik der Geisteskrankheiten in Würtemberg, Stuttgart, 1878. The above and Hagen's contribution are summarized by F. Moeller (Heppenheim), Beitrag zur Lehre von dem im Kindesalter entstehenden Irrsein, Statistische Untersuchungen über Geisteskrankheiten, Erlangen, 1876. The statistics of Oldendorff are not cited here, as they take no account of the insane outside of asylums, and do not discriminate between insanity and idiocy.

[2] Imbecility and idiocy were strictly excluded from this computation.

On carefully examining the antecedent history of the adult inmates of asylums, it is frequently possible to trace the premonitory signs and even well-marked outbreaks of insane behavior to childhood. In our experience, limiting the term insanity so that it does not include mere singularity of behavior, but such acts and symptoms as throw the individual out of sane harmony with his surroundings, over four per cent. of 3244 adult recorded private cases may be justly regarded as having been insane in childhood, while but 12 patients afflicted with infantile forms of insanity came under our observation during their infancy.[2] Just as there are more persons insane in the population of the same age who have passed the fortieth year than among those who have passed the thirtieth, so there are more insane among those between the twentieth and twenty-fifth years than among those who are under twenty, twice as many insane among those between the fifteenth and twentieth years as among those under fifteen, and nearly four times as many insane between the fifth and tenth years as among those in the first half-decade of life. These figures find their explanation in the increase of injurious influences—vicissitudes, intoxicants, wear and tear—with advancing age. That there exists any insanity in childhood whatever is but an illustration of the immutability of the laws of hereditary transmission on the one hand and of the vulnerability of the growing brain to nutritive disorders on the other.[3]

Another reason for the rarity of demonstrable mental disorder in children is the slow development of the human mental functions where these are yet rudimentary; the building-material for such complex mental disturbances as delusions and morbid projects is lacking, and hence these symptoms do not often develop. It is conceivable that a brain may be predestined to insanity even before the faculties of language and facial expression have become manifest, and it were just to pronounce its bearer insane if other means of so proving him existed[1] than those words and actions which the immature mind has not yet attained the faculty of evolving. Under these circumstances advance in education, with the powers and privileges it confers, also extends the soil on which the dread harvest of moral obliquity, illogical construction, morbid fancy, and brooding melancholy is to be reaped in distress, disaster, often in suicide or crime, and ultimately in the derelict accumulations of our numerous asylums.

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[1] In Würtemberg, of 104 infantile lunatics 24 were resident in and 80 out of asylums, the total number of insane of all ages in the country being 3948. It should be added that, among the adult insane, in 380 the disorder could be ascertained to have commenced in childhood.

[2] *Pavor nocturnus* and cerebral exhaustion, although discussed in this article, are not included in this computation. From a strictly scientific point of view the mental phenomena of febrile deliria and the somnolence attendant on cerebral diphtheritic infection are inseparable from other mental disorders. For practical reasons, the term insanity is here used in its popular sense.

[3] Choreic mental disorder, epileptic mental states, and the mental features associated with cardiac lesions, tubercular and other forms of meningitis, are, to avoid repetition, not discussed here: they will be found described in connection with the corresponding fundamental disorders. The cases of "reflex insanity" (furor with amnesia) caused by splinters in the feet (Jördens), head-injuries (Savage), whether by forceps-application or other traumatism, and intestinal worms (causing ecstasy, hallucinatory mania, etc.), properly are discussed in association with "reflex epilepsy" and "eclampsia."

Deliria before the speech-faculty is developed are conceivable, but they are not demonstrable. Hallucinations must occur even in the infant at the breast, when the vascular current rushes over the cortical registrating mosaic laden with the toxic elements of zymotic disease! But there is neither power to enunciate nor systemized memory to recollect the phantasmagoria of infantile illusions and hallucinations.[2]

From birth and up to the end of the first month of life the central nervous system, commensurate with the immaturity of the higher tracts,[3] is capable of mediating only reflex and crude co-ordinated acts. It is claimed that taste, touch, and smell precede sight and hearing, but even taste is so undiscriminating that the most nauseous articles are swallowed by infants, if not with relish, without apparent disgust.[4] As regards touch, it is many months before the space-sense is even crudely developed.[1] Whether the new-born infant distinguishes light and darkness is a question: it certainly does not acquire the association which enables it to follow a light with its eye before the seventh or ninth day, while less brilliant objects are not fixated prior to the tenth or fourteenth day.[2] A long and tedious apprenticeship must be served by the limbs, the eyes, and the equilibrium-centres, before distance, perspective, and direction become even the crude and unconscious conceptions they are in the two-year-old child. Of hearing no evidence can be obtained during the first days;[3] during the following weeks a reflex contracture of the eyes is noted after loud noises, but it is not before the second and sometimes not before the fourth month that infants learn to appreciate the direction of a sound.

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[1] The brain of an eight-months old child, whose father is a paranoiac and whose (tuberculous) mother is of an insane stock, is asymmetrical in weight of hemispheres in the basilar parts and asymmetrical as well as atypical in the gyri. Remarkable, though as inexplicable as noteworthy, is the fact that there is a superficial resemblance between it and the brain of H. J. Boldt's case of acromegaly which I have in my keeping. The atypical brain of Muhr's classical case and those of three similar ones in our possession were as atypical in the infancy of their once possessors as they now are found to be.

[2] Nutritive brain-disorders, as far as these merit a discussion separately from the underlying diseases which lead to them, are discussed in this paper. Precedent seems to justify the discussion of anæmia and hyperæmia of the brain in children. Aside from hyperæmia and anæmia incident to other diseases with which they are discussed in other parts of this work,—as, for example, hydrencephaloid in conjunction with cholera infantum,—there are no distinctive conditions in children justifying the designations of cerebral anæmia or hyperæmia, pure and simple. Those rare conditions of which vascular or nutritive conditions of the brain, and of *the brain alone*, are the important pathological factors, are discussed herein. For reasons opposing the now nearly obsolete views of older writers on this head, see the writer's article on Anæmia and Hyperæmia of the Brain in Pepper's System of Medicine, vol. v.

[3] Whose myelin is not yet developed: the associating tracts still have the apparent vascularity and translucent grayish-reddish tinge of the embryonic period. The cortical nerve-elements, especially the large pyramids of the paracentral region (Betz), are also as yet undeveloped.

[4] In attempting, by means of a liberal application of asafetida, to check the thumb-sucking proclivities of an infant, the experimenter's attention was ludicrously directed to the possibility of inculcating any drug habit, provided it be begun early enough. Up to

Emotions are at first limited to pleasure in its most animal form (during suckling), and pain (undifferentiated as regards its quality or interpretation of cause). Observers like Darwin state that fear is the first emotion manifested.[4] This is not always the case; but, be it as it may, anger, the next emotion to develop, is in some infants a reaction from fear, in others an expression of disappointment in obtaining a coveted object. It is under the latter circumstances that the watchful parent or guardian may do much in properly moulding a character inclined to morbid egotism.

During the first few months of life, some infants exhibit, to careful observers, singular and isolated manifestations, indicating that the crude functional impulse occasionally falls into a pre-existing rut, fragmentary though it be. Peculiar habits, movements, and grimaces of the parent can thus be recognized in the child.[5] Good observers believe, with some reason, that

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and beyond the eighth month the child—an intelligent one, and since become a good discriminator—was not broken of the habit by this, *a priori* one would suppose, radical measure.

[1] The reader is referred to Meynert's fascinating and philosophical description of the gradual development of the sense of personal identity and differentiation of the outer world,—two of the fundamental factors of healthy individuality.

[2] An enormous individual difference exists in this respect. As Darwin has shown, curiosity is the underlying incentive to higher intellectual development, and it is not difficult to read the horoscope of children who fail to fixate objects as late as the eighth or tenth week of life.

[3] The pathetic tale of the deaf-mute mother who raised a rock and let it fall near the infant, in order to test its hearing, and was overjoyed at the result, is apocryphal.

[4] We have an observation of a child at the second month, whose first emotion was an uncontrollable outburst of laughter, in consequence of an impatient movement of a ludicrous character on the part of the nurse, which the child provoked again and again, to indulge in the same cachinnation each time. The same child at four months while sitting on the carpet was suddenly attacked by a young fleecy dog; he started back, trembling in his hands for a moment, and then, flushing up, threw himself forward, grasped the dog on both sides by the neck, held him there, and broke out in exclamations of anger.

[5] A child of German parentage aged nine weeks in our presence said "Gockelgock," but no child of German parents would ever be capable of saying, as a child of Bohemian maternity said at the eleventh week, also in our presence, "Tszätz." In both cases the occurrence was isolated, not preceded nor followed for months by a similar one.

imperative conceptions, morbid fears, and imperative movements may be foreshadowed at this early period.[1]

The first educational step in the civilized infant's career is the inculcation of cleanliness. A fair estimate of a child's ancestry may usually be derived from its tolerance or intolerance of a soiled or wet diaper.[2] One of the most marked antipathies shown by the child of fastidious parents at the early age of a few weeks or months is to moisture, and symbolic expressions of this dislike lead the watchful nurse to establish the first *étape* of mental discipline.

Dislike to fellow-creatures, manifested by aggressive pushing or passive pouting, is not usually shown before the tenth or twelfth month, and is often as apparently inexplicable as the so-called instinctive aversion or attachment of animals. These apparently trivial acts merit more careful study than they have yet obtained.

As with animals, the power of the infant to understand words and short sentences exceeds the power to express them. With the completion of the first year the child, rising erect, passes rapidly through that great step in evolution accomplished by its ancestry slowly and gradually in the geological past, and with a wider horizon of vision and the added gauge of its own unfettered steps, its ability to react on the impressions made by its surroundings increases mentally, week by week, in a progressing ratio. That it is the bearer of the vices as well as of the virtues of a long ancestry it now manifests by developing self-consciousness, obstinacy, and egotism,—or, rather, born and naturally developing as an egoist, it soon learns the immediate profit to be derived from low cunning and simulation. To steal, to lie, to hide, and to sham are as natural acts at this age as any act of self-preservation, aggressive or defensive, is in the lower animals. Here, again, the nurse may lay an important foundation by teaching the egoist, through reward and punishment, a higher egoism, of which truthfulness and honesty are inseparable elements. And at this period also the outcroppings of the imitative tendency cannot be neglected, either by the moralist who knows the great force of example, the sociologist who appreciates the influence of environment, or the alienist who sees their tragical influence when of a bad character in communicated insanity.[3]

From the end of the first to the third year the hand of the child is used. Much more than theorists admit rests on the proper employment of that wonderful mechanism, to the overgrowth of one part of which man owes his superiority to the troglodyte apes.[1] Even a defective or lethargic child, as the elder Seguin demonstrated, can be made mentally brighter, a vivacious and even a useful member of the community, by drilling this important instrument,—in other words, reforming the mechanic by adjusting and adding to his instruments.

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[1] In pseudo-hypertrophic muscular atrophy, a disease frequently found in families afflicted with neural degeneration, we have seen imperative movements of a peculiar character. In one case this movement was a unilateral running of the index knuckle across the tip of the nose, and had been noticed since the child was able to sit up, and continues now to his seventeenth year.

[2] Curious estimates have been made as to the period when this intolerance is first manifested, varying so much that we must attribute them to the varying customs of different nationalities.

[3] Mental contagion, *folie à deux*, *folie communiquée*.

In addition to the predisposing causes of insanity in children, there are a number of exciting ones, differing in the intensity of their operation from the corresponding causes in the adult. Among these, sudden changes of temperature are remarkable in that they, uncomplicated by any other cause, may provoke transitory furor and acute delirious states. This is equally true of both extremes of temperature. Over-exposure to the sun[2] and permitting infants to sleep with the unprotected head exposed to a hot stove have been declared responsible for the same acute delirious or furious outbreaks that Reich observed in children who, having been long exposed to severe wintry cold, suddenly entered a warm room.

Of other accidental causes operating directly on the nerve-centres or their envelopes, head-injuries are notorious for their dire effect in children. As a rule, they cause various forms of idiocy and imbecility, which are treated of in another part of this work, or appear as complications of epileptic states. In some exceptional instances acute or protracted maniacal excitement has ensued. Here, as elsewhere, it is noteworthy that infantile insanity differs from the insanity of the adult when provoked by the same causes, by producing a blinder, more instinctive and impulsive fury. In several of the recorded (and rare) cases the maniacal excitement recurred at intervals of weeks and months, the intelligence remaining unaffected in the intervals, and eventually a periodical psychosis developed.

Injuries to the peripheral nerves have been credited with producing mania in children, but in most instances of this character the complete amnesia of the child and the disappearance of the attacks on the removal of the cause demonstrate that the mania was a psychical equivalent of a reflex epilepsy.[3] That extreme pain alone does not produce delirium or mania more frequently seems remarkable when we bear in mind the sensitiveness and mobility of the infantile nervous mechanism. Even the intense and agonizing pain accompanying disease of the middle ear is rarely productive of abnormal mental states, and the evil influence of ear-disease on the mind of the developing child is usually of a negative character, operating as would the cutting off of any other channel of sensory training,—through the ensuing deafness.[4]

Diseases of the throat and nose rarely lead to noteworthy mental disturbance. Chronic nasal catarrh, through the associated headaches, often causes dulness, which interferes with mental concentration and consequently with the education of children thus afflicted.[1]

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[1] Sir Charles Bell, The Human Hand.

[2] Steiner, Compendium der Kinderkrankheiten, and A. Vogel, Kinderkrankheiten.

[3] As in the classical cases of Tördens and Engelken, where the causes were respectively a glass splinter in one great toe and a carious tooth. Pressure on the toe in the former case, and attempts to extract the tooth in the latter, provoked acute mental disturbance.

[4] See Deaf-Mutism, in Dr. Burnett's article on Diseases of the Ear, *ante*, p. 36.

Abnormal states of the digestive apparatus are frequently accused of causing mental disturbances in children, chiefly hypochondriacal in character. There is a peculiar condition observed in girls about the period of puberty, which, whatever its original cause, is intensified and modified by the state of the stomach. This state does not appear to be continuously pathological, for even where death from starvation has ensued the stomach was found normal in structure. It seems to be rather a functional perversion. A patient of this class develops a slight dyspepsia, and, rendered morbidly sensitive by an existing nervous predisposition, exaggerated by the pubescent state, she contracts a positive dislike for food. Improper food—for injudicious parents attempt to humor the patient with pies, pastries, and candies—causes further distress; then tonics, appetizing cordials, and chalybeates, with which a mistaken if well-intentioned therapeusis drenches her, increase the dislike, which ultimately becomes metamorphosed into the conviction that all food is injurious. The patient's energies then become devoted to the one fixed purpose of resisting its introduction, and, if the religious training be of a certain kind, the delusion may develop that eating is sinful and that God has forbidden the patient to eat. Meanwhile, the stomach, originally perhaps but slightly diseased, ceases to present any active signs of gastric catarrh; yet the patient does not resume eating, but lives for months on an occasional sip of tea or broth, and is visited by crowds of marvel-lovers, to be canonized as a "fasting girl." The proper place for such a child is the asylum, where the apparatus employed for forced feeding with digestible and assimilable food would soon overcome the starvation and the delusion which first grew out of and later maintained it.

Much interest attaches to the influence of intestinal parasites in insanity. That these are competent to provoke a variety of nervous troubles, such as eclampsia, epilepsy, chorea, hemiplegia, and paraplegia, is admitted, but few authors recognize that actual insanity may thence result. The rapid cure of a maniacal delirium after the expulsion of ascarides, in several cases, proves that this etiological assignment is a correct one.[2] As a rule, the mental disorders thus caused are of an acute type and often associated with eclampsia. Those parasites which have their seat low down in the bowel or rectum, such as the oxyuris, have a less direct relation to mental disturbance, when, as in not a few cases, they provoke masturbation, either by the scratching of the anus they cause, or by wandering into the genital passages.

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[1] There is no justification for the creation of a special clinical type, "Aprosexia nasalis," as Guye (Allgemeine Zeitschrift für Psychiatrie, 1888, p. 537) claims. This writer seems to ignore the frequent presence of thickening and varicose developments of the nasal mucous membrane in masturbators.

[2] However, care should be taken not to confound the results of parasitic irritation with those due to the drug employed in removing it.

Self-abuse bears such intimate relations to the production and clinical modification of mental disease in children, that it requires a separate consideration, together with the special form of insanity it causes and modifies.

Febrile and other acute disorders sometimes cause serious and incurable insanity, usually combined with more or less arrest of development. This result is most apt to follow when some moral cause, such as a shock or fright, is superadded.[1] Authors attribute from ten to thirty per cent. of infantile insanity to the acute diseases of childhood, the overwhelming majority thereof being sequelæ of the exanthemata. Excluding those cases followed by simple imbecility, amnesia, aphasia, and dementia, the writer finds but seven per cent. of his cases to be attributable to these causes. This low percentage may be accounted for by the fact that the writer's figures do not include those cases which terminate fatally, nor those ephemeral deliria which sometimes in children replace malarial attacks, and which are benign, self-limiting, and not apt to fall under the ken of the alienist, like the more serious and protracted mental complications of the paludal dyscrasia, which may attain the degree of a pseudo-cretinism.

Aside from a few cases of hallucinatory delirium complicating pertussis, which naturally takes the form of precordial terror, it is scarlet fever, measles, typhoid, and acute articular rheumatism which are most to be dreaded in reference to their immediate and remote mental sequelæ. It is particularly the third-named which exerts a disastrous effect on the developing mind. The bright child becomes a laggard, the brilliant memory enfeebled and obliterated, and the moral nature apparently revolutionized. That it is to the acute disorder, and to no predisposition, that this dire result is attributable, is proved in every physician's experience with families of excellent physical and mental health, of whom the single dullard, lunatic, or black sheep has become such after a typhoid, a typhus, or a scarlatina. It is not yet determined whether these psychical results are due to the specific disease-germ and its direct noxious influence on the nerve-céntres, or are produced more indirectly by the profound nutritive disturbance. The former is, however, the more probable hypothesis. It is, in the writer's opinion, based on the following series of facts: 1st. Analogous affections, such as the progressive fatal sopor following diphtheria, are accompanied by evidences of microparasitic invasion of the nerve-centres.[2] 2d. The psychical results of post-febrile insanity are different from those resulting from simple nutritive disturbance. 3d. The organic nervous diseases exceptionally following essential and exanthematous fever are usually multilocular,[1] and indicate the operation of an irritant distinct from a mere deprivation of nutriment.

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[1] A distressing case of this kind is that of Louisa W., demonstrated by the writer before the New York Neurological Society (American Journal of Neurology and Psychiatry, 1882, vol. i. p. 502). She discovered her brother's death from scarlatina while herself ill on the third day after the outbreak of the disease in her. Epileptiform attacks and maniacal furor alternated, and left her in a demented state, in which she continually repeated, "My name is Louisa W—— and Papa W—— and Mamma W—— and Baby W——." The latter referred to the deceased brother.

[2] Letzerich, of Brunswick, Germany, in the case of his own child, found the perivascular and pericellular spaces of the cerebral cortex crowded with micrococci. I have found

Nasse[2] classifies the febrile insanities according to their period of development: *A*. Coinciding with the fever as to time, and ranging from delirium to outspoken insanity. *B*. Developing as an apparent continuation of the specific somatic disorder, so that a true convalescence cannot be spoken of. This is often associated with hyperthermic states. *C*. Developing during convalescence. The latter group, in the writer's opinion, belong to a different class from the other two; they are probably merely anæmic or adynamic, and are much more benign in character and prospect. The first two groups are more frequent in children, the third is more often found in adults.

Of moral causes, fright is the most influential in producing mental aberration,—as, indeed, it is with regard to the neuroses generally. Its frequent *rôle* as an important accessory factor in febrile and post-febrile infantile insanity has been referred to. Independently of somatic complications, sudden terror seems competent to unbalance even the healthy infantile mind; and many morbid fears, imperative conceptions and acts, which torture the individual throughout an otherwise healthy career, have unquestionably their origin in the earlier periods of life. Anxious mothers, in impressing their young with caution regarding real and imaginary dangers, are apt also to impress them with a morbid anxiety anent the latter. Ghost-stories have been responsible for numerous cases of neurotic disorder, and silly jokes perpetrated by thoughtless adults on timid children have produced most serious results as regards their mentality. Lyssophobia with a fatal termination counts more victims, particularly among the young, than real rabies.[3] The somatic signs of terror and fear in children are in their higher grades evidences of a serious disturbance of innervation. Restlessness, repelling motions to strangers and even parents, tearing off of the clothing, vague destructiveness, are accompanied by pallor, cyanosis, diminution of conscious-

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a similar condition in a case of erysipelas with delirium, and one case of invasion by an unidentified micro-organism in a patient dying with signs of *delirium grave*. Mental disorder can be produced by experimental inoculation of the brain in animals without demonstrable organic reaction of that organ or its membranes (Mollenhauer, Journal of Nervous and Mental Diseases, September, 1890).

[1] Multiple sclerosis following variola, scarlatina, etc., according to Westphal.

[2] Allgemeine Zeitschrift für Psychiatrie, 1870; see also American Journal of Neurology and Psychiatry, 1883, p. 206.

[3] A distinguished physician of New York City, being called to a boy aged eleven years who had been frightened into lyssophobia by his comrades and by some adult persons and who already presented laryngeal spasms, told him that if he passed the "crisis," predicted to occur at exactly nine o'clock, he would be safe. The boy's expectant attention thus directed to the minute-hand of the clock, instead of to his sensations, he recovered. Need the writer state what the result would have been if any other course had been followed?

ness, dilatation of the pupils, and increased and intermittent heart-action. Attacks of this character are sometimes accompanied by diarrhœa, and usually followed by copious discharges of urine possessing the hysterical character. Intercurrent with the disturbed consciousness, incomprehensibly strange acts are noticed,—meaningless laughter, rhythmical sing-song, and wild explosions of passionate attachment to the parents or other relatives, in singular contrast with the preceding and ensuing defensive and repelling acts and cries.

Children affected with organic cardiac disease or defect are particularly liable to these distressing results of fear and terror, and a fatal result is exceptionally recorded in cases which have become historical. A distressing form of mental aberration, *pavor nocturnus*, to which also healthy children are liable, has been in several of these instances the apparent cause of death. This peculiar disturbance may be disposed of at this point: a child, as the result either of a sudden fright, or of the reproduction of that fright in the form of a dream, awakes suddenly, exhibits the signs already detailed, and after full return of consciousness becomes calm and goes to sleep. The attacks under proper dietetic treatment and moral management become less and less frequent and severe: the importance of early instituting these latter is not to be underrated, for in the author's experience two cases have occurred in which true epilepsy developed from what originally was a simple *pavor nocturnus*.[1]

Masturbation is, of all accessory causes of mental disorder in children, the one whose influence is most readily recognized, although a small minority of writers deny altogether its power for doing such evil.[2] The classical writers regarded it as an important and frequent etiological factor. Some attribute as high as twenty-five per cent. of all cases to this cause (Ellinger). Luther Bell, who furnishes one of the earliest and best pictures of this condition, gives as its leading features loss of self-respect, a mischievous, dangerous disposition, and a tendency to dementia. Griesinger adds that the majority of cases are marked by a profound dulness of sentiment, by mental exhaustion, and by religious delusions, with hallucinations of hearing. What the writer regards as the typical masturbational psychosis occurs between the thirteenth and the twentieth year. In younger subjects the symptoms found between these ages are seldom developed, the dementia is not so peculiarly variable, and is more like a true imbecility. In addition, infantile insane masturbators are more liable to epileptiform complications and blind impulsive outbursts of maniacal fury. Both these features are characteristic of other forms of insanity in the very young.

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[1] In one case the child had been liberally dosed with whiskey,—a panacea, as I am informed, even among comparatively well-educated people in certain districts of Alabama; in the other, gross dietetic errors were permitted after an alarm occasioned by a lightning-stroke. In the latter case the patient had been psychically shocked only, and the transformation to a true epilepsy occurred over three months after the original fright.

[2] I believe but a single one, Möbius, who, in Schmidt's Jahrbücher, in reviewing the writer's monograph (Journal of Mental Science, 1887, 1888), asserts that the vice never causes insanity! It is but justice to this distinguished neurologist to add that in making this statement he believed himself to voice the opinion of all German alienists; and it is due to the large and erudite body of German alienists to state that Möbius labored under a misconception of his representative position when he indulged in this pronunciamento.

The progress of this disorder is characteristically variable. Destructive and aggressive tendencies preponderate at some periods, depression, anxiety, and mental and physical lethargy at others. As a rule, there is a gradual deterioration, interrupted by abrupt temporary improvement and equally abrupt descents, attributable to remissions and exacerbations of the underlying vice. These more rapid changes on the background of the general decline mainly involve the memory and the moral sense.

The variability in the intensity and nature of the symptoms is so great as to be noticeable even within the confines of a single day. The boy who rises confused, with an absent expression, or listlessly remains in bed, may improve as the day advances, and be vivacious, intelligent, and active in the afternoon. This is attributable to the direct effect of single, as the more prolonged conditions above alluded to are to serial, excesses. The act is more apt to be practised during the night, involuntary losses to occur towards morning, and the effect is noticeable on rising. As the day progresses, the recuperative powers of the youthful organism succeed in obliterating these immediate effects. As long as these efforts of struggling nature are noticeable, recovery is possible, provided the habit and its immediate sequelæ be checked. In the later periods of the confirmed disease, in those cases where the habit has been discontinued because of impotence, an opposite condition, of sinister import, is found: the patient awakens comparatively bright, coherent, and active, as the day wanes he becomes irritable and silly by spells, and in the evening he is the dement of the evening previous. This indicates an extreme exhaustibility of the nervous system, which, refreshed by sleep, is not sufficiently so to bear the strain of continued effort, thus leading to an anergic stupor.[1]

Besides the type of mental disorder to which the name masturbational is properly given, other forms of insanity may appear in masturbating children. Thus, simple mania and melancholia are reported to have thus occurred. It is not, however, certain that delirious episodes of other diseases and maniacal or depressed phases of hebephrenia have not been confounded therewith. Imperative conceptions, morbid fears, and *folie du doute* are frequent in infantile masturbators, and, as already stated, these conditions, though usually noticed only in more advanced life, probably owe their origin to neurotic causes operating in childhood or adolescence. Hypochondriacal and persecutional paranoia in a crude form are similarly detected at this period. A most favorable form of mental disturbance is simple stupor,[1] which in young masturbators is sometimes associated with anxious or hypochondriacal states. Over ninety per cent. of this class of cases recover, in the writer's experience.

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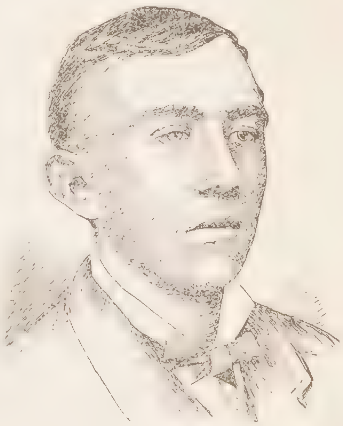
[1] In simple recoverable stupor a similar diurnal change is noted, but there is no irritability or silliness, and the depression appears at a later and later period of successive days until finally it ceases to recur.

Few patients suffering from the fully-developed masturbational psychosis recover. The most favorable outlook is in those cases in which it is rapidly produced as a consequence of accumulated excesses extending over a brief period of time. Reaction of a favorable kind is more apt to occur under these circumstances than where the nervous system becomes broken down by slow stages and by repeated or long-continued assaults. Often it is a question, practically speaking, as to which of two factors does precede the other,—extinction of sexual desire by overstimulation, or dementia. The supervention of a stuporous state[2] through the intense exhaustion induced by the excesses which often follow the "first lesson" in the habit may prove the salvation of the patient, by directing attention to the vice and extinguishing the temptation to indulge in it.

Of individual symptoms the most unfavorable one in a prognostic respect is a loss of the sense of shame.[3] As long as shame is present, an incentive to control the habit can be created, provided it does not already exist in the patient's mind, and the ability to refrain from indulgence, after the serious consequences are pointed out or the patient's pride is called into play, is the most efficient guarantee of recovery. As a rule, the younger the child the easier is it to check the habit by methods purely supervisory and coercive. It is in older *habitués* that the habit and the entailed deterioration are sometimes arrested because temporary or lasting impotency enforces continence. At best, recoveries under these circumstances are but imperfect. More frequently seminal leakage, diurnal or nocturnal emissions, continue the damage after puberty. For this reason, the prognosis generally becomes more and more serious with advancing years.

FIG. 1.



The expression of countenance characteristic of patients who in this condition reach the period of puberty and continue in it is well illustrated in the accompanying wood-cut.

The morbid anatomy of masturbational insanity is unknown as far as the infantile period is concerned. That the nutrition of the brain and spinal cord is seriously impaired by this habit is not alone supported by the frequent association of signs of so-called cerebro-spinal exhaustion and irritation with the masturbational neurosis, but also by the profound necrotic passive brain-changes discovered in one subject suffering from its most aggravated form.[1]

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[1] Not to be confounded with delusional stupor.

[2] Inaptly called "primary dementia" by some English writers.

[3] Providing always that it be a real loss of modesty and absence of contrition, and not an affectation, such as masturbators are apt to indulge in, either from motives of bravado or of concealment.

The treatment of this form of mental disorder it is customary to regard as among the most difficult if not hopeless tasks of the alienist. The majority of those who have interested themselves in the subject are inclined to adopt the adage, "once an onanist, always an onanist," or to fall into the still more disastrous error of regarding marriage, or what is its physiological equivalent (if a sorry moral alternative), as the panacea for all masturbatory disorders, mental as well as physical.[2] While the desirability of permitting the sexual organs to functionate in a normal instead of an abnormal direction in precocious individuals is evident, yet the limits of this work confine us to the treatment of the habit in those who have not passed the fourteenth year. This may be briefly epitomized as follows. In infants, painful corporal punishment should follow every attempt at touching the privates or executing thigh-friction.[3] To no other argument is so young a child accessible, and its effect is rapid and radical. Nowhere would a certain kind of sentimentality directed against corporal punishment deserve the adjective foolish so much as if exerted in such a case.

In children who have reached the period when the sense of shame becomes developed, constant observation to determine the frequency and extent of indulgence should be made. No false accusation should be risked as a venture. By such, attention might become directed to the very point it is so desirable not to call a child's attention to. But if the existence of the vice become established, a well-acted performance on the part of the parent, such as expressing surprise at the child's bad appearance or capricious appetite, a frequent recurrence to the same topic through the day, and particularly that comparison with other children[4] which arouses emulation and hence introspection, may suffice to check the habit in sensitive and sensible children, whom a brutally formulated accusation would certainly confirm in the bad habit from the very perverseness which grows out of it. Should these indirect and diplomatic measures fail, the direct accusation must be made, and made by a stranger, preferably the physician, still permitting the child to believe that its parents are kept in ignorance. Thus we appeal to that last remnant of self-respect, and one which involves a most powerful motive for self-restraint,—the fear of becoming an object of contempt in its own household and of losing the confidence of its nearest on earth. Constant observation must be continued at every sacrifice, and should the vice be repeated, a threat to divulge the fact, which is as yet kept a supposed secret between physician and child, may be made, but if made must be executed, and mechanical restraint applied. The various forms of restraint it is not our purpose to discuss in full here; they will vary with the sex, age, and character of the child. The endless sleeve will suffice for the manual performer. Thigh-friction can be prevented by leather knee-caps held apart by a firm wooden rod connected with the knee-caps by ball-and-socket joints. It is needless to add that any local source of irritation, *oxyuris*, adherent prepuce, or accumulated *smegma præputialis*, should be inquired into, and, if discovered, removed.[1]

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[1] It was particularly the lumbar spinal cord which exhibited general atrophy and the occipital lobes of the cerebrum which showed calcification and lacunar softening in symmetrical patches. In this connection the eye-disturbances of masturbators are to be borne in mind: concentric limitation of the field of vision is found in adult sufferers, and temporary amblyopia in youthful *habitués*.

[2] A case of epileptiform status followed by a condition resembling paretic dementia and recovered from *through continence* in a youth who had never indulged in artificial sexual acts, but who had unlimited access to the opposite sex at the age of thirteen, and another where coitus at eleven was followed by seminal losses and diabetes,—also recovered from through continence,—teach a different lesson. It is also to be borne in mind by those who advise illicit intercourse for so-called medical reasons, that the spermatorrhœa for which it is recommended is not improved by an added gonorrhœa, but apt to be aggravated thereby.

[3] The usual and, from its significance being misunderstood, most occult and hence most dangerous form of the habit. See Journal of Mental Science, *loc. cit.*

[4] With this it is assumed that the standards of comparison shall not be the very children from whom the habit may have been acquired by imitation. It is Mantegazza, the writer

Should all these measures fail, and what is known as "psychical onanism"—that is, the ability to provoke the orgasm without manipulation or friction, and, worse still, without erection—be acquired, there is no other refuge but asylum treatment. Unfortunately, we have in this land as yet no institutions calculated for or competent to treat such cases, and in Europe there is but a single institution, the "Medical-Paedagogium" of Görlitz (Prussia), whose medical directors devote attention to this numerous and sad class of invalids.

Another form of mental perversion occurring in infancy is a peculiar form of moral imbecility. Morality in the higher sense being an acquirement of more mature life, the moral insanity of children does not manifest itself so much in an absence of that moral sense which is regarded as an

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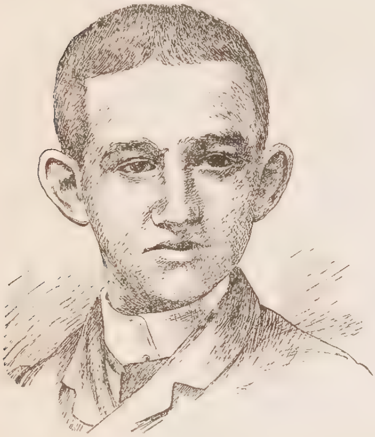
believes, who, in his "Fisiologia dell' Amore," says that if an inhabitant of some distant planet were to visit the earth and describe the various living forms inhabiting it, he could not fail to incorporate the masturbational habit as a character of our race. While his exaggeration is obvious in more than one direction, yet it must be admitted that among males of civilized races the habit is frightfully common. In captivity male monkeys, elephants, bears, and other animals frequently fall victims to self-abuse; and it appears that civilization, with its associated artificial conditions, is responsible for the great preponderance of this injurious habit in the male of our own species.

[1] The exaggerated claims made by authority too eminent to attack lightly, that paralysis, imbecility, and epilepsy could be relieved and cured by removing preputial adhesions, have led to as extreme a reaction. No doubt exists to-day that the claims above stated were based on self-deception, and that they were scientifically indefensible, but it is not the less true that a profound influence for good can be exerted on the development of a child, mentally as well as physically, by remedying redundancy or adhesions of the prepuce.

integral of normal adult mentality, but in an absence of "good nature;" the child does not exhibit that open confidence towards its companions, that joyous caressing of its parents and brethren, that self-forgetfulness at play, which form one of the chief charms of this period of life. In infancy convulsions or fainting-spells are sometimes noted; great irritability, particularly at dentition, and a tendency to outbreaks of fury, with violent motor acts, such as butting the head against others, stamping on the floor, are observed, of such severity as to lead to the apprehension (sometimes justified) that a convulsion may close the scene. Such children either remain cold-hearted and selfish, or, in addition, become actively cruel, delighting in torturing animals, destroying whatever gives pleasure to others, and after the third year may develop premature sexual desires leading to onanism, indecent exhibitions, and, finally, to a complication with the mental results of self-abuse already described. Singular impulses are occasionally developed, usually growing out of an extravagant fondness for witnessing some act of destruction or torture. Pyromania[1] is a term applied to one form of this impulse, which exhibits itself in acts of incendiarism. Maiming and cutting of other children, even to the point of murder, is the form taken by the morbid impulse in others, as in the case of the boy monster Pomeroy. The difficulty of estimating the pathological nature of such impulses and the associated "moral color-blindness" is enhanced by the fact that the ordinary intellectual powers memory and judgment, while notably affected in some of these children, are not alone unimpaired in others, but even better developed than in average healthy children. However, even where these faculties remain intact, as the child grows older they become perverted to subserve the ingenious trickery and knavery with which the unfortunate being carries out the most unprofitable criminal plans, becomes the terror of its fellows, the *bête noire* of the successive schools, institutions, and prisons into which it drifts, and the disgrace of its family. In such a case crafty dissimulation enables the child to deceive even the expert observer, and as this condition, although involving the fundamental character, is not an unvarying one, the actual appearance and conduct of the subject may

FIG. 2.



A subject of moral imbecility, who at the age of nine years exhibited great cruelty to his brothers, sisters, and mother, was easily cowed by an overbearing, paranoiac father, and developed considerable artistic, calligraphic, and musical ability.

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[1] It is manifestly improper to use this name, which merely designates a single symptom and a single form of morbid impulse, as a term designating a form of insanity. Incendiary acts *not* of an impulsive nature occur with other forms of mental disease, and even in adults.

deceive the alienist and criminal jurist, leading the former to regard it as having recovered, the latter to consider it fitter for the reformatory than for the asylum.[1]

Among these cases of "original" moral imbecility[2] there are many which exhibit a tendency to the formation of delusional opinion and morbid projects. Nothing is more common in this field of the alienist's experience than to have a child, precociously bright in some respects, but morally defective, and the curse of the household in every other, pick up some notion from the daily papers and form a sentimental attachment to some particular person, of whom it is as extravagantly fond as it is cruel and cold to others. In connection with such an attachment, sometimes shown to a teacher or to a neighboring mechanic or official personage, some particular branch of learning, or, more often, of mechanical labor, is cultivated by the child. If a healthy emulation can be excited on this basis,—that is, if the child can be induced to excel in its performances in order to please the person to whom it has formed an attachment,—a channel is opened for reclaiming it.

Usually the home surroundings of children organically tainted with moral imbecility, paranoiac tendencies, and mental lethargy are of a most unhappy character. Numerous cases recur in the annals of every asylum for defective children and for the insane where such children led a comparatively healthful and useful life as long as they remained at the institution, and relapsed at home owing to injudicious management. An hysterical mother, a perverse project-making father, or both parents thwarting each other, are not calculated to combat morbid tendencies inherent in the infantile organism. The management, control, education, and civil as well as criminal competency of such children are topics which, in view of the approaching problems likely to grow out of the threatened over-population of our large cities, if not of the country at large, may need to be discussed from other than strictly medical and sentimental points of view.

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[1] The writer is prepared to say that the demoralizing influence of such a child on other children is so great that, if the principle "the greatest good to the greatest number" be made the leading one in the asylums for idiotic and defective children, it were better that one incurable unfortunate should be imprisoned for life in a common jail than that twenty curable children should have their cases jeopardized by being compelled to associate with it.

[2] The writer is not acquainted with any better English equivalent for the word *originär*, as used by the Germans, unless it be our familiar term "congenital."

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THE END.

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